ADA Standards of Medical Care in Diabetes (2025)

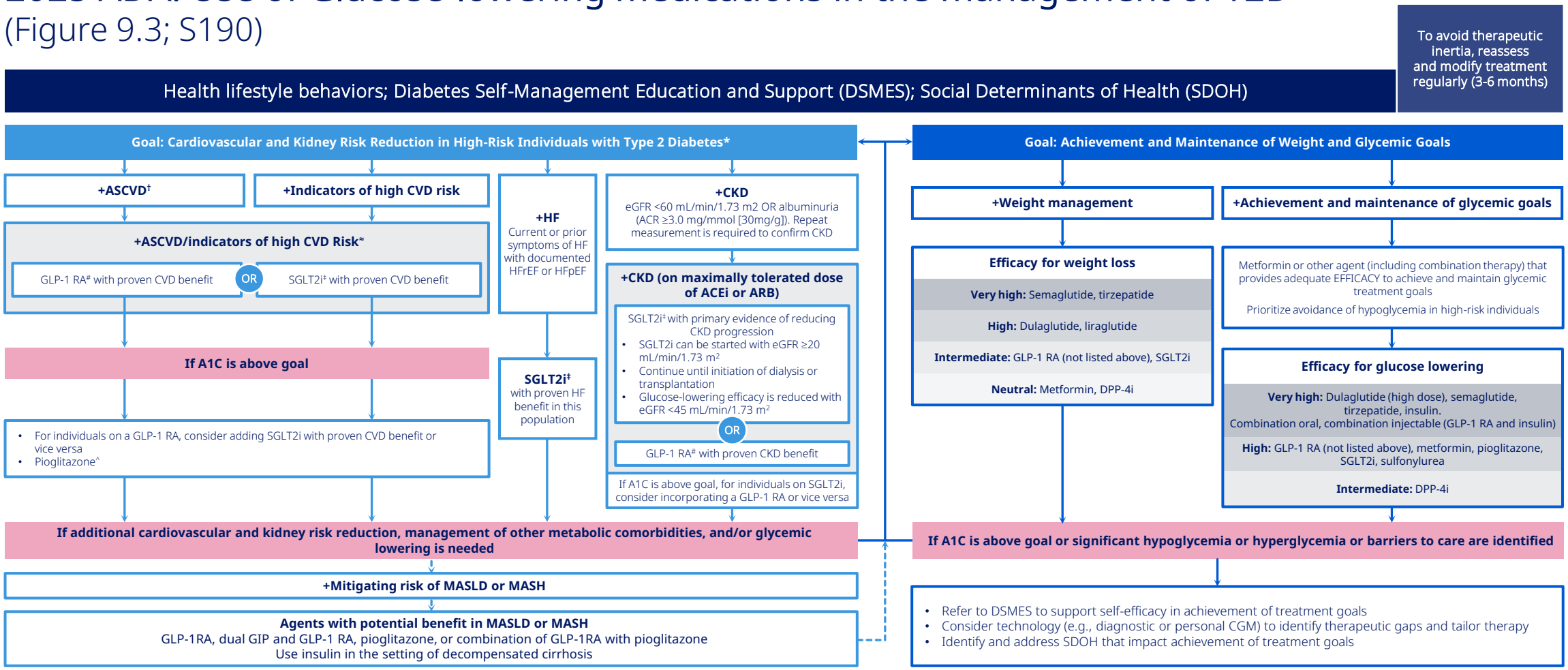
Diabetes Care Volume 48, Supplement 1, January 2025

This is not an all-inclusive list. Please refer to source document for full recommendations, including level of evidence rating

ADA, American Diabetes Association

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

2025 ADA: Use of Glucose-lowering medications in the management of T2D (Figure 9.3; S190)



*In people with HF, CKD, established CVD, or multiple risk factors for CVD, the decision to use a GLP-1 RA or SGLT2i with proven benefit should be made irrespective of background use of metformin or A1C.
† ASCVD: Defined differently across CVOTs but all included individuals with established CVD (e.g., MI, stroke, and arterial revascularization procedure) and variably included conditions such as transient ischemic attack, unstable angina, amputation and symptomatic or asymptomatic coronary artery disease. Indicators of high risk: While definitions vary, most comprise ≥55 years of age with two or more additional risk factors (including obesity, hypertension, smoking, dyslipidemia, or albuminuria).
≈ A strong recommendation is warranted for people with CVD and a weaker recommendation for those with indicators of high-risk CVD. Moreover, a higher absolute risk reduction and thus lower numbers needed to treat are seen at higher levels of baseline risk and should be factored into the shared decision-making process. See text for details.
For GLP-1 RAs, CVOTs demonstrate their efficacy in reducing composite MACE, CV death, all-cause mortality, MI, stroke, and kidney end points in individuals with T2D with established or high risk of CVD. One kidney outcome trial demonstrated benefit in reducing persistent eGFR reduction and CV death for a GLP-1 RA in individuals with CKD and T2D.
‡ For SGLT2is, CV and kidney outcomes trials demonstrate their efficacy in reducing the risks of composite MACE, CV death, all-cause mortality, MI, HHF, and kidney outcomes in individuals with T2D and established or high risk of CVD.
^ Low-dose pioglitazone may be better tolerated and similarly effective as higher doses
Diabetes Care 2025;48(Suppl. 1):S181–S206 | doi: https://doi.org/10.2337/dc25-S009; Adapted from Davies et al Diabetes Care 2022;45:2753–2786

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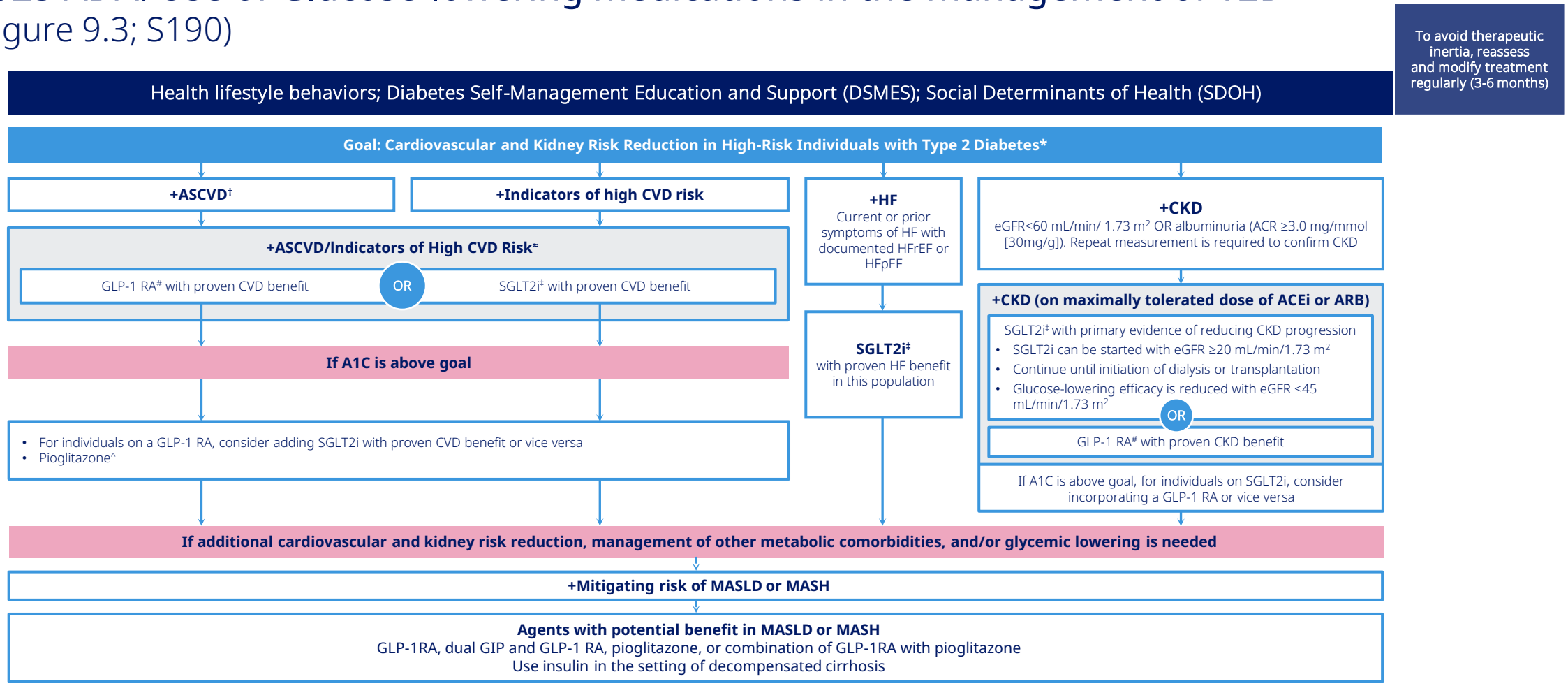

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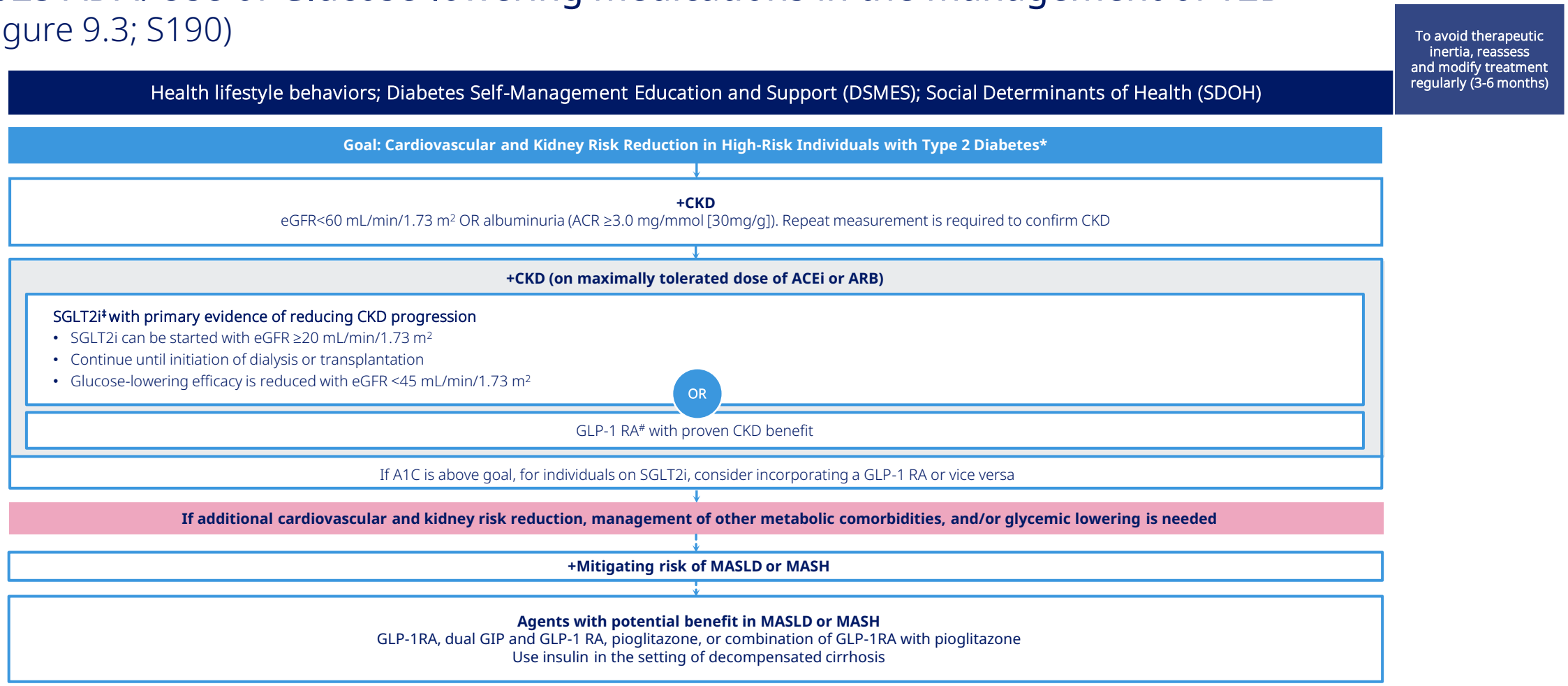

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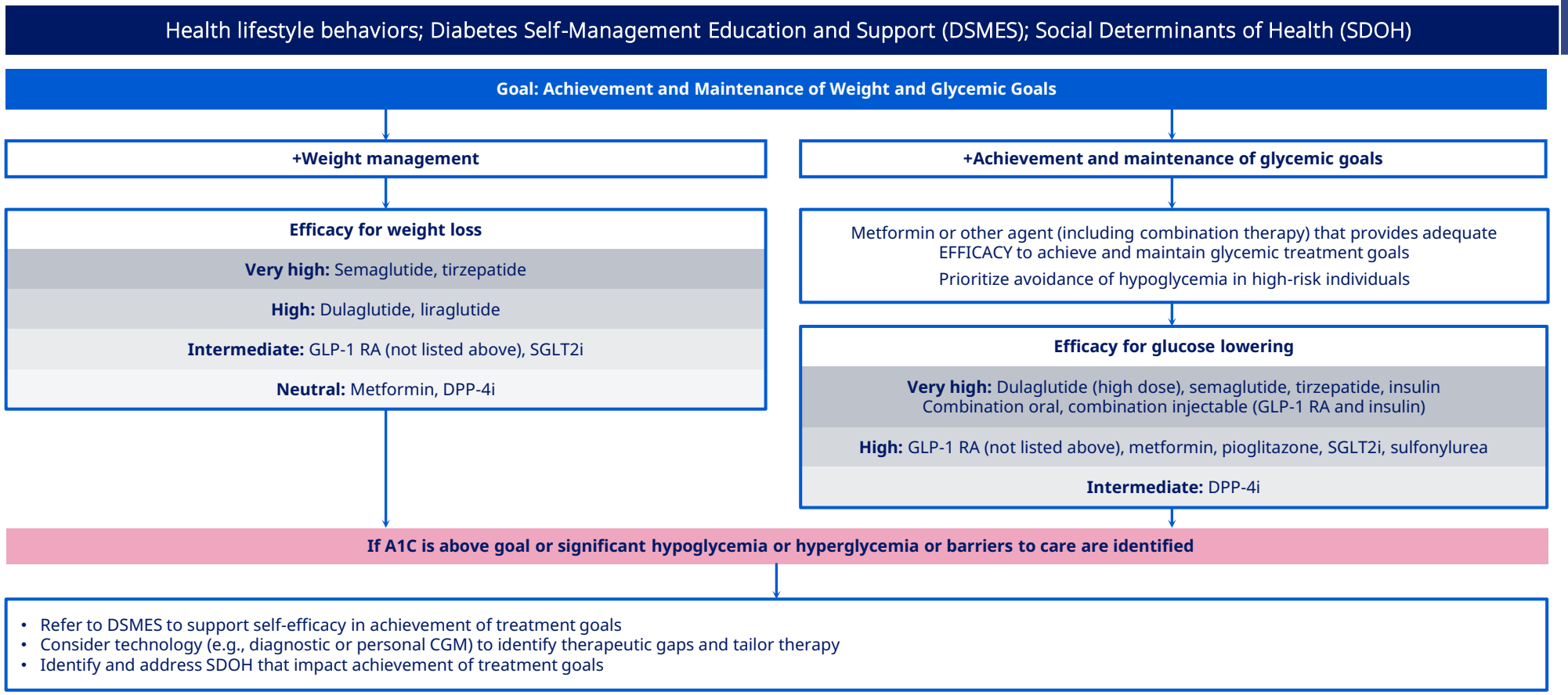
ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

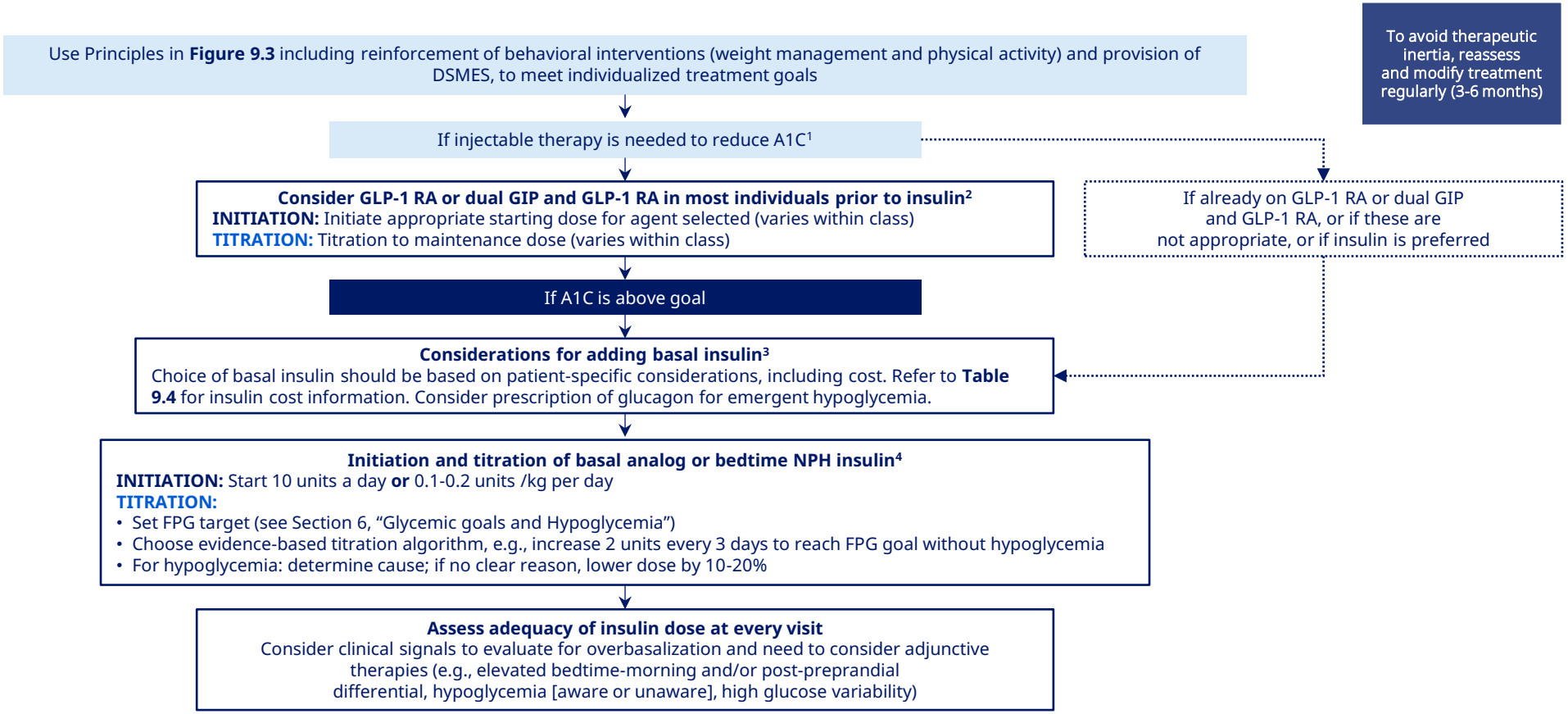
2025 ADA: Use of Glucose-lowering medications in the management of T2D (Figure 9.3; S190)

To avoid therapeutic inertia, reassess and modify treatment regularly (3-6 months)



Diabetes Care 2025;48(Suppl. 1):S181–S206 | doi: https://doi.org/10.2337/dc25-S009; Adapted from Davies et al Diabetes Care 2022;45:2753–2786

2025 ADA: Algorithm for intensifying to injectable therapies in T2D (1/2)
(Figure 9.4; S192)



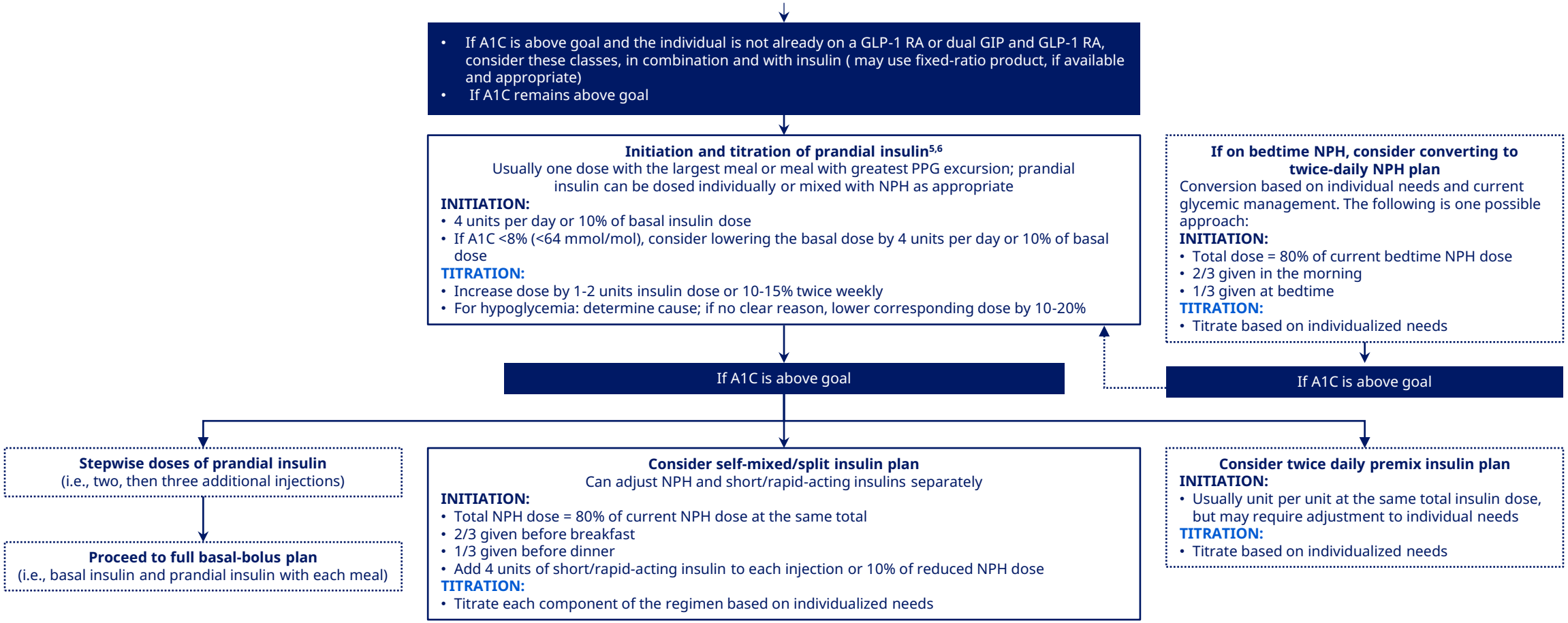
1. *Consider insulin as the first injectable if symptoms of hyperglycemia are present, when A1C or blood glucose levels are very high(i.e. AIC (>10% [86 mmol/moll) and blood glucose ≥300 mg/dL [≥16.7 mmol/L]), or a diagnosis of type 1 diabetes is a possibility.*
2. *When selecting GLP-1 RAs, consider individual preference, A1C lowering, weight-lowering effect, and frequency of injection. If CVD, is present, consider GLP-1 RA with proven CVD benefit; oral or injectable GLP-1 RAs are appropriate.*
3. *For people on GLP-1 RA and basal insulin combination, consider use of a fixed-ratio combination product (iDegLira or iGlarLixi).*
4. *Consider switching from evening NPH to a basal analog if the patient develops hypoglycemia and/or frequently forgets to administer NPH in the evening and would be better managed with a morning dose of a long-acting basal insulin. . Consider dosing NPH in the morning for steroid-induced hyperglycemia*

A1C, glycated hemoglobin; CVD, cardiovascular disease; DSMES, diabetes self-management education and support; FPG, fasting plasma glucose; GLP-1 RA, glucagon-like peptide 1 receptor agonist; GIP, glucose-dependent insulinotropic peptide; NPH, Neutral Protamine Hagedorn

Diabetes Care 2025;48(Suppl. 1):S181–S206 | doi: https://doi.org/10.2337/dc25-S009

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

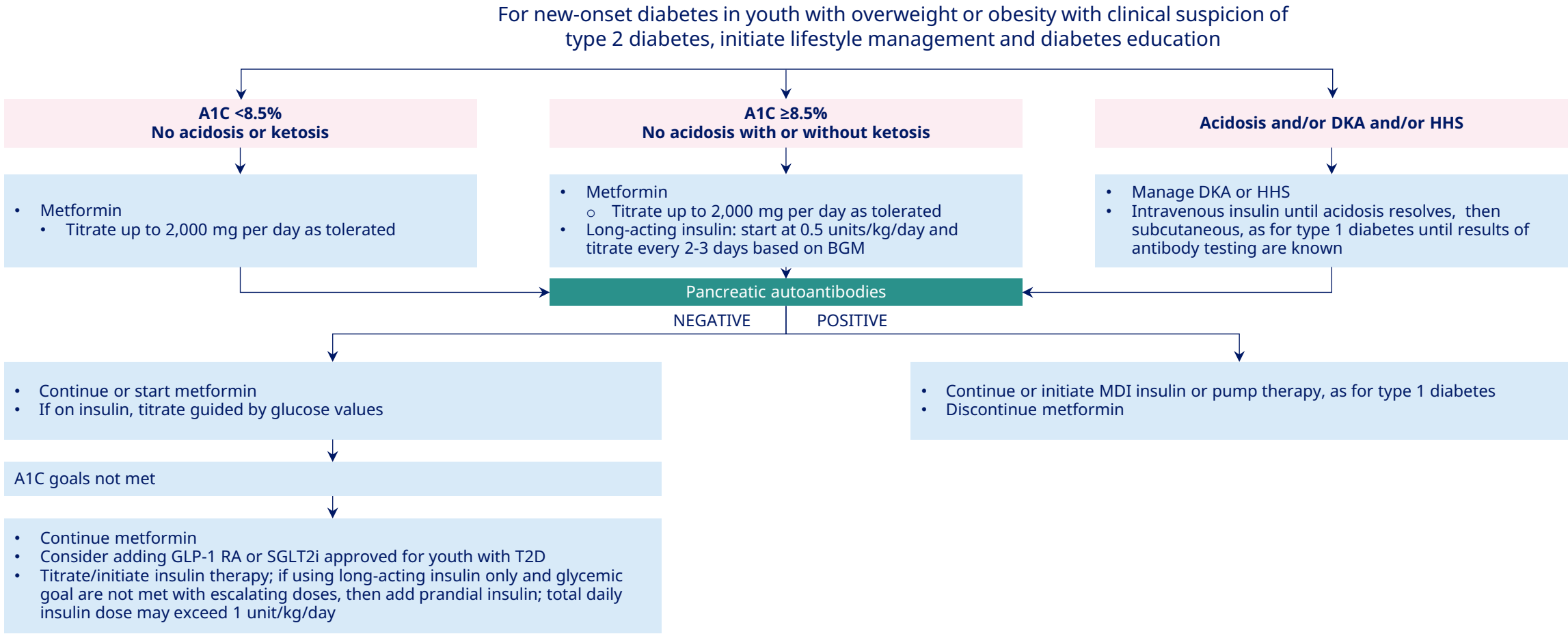
2025 ADA: Algorithm for intensifying to injectable therapies in T2D (2/2)
(Figure 9.4; S192)



5. Prandial insulin options include injectable rapid- and ultra-rapid-acting analog insulins, injectable short-acting human insulin, or inhaled human insulin.
6. If adding prandial insulin to NPH, consider initiation of a self-mixed or premixed insulin plan to decrease the number of injections required.
A1C, glycated hemoglobin; GLP-1 RA, glucagon-like peptide 1 receptor agonist; GIP, glucose-dependent insulinotropic peptide; NPH, Neutral Protamine Hagedorn; PPG, postprandial glucose
Diabetes Care 2025;48(Suppl. 1):S181–S206 | doi: https://doi.org/10.2337/dc25-S009

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

2025 ADA: Management of new-onset diabetes in youth with overweight or obesity with clinical suspicion of type 2 diabetes (Figure 14.1; S295)

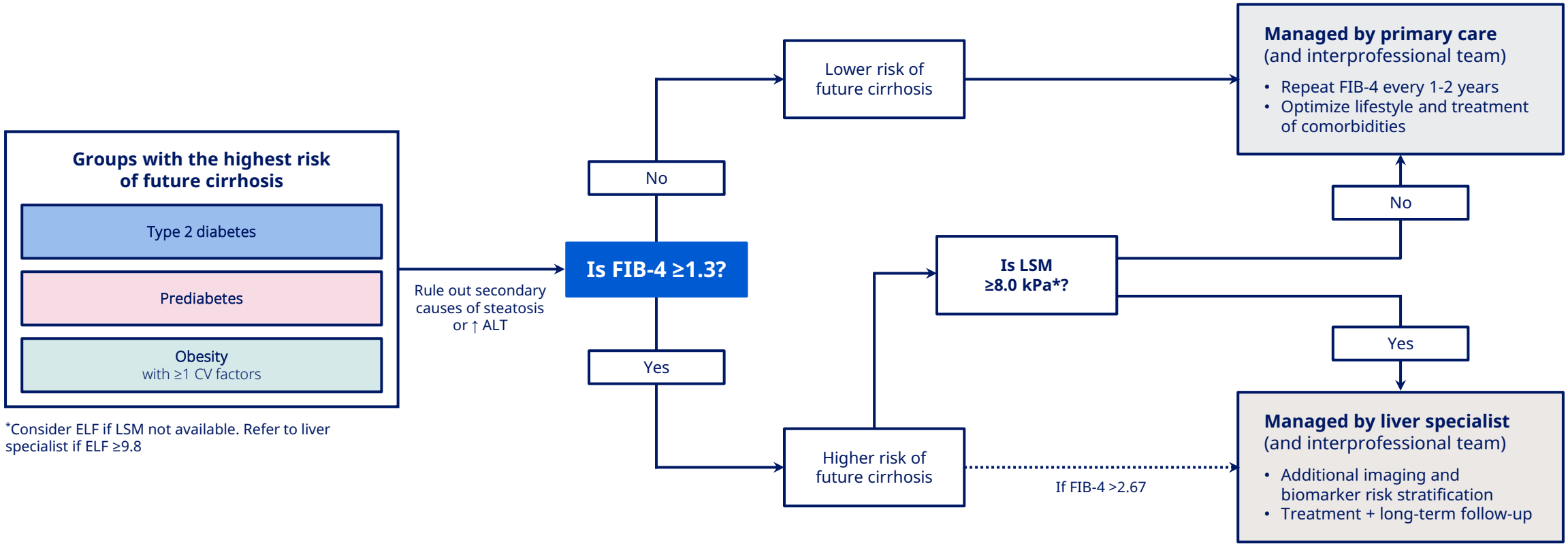


A1C 8.5% = 69 mmol/mol. Adapted from the ADA position statement Evaluation and Management of Youth-Onset Type 2 Diabetes[3]
BGM, blood glucose monitoring; CGM, continuous glucose monitoring; DKA, diabetic ketoacidosis; GLP-1, glucagon-like peptide 1; HHS, hyperosmolar hyperglycemic state; MDI, multiple daily injections; SGLT2, sodium–glucose cotransporter 2
Diabetes Care 2025;48(Suppl. 1):S283–S305 | doi: https://doi.org/10.2337/dc25-S014

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

2025 ADA: Diagnostic Algorithm for the Prevention of Cirrhosis in People with Metabolic Dysfunction-Associated Steatotic Liver Disease (MASLD)

(Figure 4.2; S74)



In the absence of LSM, consider ELF a diagnostic alternative. If ELF ≥9.8, an individual is at high risk of metabolic dysfunction–associated steatohepatitis with advanced liver fibrosis (≥F3–F4) and should be referred to a liver specialist

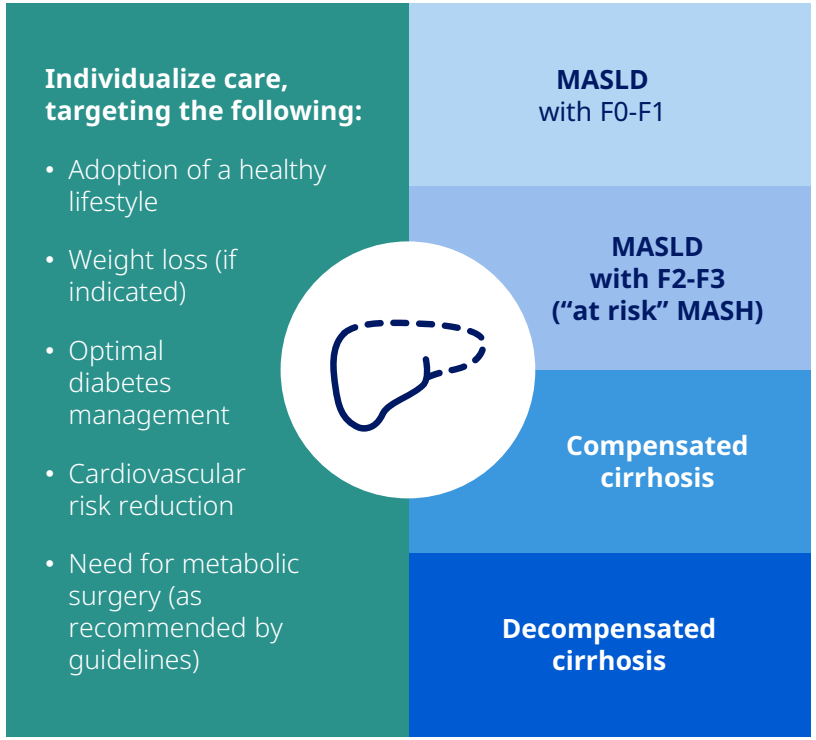
CV, cardiovascular; ELF, enhanced liver fibrosis test; FIB-4, fibrosis-4 index; LSM, liver stiffness measurement, as measured by vibration-controlled transient elastography

Diabetes Care 2025;48(Suppl. 1):S59–S85 | doi: https://doi.org/10.2337/dc25-S004

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

ADA 2025: Metabolic Dysfunction–Associated Steatotic Liver Disease (MASLD) Treatment Algorithm
(Figure 4.3; S76)

Metabolic Dysfunction–Associated Steatotic Liver Disease (MASLD) Treatment Algorithm



Obesity pharmacotherapy	Diabetes pharmacotherapy	MASH pharmacotherapy
Prefer GLP-1 RA, dual GIP and GLP-1 RA	Prefer GLP-1 RA, dual GIP and GLP-1 RA, pioglitazone, SGLT2i	Not indicated

Obesity pharmacotherapy	Diabetes pharmacotherapy	MASH pharmacotherapy
Prefer GLP-1 RA, dual GIP and GLP-1 RA	Prefer GLP-1 RA, dual GIP and GLP-1 RA, pioglitazone	Resmetirom

Obesity pharmacotherapy	Diabetes pharmacotherapy	MASH pharmacotherapy
As with F2-F3 with caution*	As with F2-F3 with caution*	! AVOID

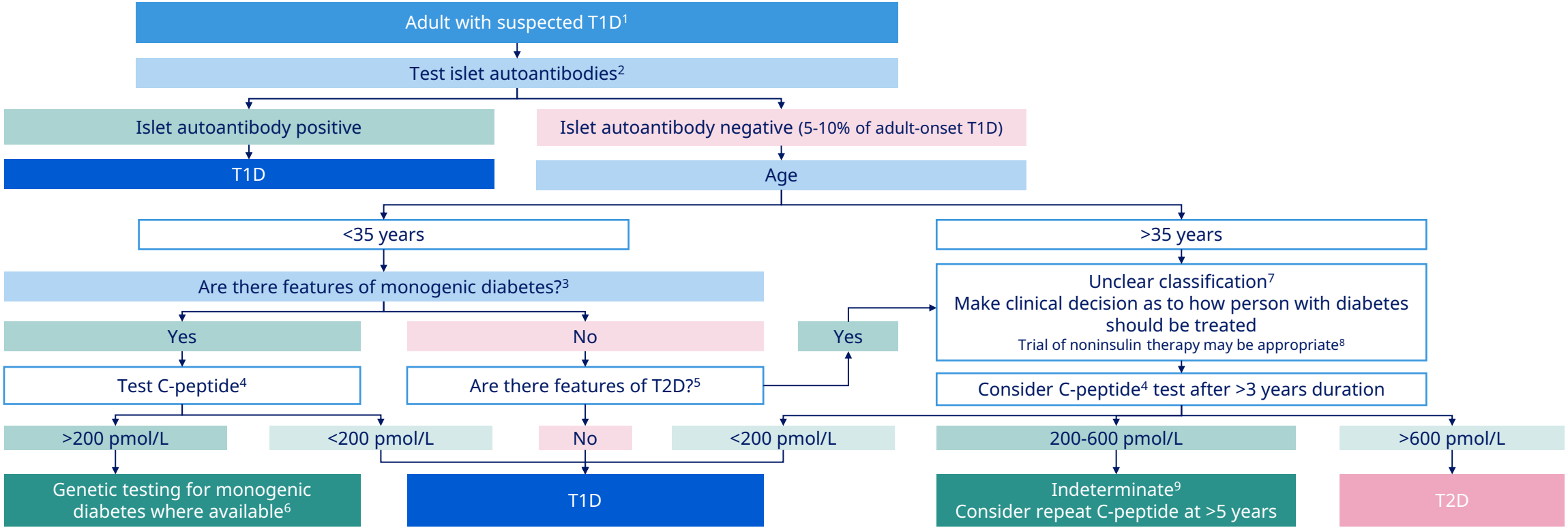
Obesity pharmacotherapy	Diabetes pharmacotherapy	MASH pharmacotherapy
! AVOID	Only use insulin	! AVOID

**Individualized care and close monitoring needed in compensated cirrhosis given limited safety data available.*
F0-F1, no to minimal fibrosis; F2-F3, moderate fibrosis; F4, cirrhosis; GIP, glucose-dependent insulinotropic polypeptide; GLP-1 RA, glucagon-like peptide 1 receptor agonist; MASH, metabolic dysfunction–associated steatohepatitis; SGLT2i, sodium–glucose cotransporter 2 inhibitor.
Diabetes Care 2025;48(Suppl. 1):S59–S85 | doi: https://doi.org/10.2337/dc25-S004

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

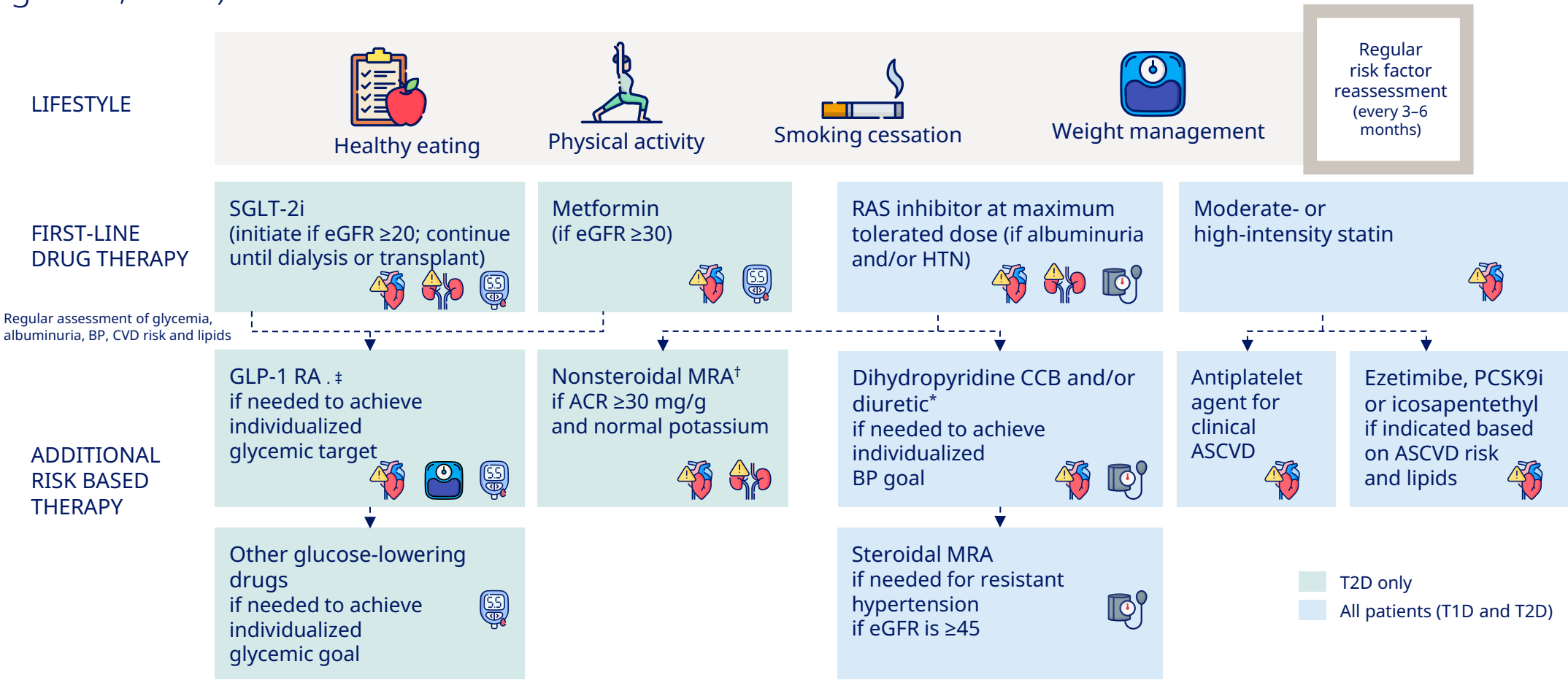
Flowchart for investigation of suspected type 1 diabetes in newly diagnosed adults, based on data from White European populations

(Fig. 2.1; S31)



[1]No single clinical feature confirms T1D in isolation. [2]Glutamic acid decarboxylase (GAD) should be the primary antibody measured and, if negative, should be followed by islet tyrosine phosphatase 2 (IA-2) and/or zinc transporter 8 (ZnT8) where these tests are available. In individuals who have not been treated with insulin, antibodies against insulin may also be useful. In those diagnosed at <35 years of age who have no clinical features of T2D or monogenic diabetes, a negative result does not change the diagnosis of T1D, since 5–10% of people with T1D do not have antibodies. [3]Monogenic diabetes is suggested by the presence of one or more of the following features: A1C <58 mmol/mol (<7.5%) at diagnosis, one parent with diabetes, features of a specific monogenic cause (e.g., renal cysts, partial lipodystrophy, maternally inherited deafness, and severe insulin resistance in the absence of obesity), and monogenic diabetes prediction model probability >5% (diabetesgenes.org/exeter-diabetes-app/ModyCalculator). [4]A C-peptide test is only indicated in people receiving insulin treatment. A random sample (with concurrent glucose) within 5 h of eating can replace a formal C-peptide stimulation test in the context of classification. If the result is ≥600 pmol/L (≥ 1.8 ng/mL), the circumstances of testing do not matter. If the result is <600 pmol/L (<1.8 ng/mL) and the concurrent glucose is <4 mmol/L (<70 mg/dL) or the person may have been fasting, consider repeating the test. Results showing very low levels (e.g., <80 pmol/L [<0.24 ng/mL]) do not need to be repeated. Where a person is insulin treated, C-peptide must be measured prior to insulin discontinuation to exclude severe insulin deficiency. Do not test C-peptide within 2 weeks of a hyperglycemic emergency. [5]Features of type 2 diabetes include increased BMI (≥ 25 kg/m2), absence of weight loss, absence of ketoacidosis, and less marked hyperglycemia. Less discriminatory features include non-White ethnicity, family history, longer duration and milder severity of symptoms prior to presentation, features of the metabolic syndrome, and absence of a family history of autoimmunity. [6]If genetic testing does not confirm monogenic diabetes, the classification is unclear and a clinical decision should be made about treatment. 7T2D should be strongly considered in older individuals. In some cases, investigation for pancreatic or other types of diabetes may be appropriate. [8]A person with possible T1D who is not treated with insulin will require careful monitoring and education so that insulin can be rapidly initiated in the event of glycemic deterioration. 9C-peptide values 200–600 pmol/L (0.6–1.8 ng/mL) are usually consistent with T1D or maturity-onset diabetes of the young but may occur in insulin-treated T2D, particularly in people with normal or low BMI or after long duration. Reprinted and adapted from Holt et al. Diabetes Care. 2021; 44:2589-2625.
T1D, type 1 diabetes; T2D, type 2 diabetes;
Diabetes Care 2025;48(Suppl. 1):S27–S49 | doi: https://doi.org/10.2337/dc25-S002

ADA 2025: Holistic approach for improving outcomes in patients with diabetes and CKD (Fig. 11.2; S245)



*ACEi or ARB (at maximal tolerated doses) should be first-line therapy for HTN when albuminuria is present. Otherwise, CCB or diuretic can also be considered; all 3 classes are often needed to attain BP targets. eGFR is presented in units of mL/min/1.73m2
†Finerenone is currently the only ns-MRA with proven clinical kidney and cardiovascular benefits. ‡Semaglutide can be used as another first-line agent for people with CKD.
ACEi, angiotensin-converting enzyme inhibitor; ACR, albumin-to creatinine ratio; ARB, angiotensin receptor blocker; ASCVD, atherosclerotic cardiovascular disease; BP, blood pressure; CCB, calcium channel blocker; CVD, cardiovascular disease; eGFR, estimated glomerular filtration rate; GLP-1 RA, glucagon-like peptide 1 receptor agonist; HTN, hypertension; MRA, mineralocorticoid receptor antagonist; ns-MRA, nonsteroidal mineralocorticoid receptor antagonist; PCSK9i, proprotein convertase subtilisin/kexin type 9 inhibitor; RAS, renin-angiotensin system; SGLT2i, sodium–glucose cotransporter 2 inhibitor; T1D, type 1 diabetes; T2D, type 2 diabetes
Diabetes Care 2025;48(Suppl. 1):S239–S251 | doi: https://doi.org/10.2337/dc25-S011

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

Decision cycle for person-centered glycemic management in T2D
(Fig. 4.1; S60)

Review and agree on management plan
- Review management plan
- Mutually agree on changes
- Ensure agreed modification of therapy is implemented in a timely fashion to avoid therapeutic inertia
- Undertake decision cycle regularly (at least once or twice a year)
- Operate in an integrated system of care

Provide ongoing support and monitoring of
- Emotional well-being
- Lifestyle and health behaviors
- Tolerability of medications
- Biofeedback including BGM/CGM, weight, step count, A1C, blood pressure, lipids

Implement management plan
- Ensure there is regular review; more frequent contact initially is often desirable for DSMES

Agree on management plan
Specify SMART goals:
- **S**pecific
- **M**easurable
- **A**chievable
- **R**ealistic
- **T**ime limited

Assess key person characteristics
- The individual's priorities
- Current lifestyle and health behaviors
- Comorbidities (i.e., CVD, CKD, HF)
- Clinical characteristics (i.e., age, A1C, weight)
- Issues such as motivation, depression and cognition
- Social determinants of health

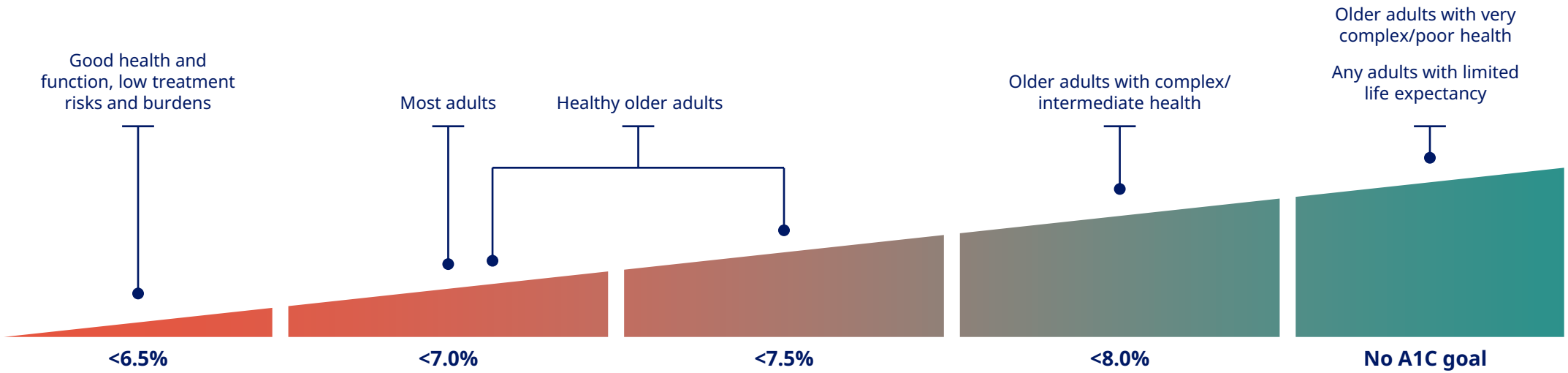
Consider specific factors that impact choice of treatment
- Individualized glycemic and weight goals
- Impact on weight, hypoglycemia, and cardiovascular and kidney protection
- Underlying physiological factors
- Side effect profiles of medications
- Complexity of treatment plan, i.e., frequency, mode of administration
- Treatment choice to optimize medication use and reduce treatment discontinuation
- Access, cost, availability of medication and lifestyle choices

Use shared decision-making to create a management plan
- Ensures access to DSMES
- Involves an educated and informed person (and the individual's family/caregiver)
- Explore personal preferences
- Language matters (include person-first, strengths based, empowering language
- Includes motivational interviewing, goal setting, and shared decision-making

; BGM, blood glucose monitoring; CGM, continuous glucose monitoring; CKD, Chronic Kidney Disease; CVD, cardiovascular disease; DSMES, Diabetes Self-Management Education and Support; HbA1c, glycated hemoglobin; HF, Heart Failure; SMBG, self measured blood glucose; T2D, type 2 diabetes;
Adapted from Davies et al (324)
Diabetes Care 2025;48(Suppl. 1):S59–S85 | doi: https://doi.org/10.2337/dc25-S004

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

ADA 2025: Individualized A1C goals for nonpregnant adults
(Fig. 6.2; S133)



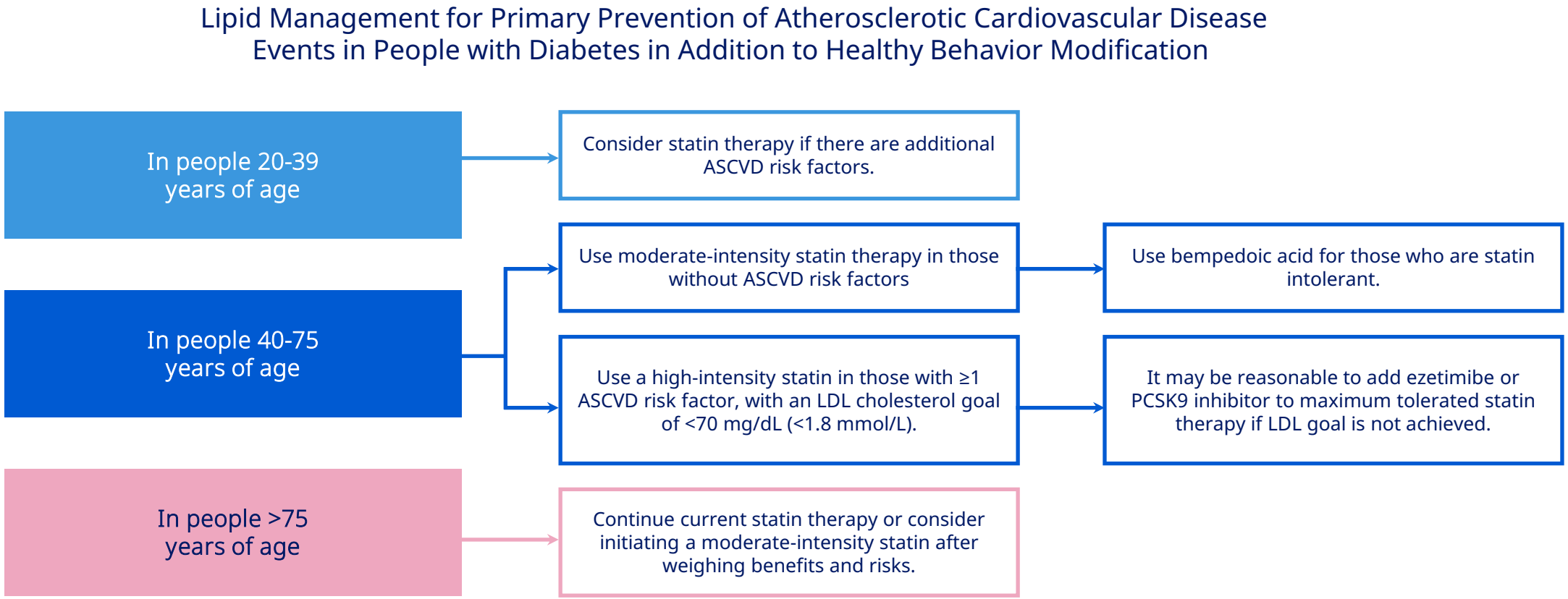
Modifying Factors

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

Consider modifying to a more or less stringent goal according to the factors listed in the table. Older adults are classified as healthy (few coexisting chronic illnesses, intact cognitive and functional status), as having complex/intermediate health (multiple coexisting chronic illnesses, two or more instrumental impairments to activities of daily living, or mild to moderate cognitive impairment), or as having very complex/poor health (long-term care or end-stage chronic illnesses, moderate to severe cognitive impairment, or two or more impairments to activities of daily living). Select glycemic goals that avoid symptomatic hypoglycemia and hyperglycemia in all individuals. Consider individuals' resources and support systems to safely achieve glycemic goals. Incorporate the preferences and goals of people with diabetes through shared decision-making.

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

ADA 2025: Lipid Management for Primary Prevention of Atherosclerotic Cardiovascular Disease Events in People With Diabetes in Addition to Healthy Behavior Modification (Fig.10.3, S216)

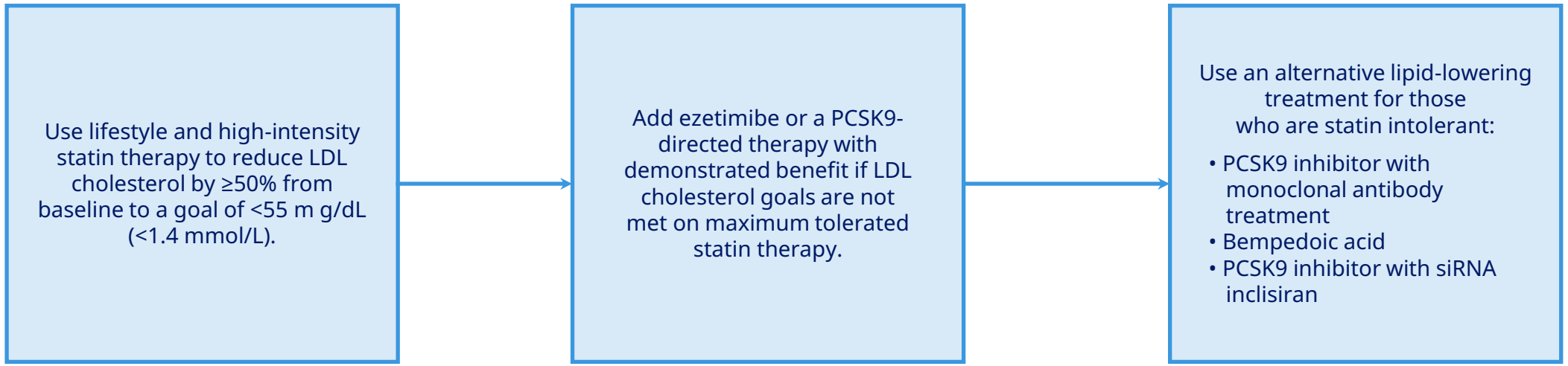


ASCVD, atherosclerotic cardiovascular disease; LDL, low density lipoprotein; PCSK9, proprotein convertase subtilisin/kexin type 9
Adapted from "Standards of Care in Diabetes—2024 Abridged for Primary Care Professionals" (325 Diabetes Care 2025;48(Suppl. 1):S207–S238 | doi: https://doi.org/10.2337/dc25-S010

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

ADA 2025: Lipid Management for Secondary Prevention of Atherosclerotic Cardiovascular Disease Events in People With Diabetes

(Fig.10.4, S217)

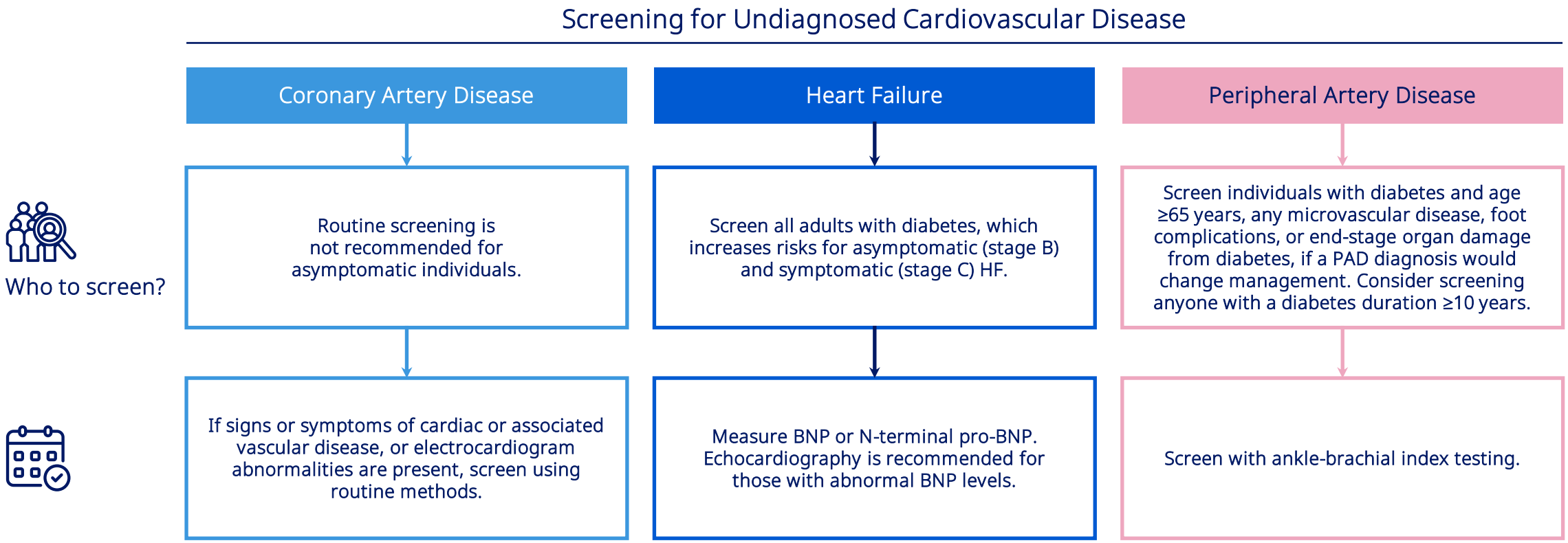


LDL, low density lipoprotein; PCSK9, proprotein convertase subtilisin/kexin type 9, siRNA, small interfering ribonucleic acid
Adapted from "Standards of Care in Diabetes—2024 Abridged for Primary Care Professionals" (325).
Diabetes Care 2025;48(Suppl. 1):S207–S238 | doi: https://doi.org/10.2337/dc25-S010

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

ADA 2025: Recommendations for screening of asymptomatic and undiagnosed cardiovascular disease

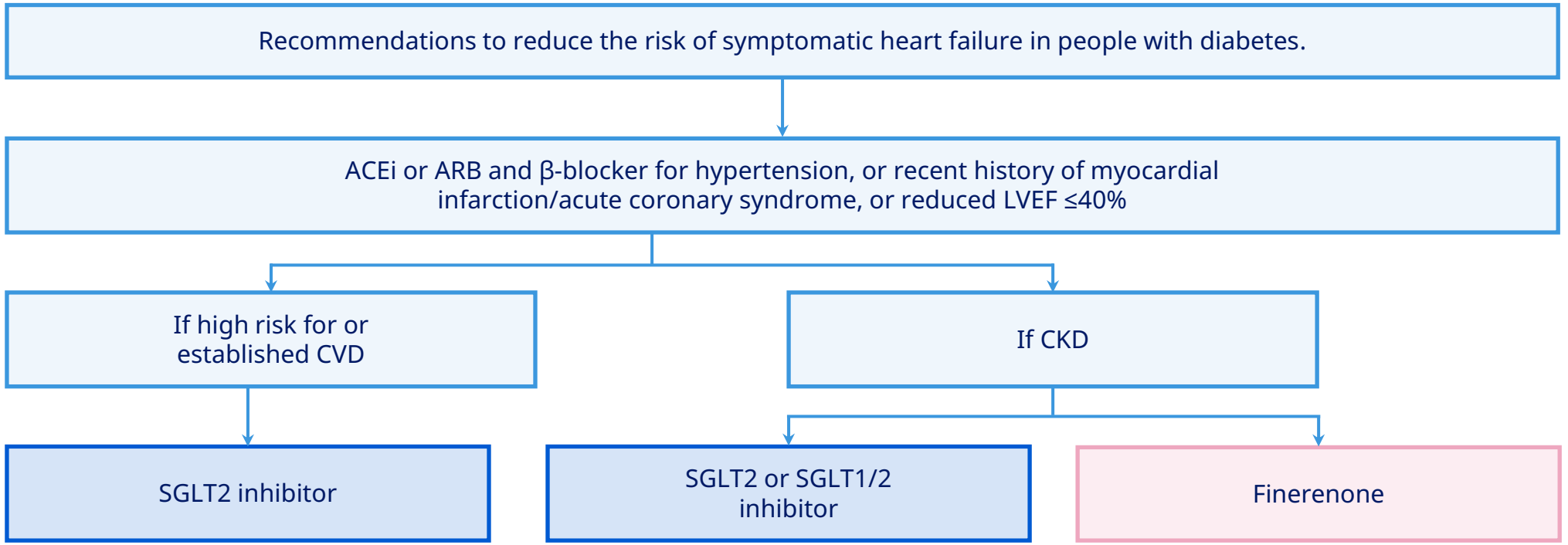
(Fig.10.5, S224)



BNP, B-type natriuretic peptide; HF, heart failure; PAD, peripheral artery disease. Adapted from "Standards of Care in Diabetes—2024 Abridged for Primary Care Professionals" (325)
Diabetes Care 2025;48(Suppl. 1):S207–S238 | doi: https://doi.org/10.2337/dc25-S010

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

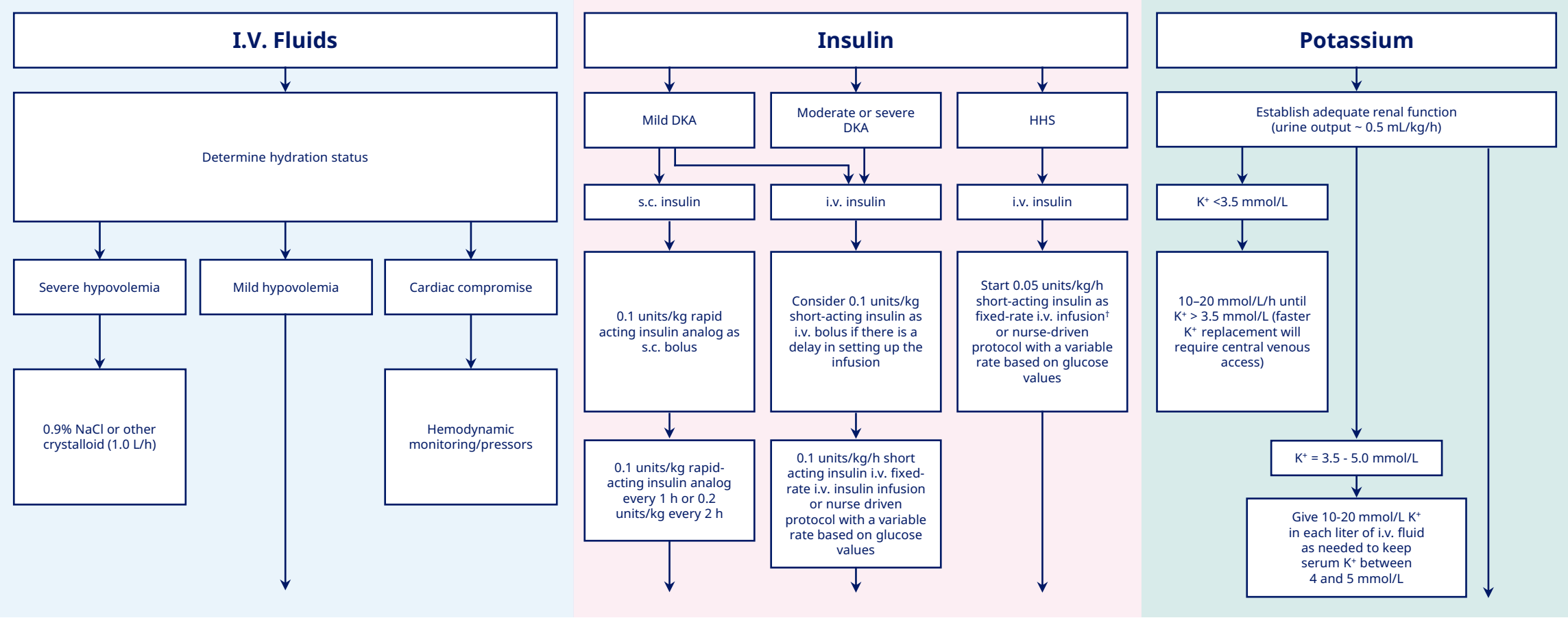
ADA 2025: Overview of recommendations for the prevention of the development of symptomatic heart failure in people with diabetes
(Fig.10.6, S226)



ACEi, ACE inhibitor; ARB, angiotensin receptor blocker; CKD, chronic kidney disease; CVD, cardiovascular disease; LVEF, left ventricle ejection fraction; SGLT2, sodium–glucose cotransporter 2. Adapted from "Standards of Care in Diabetes—2024 Abridged for Primary Care Professionals" (325) Diabetes Care 2025;48(Suppl. 1):S207–S238 | doi: https://doi.org/10.2337/dc25-S010

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

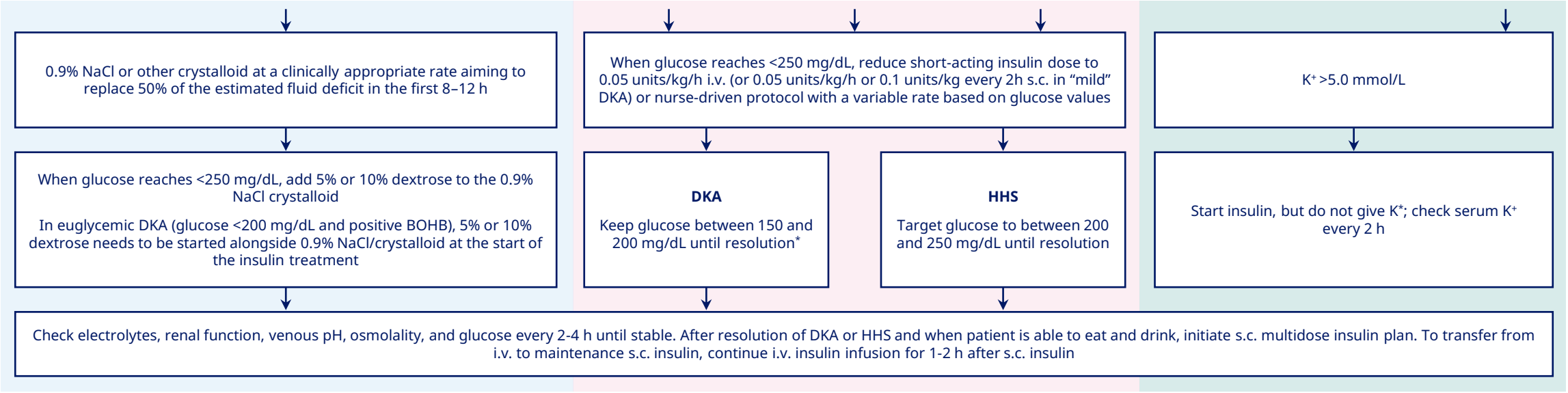
ADA 2025: Treatment pathways for diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) (1/2)
(Fig.16.1, S329)



+ Some have recommended that insulin be withheld until glucose has stopped dripping with fluid administration alone
NaCl, sodium chloride; K, potassium; IV, intravenous, s.c subcutaneous
Diabetes Care 2025;48(Suppl. 1):S321–S334 | doi: https://doi.org/10.2337/dc25-S016

ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

ADA 2025: Treatment pathways for diabetic ketoacidosis (DKA) and hyperglycemic hyperosmolar state (HHS) (2/2)
(Fig.16.1, S329)



* Definitions of resolution (use clinical judgment and do not delay discharge or level of care if these are not met):
- > **DKA**: Venous pH >7.3 or bicarbonate >18 mmol/L and plasma/capillary ketones <0.6 mmol/L
- > **HHS**: Calculated serum osmolality falls to <300 mOsm/ kg and urine output is >0.5 mL/kg/h and glucose is <250 mg/dL

150 mg/dL = 8.3 mmol/L
200 mg/dL = 11.0 mmol/L
250 mg/dL = 13.9 mmol/L
300 mg/dL = 16.6 mmol/L

! Bicarbonate should only be considered if pH is <7.0

! Phosphate should not be given unless there is muscle weakness, respiratory compromise, and a phosphate <1.0 mmol/L

BOHB, b-hydroxybutyrate. Adapted from Umpierrez et al. Diabetes Care 2024;47:1257–1275.
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Features of medications for lowering glucose in type 2 diabetes (1/2) Table 9.2; S191

Medication (route of administration)	Glucose lowering efficacy[1]	Hypoglycemia risk	Weight effects[2]	CV effects		Kidney effects		MASH Effects
				Effect on MACE	Effect on HF	Progression of CKD	Dosing / Use considerations*	
Metformin (oral)	High	No	Neutral (potential for modest loss)	Potential benefit	Neutral	Neutral	Contraindicated with eGFR <30 mL/min/1.73 m^2	Neutral
SGLT2 inhibitors (oral)	Intermediate to high	No	Loss (intermediate)	Benefit: canagliflozin, empagliflozin	Benefit: canagliflozin, dapagliflozin, empagliflozin, ertugliflozin	Benefit: canagliflozin, dapagliflozin, empagliflozin	• See labels of individual agents for dosage considerations for kidney function • Glucose-lowering effect is minimal at eGFR <45 mL/min/1.73 m^2 and lower; continue for cardio-vascular and kidney benefit until dialysis or transplantation	Unknown
GLP-1 RAs (SQ; semaglutide also available in oral formulation)	High to very high	No	Loss (intermediate to very high)	Benefit: dulaglutide, liraglutide, semaglutide (SQ)	Neutral	Benefit for renal end points in CVOTs, driven by albuminuria outcomes: dulaglutide, liraglutide, semaglutide (SQ)	• See labels of individual agents for dosage considerations for kidney function • No dose adjustment for dulaglutide, liraglutide, or semaglutide • Monitor kidney function when initiating or escalating doses in individuals with kidney impairment reporting severe adverse GI reactions	Potential benefit
				Neutral: exenatide once weekly, lixisenatide		Demonstrated benefit for progression of CKD for semaglutide (SQ)		
Dual GIP and GLP-1 RA (SQ)	Very high	No	Loss (very high)	Under investigation	Under investigation	Under investigation	• See labels of individual agents for dosage considerations for kidney function • No dose adjustment • Monitor kidney function when initiating or escalating doses in individuals with kidney impairment reporting severe adverse GI reactions	Potential benefit

*CKD, chronic kidney disease; CV, cardiovascular; CVOT, cardiovascular outcomes trial; DKA, diabetic ketoacidosis; DPP-4, dipeptidyl peptidase 4; eGFR, estimated glomerular filtration rate; FDA, U.S. Food and Drug Administration; GI, gastrointestinal; GIP, glucose-dependent insulinotropic polypeptide; GLP-1 RA, glucagon-like peptide 1 receptor agonist; HF, heart failure; MACE, major adverse cardiovascular events; MASH, metabolic dysfunction-associated steatohepatitis; SGLT2, sodium–glucose cotransporter 2; SQ, subcutaneous; T2D, type 2 diabetes. *For agent-specific dosing recommendations, please refer to manufacturers' prescribing information. 1Tsapas et al. (106). 2Tsapas et al. (241). Adapted from Davies et al. (89).*

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ADA STANDARDS OF MEDICAL CARE IN DIABETES – 2025

Features of medications for lowering glucose in type 2 diabetes (2/2) Table 9.2; S191

Medication (route of administration)	Glucose lowering efficacy[1]	Hypoglycemia risk	Weight effects[2]	CV effects		Kidney effects		MASH Effects
				Effect on MACE	Effect on HF	Progression of CKD	Dosing / Use considerations*	
DPP-4 inhibitors (oral)	Intermediate	No	Neutral	Neutral	Neutral (potential risk: saxagliptin)	Neutral	• Dose adjustment required based on kidney function (sitagliptin, saxagliptin, alogliptin) • No dose adjustment required for linagliptin	Unknown
Pioglitazone (oral)	High	No	Gain	Potential benefit	Increased risk	Neutral	• No dose adjustment required • Generally, not recommended in kidney impairment due to potential for fluid retention	Potential benefit
Sulfonylureas (2nd generation) (oral)	High	Yes	Gain	Neutral	Neutral	Neutral	• Glyburide: generally, not recommended in CKD • Glipizide and glimepiride: initiate conservatively to avoid hypoglycemia	Unknown
Insulin (human) (SQ; regular insulin also available as inhaled formulation)	High to very high	Yes	Gain	Neutral	Neutral	Neutral	• Lower insulin doses required with a decrease in eGFR; titrate per clinical response	Unknown
Insulin (analogs) (SQ)								

*CKD, chronic kidney disease; CV, cardiovascular; DKA, diabetic ketoacidosis; DPP-4, dipeptidyl peptidase 4; eGFR, estimated glomerular filtration rate; FDA, U.S. Food and Drug Administration; GI, gastrointestinal; GIP, glucose-dependent insulinotropic polypeptide; GLP-1 RA, glucagon-like peptide 1 receptor agonist; HF, heart failure; MACE, major adverse cardiovascular events; MASH, metabolic dysfunction-associated steatohepatitis; SGLT2, sodium–glucose cotransporter 2; SQ, subcutaneous; T2D, type 2 diabetes. *For agent-specific dosing recommendations, please refer to manufacturers' prescribing information. 1Tsapas et al. (106). 2Tsapas et al. (241). Adapted from Davies et al. (89).*

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