**Diabetes:**

Reference source: Canadian Diabetes Association 2008 Clinical Practice Guidelines <http://www.diabetes.ca/files/cpg2008/cpg-2008.pdf>

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| **1. Given a symptomatic or asymptomatic patient at high risk for diabetes (e.g., patients with gestational diabetes (GDM), obese, certain ethnic groups, and those with a strong family history), screen at appropriate intervals with the right tests to confirm the diagnosis.**  Risk factors for type 2 diabetes:   * Age ≥ 40 years * T2DM in a first degree relative * High risk population (Aboriginal, Hispanic, South Asian, Asian, African descent) * History of impaired glucose tolerance or impaired fasting glucose * Presence of complications associated with diabetes * Vascular disease * History gestational diabetes * History of delivery of a macrosomic infant * Hypertension * Dyslipidemia * Overweight * Abdominal obesity * Polysyctic Ovarian Syndrome * Acanthosis nigricans * Schizophrenia * Other (see Canadian Diabetes Association 2008 Clinical Practice Guidelines – Appendix 1)   Screening recommendations:   * No screening for type 1 diabetes - no evidence for interventions to prevent or delay disease * For all adults ≥ 40 years, perform fasting plasma glucose (FPG) every 3 years * Consider testing more often and/or earlier in those with risk factors. * FPG of 6.1 to 6.9 🡪 75g oral glucose tolerance test (OGTT) * FPG of 5.6 to 6.0 + risk factors 🡪75g OGTT * Patients with GDM should have a 75g OGTT at 6 weeks to 6 months postpartum * Those diagnosed with GDM are considered high risk, consider screening earlier/more often and screen prior to subsequent pregnancy.   \*See flow diagram on page S15 of the Canadian Diabetes Association 2008 Clinical Practice Guidelines or BC Clinical Practice Guidelines on Diabetes Care – Appendix A |
| **2. Given a patient diagnosed with diabetes, either new-onset or established, treat and modify treatment according to disease status (e.g., use oral hypoglycemic agents, insulin, diet, and/or lifestyle changes).**  Optimal diabetes care ultimately depends on the daily commitment of the individual to self manage, with support from a multidisciplinary team. High importance placed on self management education and individualized treatment. In addition to diet, nutrition and exercise, smoking cessation should be encouraged and supported.  Diet: Should be referred to a dietician.   * Follow Eating well with Canada’s Food Guide. * Meal and carbohydrate regularity and spacing is important * Carbohydrate 45-60% of energy and choose complex carbohydrates * Protein 15-20% of energy * Fat <15% of energy * Alcohol has risk of delayed hypoglycaemia if using insulin or insulin secretagogues. Limit to 1-2 drinks per day (≤ 12 per week for men, ≤9 per week for women) * See page S43 of Canadian Diabetes Association 2008 Clinical Practice Guidelines   Exercise:   * Those who have been previously sedentary should be screen appropriately prior to engaging in any activity more strenuous than a brisk walk. Exercise should be started slowly, even with 5-10 mins per day. Several short sessions per day may be easier than one extended session * Goal = moderate to vigorous aerobic exercise for at least 150 mins per week, divided in at least 3 session, with never more than 2 days off at a time. * Goal = resistance exercise at least 3 times per week, even in the elderly * See page S38 Canadian Diabetes Association 2008 Clinical Practice Guidelines for definitions and examples of appropriate aerobic and resistance exercise.   Pharmacologic management of Type 2 Diabetes:   * Treatment and targets should be individualized. General guidelines follow. * HgA1c <9% at time of diagnosis 🡪 2-3 month trial of lifestyle management 🡪 add anti-hyperglycemic pharmacotherapy if not at target after that time. * HgA1c ≥9% at time of diagnosis 🡪 lifestyle management + anti-hyperglycemics at same time, consider initiating combination therapy with 2 oral agents or starting insulin. * Goal is to attain HgA1c target within 6 to 12 months. Lag period before adding another agent should be kept to a minimum. * Metformin should be the initial drug used – for both overweight and non-overweight patients * Add additional agents from different classes if needed * Insulin may be added as a single injection of intermediate or long acting insulin at bedtime initially. As type 2 diabetes progresses, insulin doses may need to be increased and additional basal doses or prandial doses of short/rapid insulin may be needed. * Insulin may also be used temporarily during illness, pregnancy, stress or for a medical procedure or surgery. * HgA1c should be checked every 3 months, with ongoing adjustments to management throughout a patient’s life and the disease process. * Full descriptions of oral anti-hyperglycemic agents can be found on pages S54-S56 of Canadian Diabetes Association 2008 Clinical Practice Guidelines.   Insulin in Type 1 Diabetes:   * Basal-prandial regimens of either multiple daily injections or continuous subcutaneous insulin infusions (CSII) are the insulin regimens of choice for adults – attempt to duplicate normal pancreatic insulin production. * Basal insulin (intermediate or long acting) is given once or twice a day. * Prandial or bolus insulin (short or rapid acting) is given at each meal – takes into account amount and glycemic index of carbohydrate consumed and exercise around meals; can be used to correct hyperglycaemia. Choose rapid (aspart, lispro) over regular insulin to improve A1C and postprandial targets, while minimizing hypoglycaemia. Rapid insulin is used with CSII * Insulin induced HYPOGLYCEMIA COUNSELLING – risks, prevention, monitoring, and treatment – for everyone. * Details on insulin types, with duration, onset and peak of action can be found on page S47 of Canadian Diabetes Association 2008 Clinical Practice Guidelines. |
| **3. Given a patient with established diabetes, advise about signs and treatment of hypoglycemia/ hyperglycemia during an acute illness or stress (i.e., gastroenteritis, physiologic stress, decreased intake.** |
| Sick Day Guidelines from Fraser Health  Type I Diabetes: <http://www.fraserhealth.ca/media/Type%201%20Diabetes%20Sick%20Days%20Colour%20Sept%202009.pdf>  Type 2 Diabetes: |

<http://www.fraserhealth.ca/media/Sick%20Days%20Type%202%20Colour.pdf>

Acute illness:

* Hypoglycemia: May occur due to nausea, vomiting, and anorexia/poor oral intake.
* Hyperglycemia: May occur due to increased peripheral insulin resistance, stress of illness, failure to take insulin or other diabetic medications, dehydration or excess intake of sweet liquids. Result can be DKA or hyperosmolar hyperglycemias state.
* Increase frequency of blood glucose monitoring
* Basal insulin and oral medications should be continued, although dosage may need adjustment. Insulin must never be omitted because hyperglycemia and diabetic ketoacidosis can develop without basal insulin.
* Frequent blood glucose monitoring is mandatory- q4h
* If blood glucose ≥14 for more than 8 hours, test for ketones – urine or serum.
* Use a sick day sliding scale if extra insulin needed for hyperglycemia.
* Seek medical attention if: elevated blood glucose for more than 8 hours, with moderate to large urine ketones or serum ketones ≥1.5mmol/hr; unable to eat or drink and you take insulin and/or diabetic medication; diarrhea lasting >24hours.

Hypoglycemia: 1) Symptoms. 2) Plasma glucose <4.0mmol/L. 3) Responds to carbohydrate.

Hypoglycemia symptoms:

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| --- | --- |
| *Neurogenic (autonomic)* | *Neurogenic* |
| * + Trembling   + Palpitations   + Sweating   + Anxiety   + Nausea   + Tingling | * + Difficulty concentrating   + Confusion   + Weakness   + Drowsiness   + Vision changes   + Difficulty speaking   + Headache   + Dizziness |

Hypoglycemia Treatment:

* Mild to moderate hypoglycaemia: 15g carbohydrate to produce increase in plasma glucose of 2.1mmol/L at 20 mins with adequate symptom relief for most people 🡪 wait 15 mins and re-test 🡪 if remain <4mmol/L repeat 15g carbohydrate dose.
  + - 15g in form of glucose tablets
    - 15ml/3tsp or 3 packets of table sugar in water
    - 175ml/ ¾ cup juice or pop
    - 6 lifesavers (1=2.5g carbohydrate)
    - 15ml/1tbsp honey
* Severe hypoglycaemia + conscious: 20g carbohydrate 🡪 wait 15 mins and re-test 🡪 if remain <4mmol/L retreat with 15g carbohydrate.
* Severe hypoglycaemia + unconscious + >5 yrs old (at home): 1mg glucagon SC or IM (should have trained support person) 🡪 call EMS
* To prevent repeated episodes, have usual meal/snack for that time of day. If meal >1hr away, have snack with 15g carbohydrate + protein.
* Patients taking alpha-glucosidase inhibitors must use glucose/dextrose tablets, milk, or honey to treat hypoglycaemia.

Hyperglycemia (in acute illness):

* There is no specific number or length of time at which symptoms of hyperglycemia may develop. It varies between individuals.
* Symptoms experienced may be due to hyperglycemia alone ( hyperosmolar hyperglycaemic state)or a combination of hyperglycemia and acidosis (DKA)
* See following questions for diagnosis and treatment.

Hyperglycemia symptoms:

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| --- | --- |
| * Polyphagia * Polydipsia * Headache * fatigue | * Decreased concentration * Blurred vision * Weight loss |

Hyperglycemia treatment:

* Take medications and insulin as prescribed
* Used sick day sliding scale
* See questions below for DKA and HHS treatment

**4. In a patient with poorly controlled diabetes, use effective educational techniques to advise about the importance of optimal glycemic control through compliance, lifestyle modifications, and appropriate follow-up and treatment.**

* Non-adherence with diabetic treatment is a problem in primary care 🡪disease control is highly dependent on self management, supported by an integrated diabetes healthcare team, including:
  + Community based diabetes education
  + Family physician
  + Diabetic educators
  + Nurses
  + Dietitian
  + …most importantly…the patient
* Interventions focusing on modifying the behaviour of people living with diabetes have better outcomes than those focusing on modifying clinician's behavior.
* Comprehensive interventions combining cognitive, behavioural, and educational components (didactic and nondidactic) are more effective than single-focus interventions.
* Diabetes education must support *self-management* through informed, independent decisions relating to the individual’s diabetes management.
  + Use open questions (What do you know about...? What do you think about...?) when addressing issues (treatment, prevention of complications, blood glucose monitoring, diet, physical activity, foot care).
  + Include problem-solving, goal-setting and active participation of patients in decision-making. This includes support in interpreting and acting on the results of self-monitoring of blood glucose or making informed decisions about insulin.
  + Share with your patients his or her goals of care, e.g. “Ms. Dale, you have agreed to lose some weight. Tell me how much weight you would like to lose by our next visit, and then together we can discuss some strategies for achieving your goal.”
  + Discuss patients’ *expectations* and try to identify specific problems related to diabetes management.
* Self-management programs must be individualized, considering the type of diabetes, current control, treatment recommendations, learning ability, ability to change, resources and motivation.
* Education should be offered in a timely and needs-based manner. Interventions that include face-to-face delivery and practical application content are more likely to improve glycemic control.
* Family support is beneficial for people with diabetes 🡪involve family if possible.

**5. In patients with established diabetes: a) look for complications. b) refer as necessary to deal with complications.**

Diabetic Retinopathy:

* Prevalence in diabetic adults in the US is 40%; prevalence sight-threatening retinopathy is ~8%.
* Most have no symptoms until very late stages (too late for effective treatment).
* Types:
* *macular edema*: diffuse or focal vascular leakage at the macula;
* *nonproliferative*: progressive accumulation of blood vessel change, including microaneurysms, intraretinal hemorrhage, vascular tortuosity and vascular malformation
* *proliferative:* abnormal vessel growth;
* *retinal capillary closure*
* Treatment: retinal photocoagulation (laser therapy) and vitreoretinal surgery.
* Initial screening: Refer to an experienced ophthalmologist or optometrist
* In T1DM, those ≥15 yrs old should be screened q1yr, starting 5 years after diabetes onset .
* In T2DM, screen at the time of diagnosis, with follow-up tailored to the severity of retinopathy. If no or minimal retinopathy, screen q1-2yrs.

Chronic Kidney Disease (CKD):

* Found in 50% of people with diabetes
* CKD associated with diabetes is the leading cause of kidney failure in Canada.
* The degree of proteinuria is characterized as either:
  + microalbuminuria (urinary albumin 30 to 300 mg/day)
  + overt nephropathy (urinary albumin >300 mg/day)
* Screen for microalbuminuria with a random urine albumin to creatinine ratio (ACR). Transient microalbuminuria unrelated to diabetic nephropathy can occur, so at least 2 to 3 postive ACRs, over a period of about 2 months, should be demonstrated before the diagnosis of microalbuminuria is made.
* 24-h urine collections are not routinely recommended, BUT can be useful if in doubt about eGFR accuracy, when screening for non-albumin urinary proteins (e.g. multiple myeloma) or when estimating daily sodium intake in an individual with refractory edema or hypertension.
* Serum creatinine (Cr) is the most commonly used measure of renal function; however, Cr may falsely indicate function is normal.
  + May lose up to 50% of renal function before serum Cr becomes abnormal
  + eGFR is more sensitive in identifying low kidney function in diabetes. In Canada, eGFR is most often calculated using the abbreviated Modification of Diet in Renal Disease (MDRD) equation, which takes into account the person’s serum creatinine, age and sex.
* Please refer to the diagram below for CKD screening:
* Consider referral to a nephrologist or internist if:
  + chronic, progressive loss of kidney function;
  + eGFR is <30 mL/min;
  + ACR is persistently >60 mg/mmol;
  + unable to achieve BP targets
  + Unable to tolerate renal-protective therapies 🡪 hyperkalemia, >30% increase in serum Cr within 3 months of starting an ACE-I or ARB.

[[1]](#footnote-1)

Foot Complications:

* Major cause of morbidity and mortality in diabetes
* Contribute to increased healthcare costs
* With neuropathy or peripheral vascular disease, minor trauma to the foot can lead to skin ulceration, infection and gangrene, often resulting in amputation.
* Foot examination by the patient and healthcare providers is integral to decrease the risk of foot lesions and amputations, and should be performed at least annually, but more frequently in those at high risk.
* Assessment by healthcare providers should include structural abnormalities (ankle and toe ROM, callus pattern, bony deformities, skin temperatures), evaluation for neuropathy and peripheral arterial disease, ulceration and evidence of infection.
* People at high risk of foot ulceration and amputation should receive foot care education (including counseling to avoid foot trauma), professionally fitted footwear, smoking cessation strategies and early referrals to a foot care professionals for arising issues.
* Management of foot ulceration requires an interdisciplinary approach that addresses glycemic control, infection, lower-extremity vascular status and local wound care – to manage and prevent amputation and recurrent problems.

Erectile Dysfunction (ED):

* 34 to 45% of men with diabetes
* Negatively impacts quality of life across all ages
* May be the earliest sign of cardiovascular disease.
* Regularly screen all adult men with DM for ED with a sexual function history.
* Validated questionnaires (e.g. International Index of Erectile Function or Sexual Health Inventory for Men) have been shown to be both sensitive and specific in determining the presence of ED and assessing response to therapy.
* Treatment: PDE5 inhibitors, reported to have a major impact on erectile function, quality of life, and should be offered as first-line therapy to men with diabetes wishing treatment for ED.
* Referral to a specialist in ED should be offered to men who:
  + do not respond to PDE5 inhibitors
  + have contraindications to PDE5 inhibitors
  + Are interested in 2nd-line therapies (e.g. vacuum constriction devices, intracorporal injection therapy) or 3rd-line therapy (penile prosthesis)

Diabetic Neuropathy:

* Under-diagnosed in primary care, which impedes the benefits of early identification and management necessary to prevent neuropathy-related sequelae.
  + Detectable sensorimotor polyneuropathy will develop within 10 years of diabetes onset in 40 to 50% of people with type 1 or type 2 diabetes
* The most frequently encountered neuropathies include:
  + distal symmetric polyneuropathy;
  + autonomic neuropathy;
  + thoracic and lumbar nerve root disease, causing polyradiculopathies;
  + individual cranial and peripheral nerve involvement (mononeuropathies, especially affecting the oculomotor and the median nerve).
* Peripheral neuropathy screening: T2DM 🡪 should begin at the time of diagnosis and occur annually. T1DM 🡪 annual screening after 5 years’ post-pubertal diabetes. Conducted with 10g monofilament or sensitivity to vibration at the dorsum of the great toe.
* Intensive glycemic control is effective for primary or secondary prevention in T1DM. In T2DM, lower blood glucose levels are associated with reduced frequency of neuropathy.
* Multiple medications are available for neuropathic pain management:
  + Antidepressants, anticonvulsants, opioid analgesics and topical isosorbide dinitrate should be considered alone or in combination
* Refer if there are significant early progressive symptoms of neuropathy or clinical suspicion of non-diabetic neuropathy.
* Significant symptomatic autonomic neuropathy may require assessment by a specialist in the affected system.

Coronary Artery Disease:

* Those with diabetes (especially women) are at higher risk of developing heart disease, and at an earlier age.
  + Often asymptomatic before either a fatal or nonfatal myocardial infarction (MI).
  + It is helpful identify patients at high risk for vascular events.
* In addition to CAD risk assessment, do a baseline resting ECG if:
  + >40 years of age;
  + duration of diabetes more than 15 years;
  + ALL (regardless of age) with HTN, proteinuria, reduced pulses or vascular bruits
  + Repeat resting ECG every 2 years in people considered at high risk for CV events.
* Investigate for CAD by exercise stress testing as the initial test if:
  + Typical or atypical cardiac symptoms (e.g. unexplained dyspnea, chest discomfort)
  + Resting abnormalities on ECG (e.g. Q waves)
  + Peripheral arterial disease (abnormal ankle-brachial ratio)
  + Carotid bruits
  + Transient ischemic attack
  + Stroke
* Pharmacologic stress echocardiography or nuclear imaging should be done if resting ECG abnormalities preclude the use of exercise ECG stress testing (e.g. LBBB or ST-T abnormalities) or if unable to exercise.
* Those who demonstrate ischemia at low exercise capacity (<5 metabolic equivalents [METs]) on stress testing should be referred to a cardiac specialist.

1. **In the acutely ill diabetic patient, diagnose the underlying cause of the illness and investigate for diabetic ketoacidosis and hyperglycemia.**

* Diabetic ketoacidosis (DKA) and hyperosmolar hyperglycemic state (HHS) should always be suspected in ill patients with diabetes. If either DKA or HHS is diagnosed, precipitating factors must be sought and treated.
* DKA risk factors: inadequate or inappropriate insulin therapy, infection, myocardial infarction, abdominal crisis, trauma.
* DKA:
  + Triad of hyperglycemia, anion gap metabolic acidosis, and ketonemia. Metabolic acidosis is often the major finding.
  + Clinical presentation: symptoms of hyperglycemia, Kussmaul respiration, acetone-odoured breath, ECFV contraction, nausea, vomiting and abdominal pain. ± a decreased level of consciousness.
  + Ketoacidosis occurs as a result of elevated glucagon and absolute insulin deficiency.
* HHS :
  + Main features: extracellular fluid volume (ECFV) depletion and hyperosmolarity.
  + Little or no ketones, with serum glucose usually more than 33 mmol/L 🡪 more prolonged duration of relative insulin insufficiency and inadequate fluid intake (or high glucose intake) results in higher glucose levels
  + Hyperglycemia causes urinary losses of water and electrolytes (sodium, potassium, chloride) and the resultant ECFV depletion.
  + High catecholamine levels suppress insulin release.
  + Often more profound ECFV contraction and decreased level of consciousness compared to DKA.
  + Can be a variety of neurological presentations that may resolve once osmolality returns to normal.
  + Neurologic complications (stupor, seizures or stroke-like state) related to higher glucose levels and serum osmolality are more common in HHS.
  + greater ECFV contraction, but minimal acid-base disturbance.
* To make the diagnosis and determine the severity of DKA or HHS, assess:
  + Serum electrolytes (and anion gap), glucose, creatinine, osmolality and betahydroxybutyric acid (beta-OHB) (if available), blood gases, serum and urine ketones.
  + Anion gap gives an estimate of the quantity of unmeasured anions in the serum (ketoacids) : anion gap  =  sodium  -  (chloride + bicarbonate)
* There are no definitive criteria for the diagnosis of DKA. Typically:
  + arterial pH <7.3, serum bicarbonate <15 mmol/L, anion gap >12 mmol/L, positive serum and/or urine ketones.
    - Plasma glucose usually > 14.0 mmol/L, but can be lower.
  + It is therefore important to measure ketones in both the serum and urine.
  + If there is an elevated anion gap, and serum ketones are negative, beta-hydroxybutyrate levels should be measured.
  + Measurement of serum lactate should be considered in hypoxic states.

1. **Given a patient with DKA, manage the problem appropriately and advise about preventing future episodes.**

* DKA and HHS is best managed in ICU or a step-down unit with specialist care.
* Volume status (including fluid intake and output), vital signs, neurologic status, plasma concentrations of electrolytes, anion gap, osmolality and glucose need to be monitored closely, initially as often as every 2 hours.
* Monitor serum glucose every hour
* Precipitating factors must be diagnosed and treated.
* IV fluid:
  + The initial goal is to restore tissue perfusion. In DKA, 0.9% sodium chloride (NS) should be administered by IV at 500 mL/hour for 4 hours, then 250 mL/hour for 4 hours.
  + Higher initial rate of NS (1–2 L/hour) should be given in the presence of shock.
* Insulin:
  + Must correct hypokalemia prior to giving insulin.
  + Continuous IV infusion of regular insulin at 0.1U/kg/hr is the treatment of choice.
  + IV bolus of regular insulin (0.1 U/kg) is recommended in some reviews.
  + When serum glucose reaches 11.1 mmol/L, switch NS to D5W. It may be possible to decrease the insulin infusion rate to 0.02 to 0.05 U/kg per hour, but DO NOT STOP INSULIN INFUSION until there is correction of Serum pH and the anion gap.
* Potassium:
  + Typical recommendations suggest that potassium should be started for plasma potassium <5.3 mmol/L, once diuresis has been established.
  + If the patient at presentation is normo-or hypokalemic, potassium should be given immediately, at concentrations in the IV fluid between 10 and 40 mmol/L.
  + For potassium ≤ 3.3 mmol/L, withhold insulin until potassium replacement at 40 mmol/hour has restored plasma potassium to >3.3 mmol/L.
* Sodium bicarbonate therapy is controversial 🡪 considered only in adult patients in shock or with arterial pH <7.0.
* Hypophosphatemia:
  + Hypophosphatemia has been associated with rhabdomyolysis.
  + Administration of potassium phosphate in cases of severe hypophosphatemia could be considered for the purpose of trying to prevent rhabdomyolysis.
* Prevention:
  + The most common precipitating factor to DKA is poor adherence to diabetes treatment. Patients may discontinue diabetes monitoring for many reasons (cost, poor understanding of the disease, psychological disorders such as depression and eating disorders).
  + Instructions should clearly indicate when to consult the physician: any symptoms that may signal DKA or dehydration 🡪 dizziness, trouble breathing, fruity breath, or dry and cracked lips or tongue.
  + Establish sick-day protocol (see previous question)

1. These include: Extreme proteinuria (>6 g/day), persistent hematuria (microscopic or macroscopic) or active urinary sediment, fapidly falling eGFR, Low eGFR with little or no proteinuria, other complications of diabetes not present or relatively not as severe, known duration of diabetes less than 5 years, family history of nondiabetic renal disease (eg. Polycystic kidney disease) or signs and symptoms of systemic disease. [↑](#footnote-ref-1)