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ABOUT THE AUTHORS

Gregory L. Hundemer, MD, MPH

Dr. Greg Hundemer is a hypertension specialist and staff nephrologist at the Ottawa Hospital, an Associate Professor of Medicine at the University of Ottawa, and a Scientist at the Ottawa Hospital Research Institute. His clinical and research interests focus on the role of aldosterone in hypertension and vascular disease. His research has been supported by the NIH, CIHR, and Kidney Foundation of Canada. He holds the Lorna Jocelyn Wood Chair for Kidney Research at the University of Ottawa and serves as Co-Chair of the Hypertension Canada Clinical Practice Guidelines Committee.

Affiliations: *Department of Medicine, Division of Nephrology, University of Ottawa, Ottawa, Ontario, Canada*

Kristin A. Terenzi, MD

Dr. Kristin Terenzi is a family physician in private practice in Woodbridge, Ontario, and a Fellow in the College of Family Physicians. She was a member of the guideline committee that developed the *2025 Hypertension Canada Guideline for the Diagnosis and Treatment of Hypertension in Adults in Primary Care*.

Affiliations: *Langstaff Medical Centre, Woodbridge, ON, Canada*

Rémi Goupil, MD, MSc

Dr. Rémi Goupil is a hypertension specialist and nephrologist at the Hôpital du Sacré-Coeur de Montréal, Associate Professor at the Université de Montréal and a Junior 2 clinician-scientist of the Fonds de recherche du Québec-Santé. His research is focused on optimising blood pressure measurements and hypertension care in various populations. His work has received multiple funding by the CIHR, the Kidney Foundation and the Heart and Stroke Foundation. Since 2024, he serves as Co-Chair for the Hypertension Canada Clinical Practice Guidelines Committee and is the president-elect of the Société québécoise d'hypertension artérielle.

Affiliations: *Department of Medicine, Division of Nephrology, Hôpital du Sacré-Coeur de Montréal, Université de Montréal, Montréal, QC, Canada*

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Primary Care Hypertension Guidelines: What's New for Primary Care

Gregory L. Hundemer, MD, MPH
Kristin A. Terenzi, MD
Rémi Goupil, MD, MSc

Hypertension remains the leading modifiable risk factor for cardiovascular disease and mortality, yet recent data demonstrate declining rates of hypertension treatment and control in Canada. Because most hypertension care occurs in primary care, improving population-level outcomes requires guidance that is pragmatic, streamlined, and easily implemented into routine primary care practice. To address this need, Hypertension Canada has released a new Primary Care Hypertension Guideline, developed as part one of a two-part guideline strategy and informed by contemporary evidence and extensive input from primary care providers and patients. This article highlights the most clinically relevant updates from the 2025 Hypertension Canada Primary Care Guideline for front-line clinicians. Major changes include adoption of a lower blood pressure threshold (≥130/80 mmHg) to define hypertension, selective initiation of pharmacotherapy based on blood pressure level and cardiovascular risk, and a simplified target of systolic blood pressure <130 mmHg for most patients when tolerated. The guideline emphasizes accurate blood pressure measurement, routine use of out-of-office monitoring, and foundational lifestyle modification for all individuals with hypertension. Importantly, for patients requiring medication, early initiation of low-dose single-pill combination therapy is recommended to improve blood pressure control and reduce therapeutic inertia. These recommendations are embedded within the World Health Organization's HEARTS framework, providing standardized diagnostic and treatment algorithms tailored to Canadian primary care. Together, these updates are intended to simplify hypertension management, enhance guideline uptake, and improve cardiovascular outcomes at the population level.

Background

Hypertension remains the most prevalent modifiable risk factor for cardiovascular disease and cardiovascular mortality worldwide.[1,2] In Canada, approximately one in four adults lives with hypertension, imposing a substantial burden on individuals, families, and the healthcare system.[3] Canada has historically been recognized as a global leader in hypertension awareness, treatment, and control.[4] However, this success has faded over the past decade. Recent national data demonstrate declining rates of hypertension treatment and control, raising concerns about increasing cardiovascular morbidity and mortality.[5,6]

Several factors have been proposed to account for this regression, including uncertainty and inconsistency surrounding optimal blood pressure thresholds, increasingly complex guideline recommendations, limited uptake of evidence into routine clinical practice, and insufficient engagement with front-line primary care providers. Given that the vast majority of hypertension diagnosis and management occurs in primary care settings, improving population-level blood pressure control relies heavily on making hypertension care simpler, more pragmatic, and easier to implement into everyday practice.

In response, Hypertension Canada has adopted a new two-part guideline strategy.[7] Alongside its forthcoming comprehensive guideline topics, a dedicated Primary Care Hypertension Guideline has been developed to provide streamlined, evidence-based, and implementation-ready recommendations tailored to real-world primary care settings.[8] This article highlights the key updates in the 2025 Hypertension Canada Primary Care Guideline and explores their practical implications for clinicians.

Why a Primary Care Focus?

Primary care serves as the cornerstone of hypertension management. Family physicians, physician assistants, nurse practitioners, nurses, and pharmacists diagnose hypertension, initiate treatment, adjust therapy, and provide long-term follow-up for the vast majority of patients with elevated blood pressure. Yet, traditional hypertension guidelines, including prior versions from Hypertension Canada, have often been lengthy, highly technical, and cumbersome to apply in busy clinical settings. Feedback from Canadian primary care providers and patients has consistently emphasized the need for guidance that is simpler, clearer, and more actionable. The 2025 Primary Care Guideline was intentionally designed to address this gap by focusing on the most common clinical scenarios encountered in primary care and by prioritizing recommendations most likely to influence outcomes at the population level.[8]

Importantly, this guideline is not intended to replace clinical judgment or to be applied to every patient. Rather, it provides a practical framework for managing the majority of adults with hypertension, while acknowledging that more complex cases may require individualized decision making or specialist involvement.

The HEARTS Framework

A defining feature of the new guideline is its integration of the World Health Organization's HEARTS framework, a globally recognized model for improving hypertension control at the population level.[9] The HEARTS framework emphasizes standardized diagnosis, simplified treatment algorithms, team-based care, and ongoing monitoring and evaluation. The framework was originally developed and implemented within the Kaiser Permanente health system, where hypertension control rates increased dramatically from approximately 44% to nearly 90% in just over a decade.[10,11]

Since its implementation, the HEARTS framework has been successfully adapted across diverse healthcare systems worldwide.[12] Importantly, HEARTS is not intended to function as a rigid protocol but rather as a flexible structure that can be adapted to meet the needs of a specific population to support consistency and efficiency of care. By embedding guideline recommendations within clear diagnostic and treatment algorithms, HEARTS aims to reduce therapeutic inertia, minimize unnecessary variation in care, and make it easier for clinicians to achieve blood pressure targets. The 2025 Primary Care Guideline's HEARTS-based diagnostic and treatment algorithms are displayed in **Figure 1** and **Figure 2**, respectively.[8]

A Lower Blood Pressure Threshold to Define Hypertension

One of the most notable changes in the 2025 guideline is the adoption of a lower blood pressure threshold to define hypertension: ≥130/80 mmHg when measured with a validated device under optimal conditions. This change represents a shift from previous Canadian guidelines and reflects accumulating evidence that cardiovascular risk begins well below traditional thresholds. Observational studies demonstrate a continuous relationship between blood pressure and cardiovascular events well below the traditional blood pressure threshold of 140/90 mmHg,[13] while randomized trials show meaningful reductions in cardiovascular events with blood pressure lowering in adults with systolic blood pressure starting at or above 130 mmHg.[14] For primary care providers, this change emphasizes earlier detection of patients at increased cardiovascular risk. While the revised definition will increase the number of individuals labelled as having hypertension, the goal is not over-treatment but rather earlier engagement in risk reduction, often through lifestyle modification alone.

Accurate blood pressure measurement is central to this approach. The guideline strongly recommends the use of validated automated blood pressure devices and standardized measurement techniques. Out-of-office blood pressure monitoring, either ambulatory or home-based,

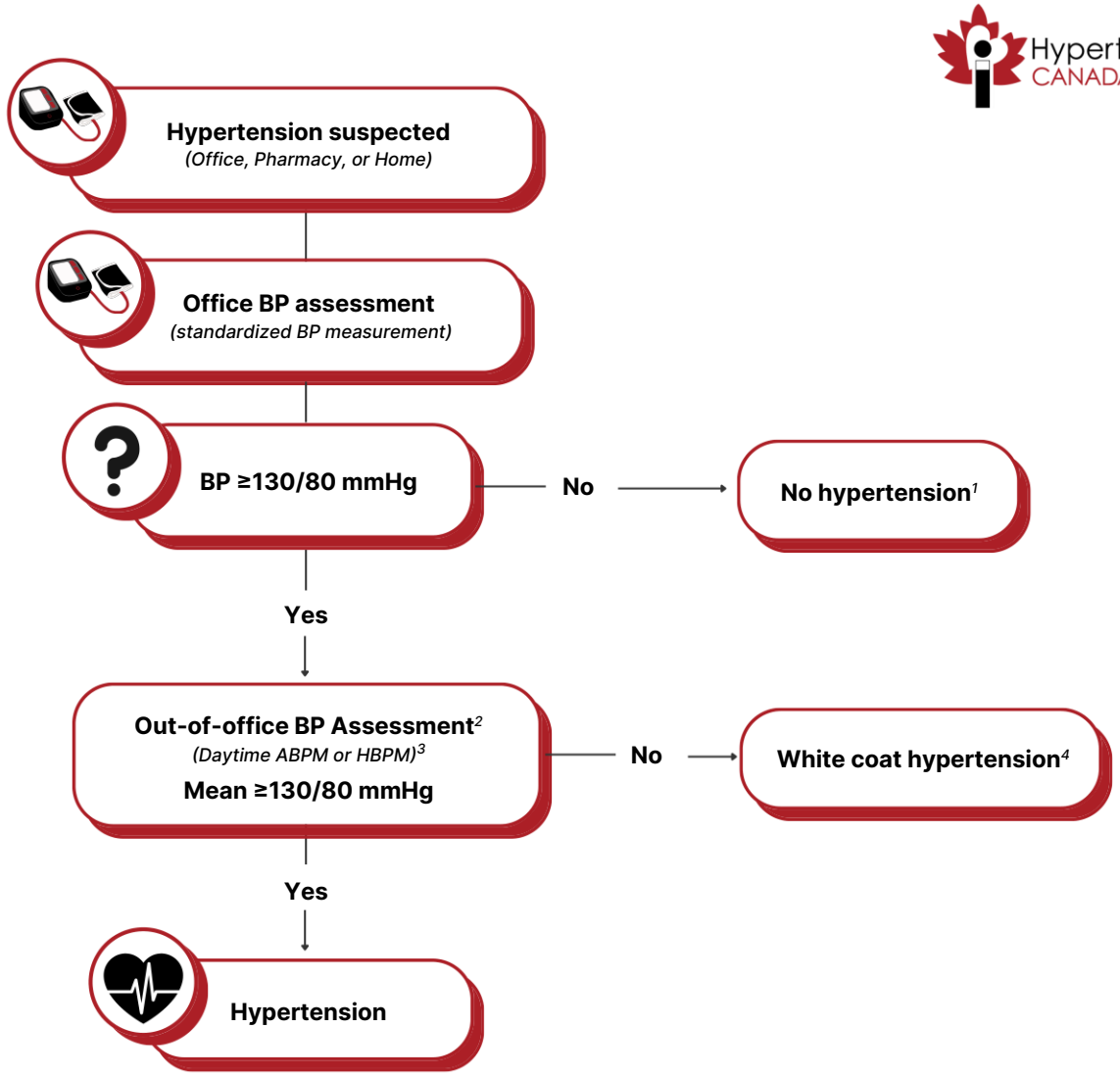

Figure 1. Suggested algorithm for the diagnosis of hypertension in primary care; *adapted from Goupil R et al. CMAJ. 2025;197(20):E549-E64)*.

1) Continue yearly or opportunistic BP monitoring; perform out-of-office BP assessment if masked hypertension is suspected.
2) If unavailable or infeasible, repeat office BP assessment at a separate visit.
3) Use Hypertension Canada-recommended or other validated home BP devices.
4) Continue yearly or opportunistic BP monitoring.

Abbreviations: ABPM: ambulatory blood pressure monitor; **BP:** blood pressure; **HBPM:** home blood pressure monitor.

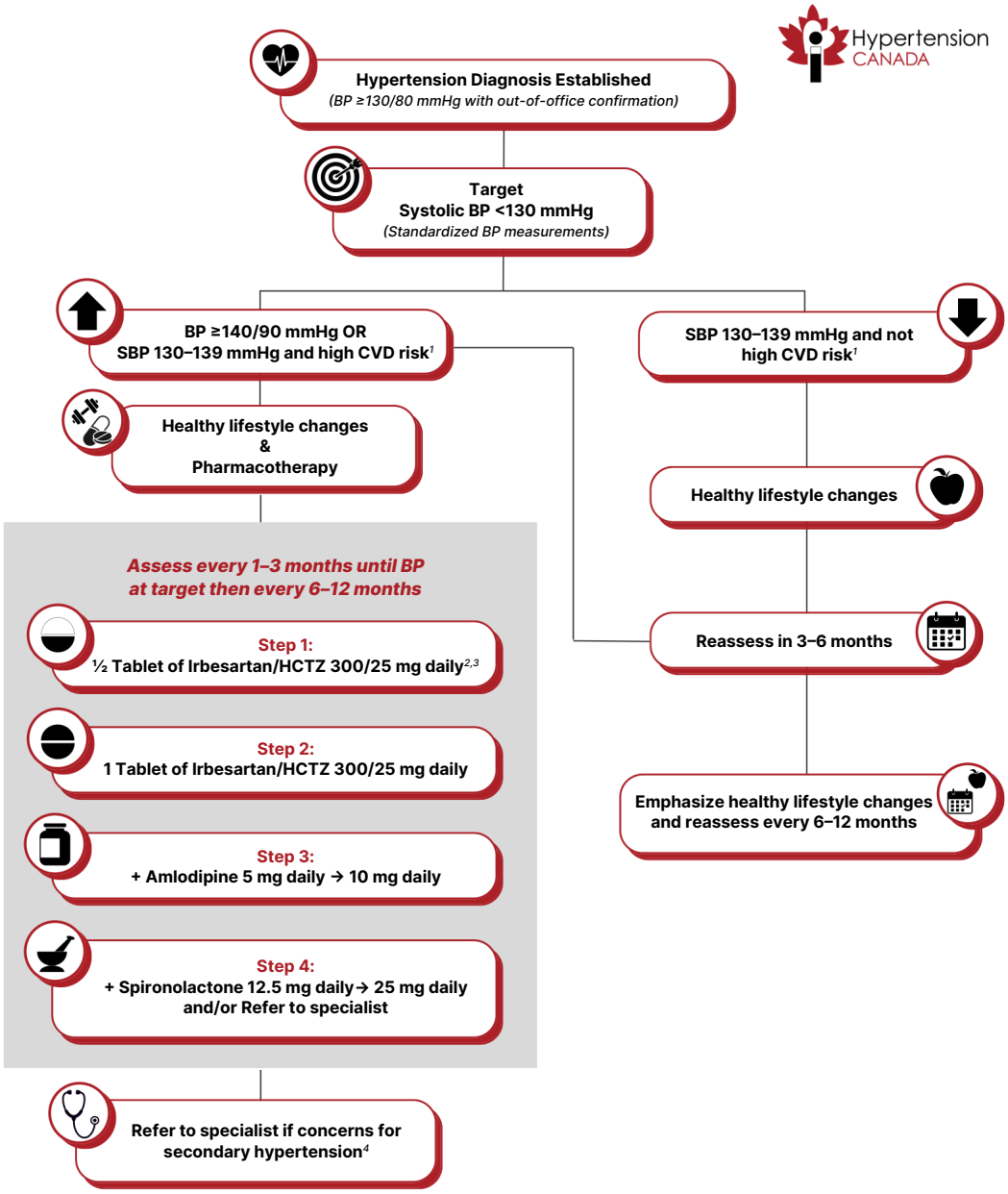

Figure 2. Suggested algorithm for the treatment of hypertension in primary care.; *adapted from Goupil R et al. CMAJ. 2025;197(20):E549-E64).*

1) High CVD risk conditions include established cardiovascular disease (coronary heart disease, heart failure, cerebrovascular disease, and peripheral arterial disease), type 1 or type 2 diabetes mellitus, chronic kidney disease (eGFR <60 mL/min/1.73 m2 or albuminuria ≥3 mg/mmol), 10-year Framingham Risk Score ≥20%, and age ≥75 years.

2) Initial single-pill combination therapy is preferred if available and covered. If unavailable, two separate pills can be used as an alternative, with preference given to a long-acting thiazide-like diuretic over a thiazide diuretic. In patients at high risk of symptomatic hypotension, treatment initiation with a single agent is reasonable. This algorithm is not intended for use during pregnancy. ACEIs and ARBs should be avoided/discontinued in all individuals who are pregnant or trying to become pregnant.

3) Acceptable alternative single-pill combinations available in Canada are listed in Table 4 of the original guideline manuscript.[8]

4) Features suggestive of secondary hypertension include unexplained sudden worsening of hypertension, hypokalemia, adrenal nodule, abdominal bruit, acute kidney injury following hypertension treatment, severe/malignant hypertension, hypertension at a young age, and resistant hypertension.

Abbreviations: ACEI: angiotensin-converting enzyme inhibitor; **ARB:** angiotensin II receptor blocker; **BP:** blood pressure; **CVD:** cardiovascular disease; **eGFR:** estimated glomerular filtration rate; **HCTZ:** hydrochlorothiazide.

is encouraged to confirm the diagnosis of hypertension and to identify white coat and masked hypertension, both of which are common and clinically relevant.[15,16]

Not All Hypertension Requires Pharmacotherapy

Despite the lower diagnostic threshold used to define hypertension, the guideline clearly distinguishes diagnosis from pharmacotherapy initiation. Not all patients with blood pressure ≥130/80 mmHg require antihypertensive medication. Pharmacotherapy is recommended for:

- All adults with blood pressure ≥140/90 mmHg, regardless of cardiovascular risk.
- Adults with systolic blood pressure 130–139 mmHg who are at high cardiovascular disease risk, such as those with established cardiovascular disease, diabetes mellitus, chronic kidney disease, or a 10-year Framingham Risk Score ≥20%.

For patients with systolic blood pressure of 130–139 mmHg who are not at high cardiovascular risk, the emphasis is on lifestyle modification, with planned blood pressure reassessment over time. This distinction is critical in primary care, where avoiding over-treatment and polypharmacy is a legitimate concern.

Lifestyle interventions remain foundational for all patients with hypertension, regardless of whether pharmacotherapy is indicated. Evidence supports meaningful blood pressure reduction through dietary sodium reduction, increased dietary potassium intake, achievement of a healthy weight, regular physical activity, reduction in alcohol intake, and smoking cessation. Importantly, lifestyle changes may reduce or delay the need for medication and enhance the effectiveness of pharmacotherapy when it is required.

A Single Blood Pressure Target for Most Patients

Another key innovation in the guideline is the recommendation of a single systolic blood pressure target of <130 mmHg for most adults with hypertension, provided treatment is well tolerated. This simplified approach responds directly to feedback from primary care providers who requested clearer and more consistent targets. Evidence from large randomized trials and meta-analyses shows that achieving systolic blood pressure below 130 mmHg substantially reduces major adverse cardiovascular events and mortality compared with higher targets.[14] While even more intensive targets (e.g., <120 mmHg) may confer additional benefit in select high-risk populations, they are more difficult to achieve in routine practice and are associated with higher rates of adverse events.[17] The <130 mmHg target represents a balance between efficacy, safety, and feasibility.

The guideline also acknowledges that this blood pressure target may not be appropriate for all patients. Factors such as frailty, orthostatic hypotension, fall risk, comorbidities, and patient preferences must be considered. In such cases, clinicians are encouraged to individualize care and aim for the lowest blood pressure that is safely achievable.

An Emphasis on Single-Pill Combination Therapy

One of the most practice-changing recommendations in the guideline is the strong endorsement of low-dose combination therapy as initial pharmacologic treatment, ideally delivered as a single-pill combination. Most patients with hypertension require more than one medication to achieve blood pressure control, particularly with lower blood pressure targets.[18] Starting therapy with combination treatment increases the likelihood of early blood pressure control, reduces therapeutic inertia, and minimizes the need for frequent dose-titration visits.[19] The guideline recommends initial therapy using two of the following three complementary first-line antihypertensive drug classes:

1. Angiotensin-converting enzyme inhibitors or angiotensin II receptor blockers
2. Thiazide or thiazide-like diuretics
3. Long-acting dihydropyridine calcium channel blockers

Beta-blockers are no longer recommended as first-line antihypertensive agents unless a specific clinical indication is present (e.g., arrhythmia, post-myocardial infarction, heart failure).

Single-pill combinations offer several advantages that are highly relevant in primary care, including improved adherence, faster blood pressure control, fewer clinic visits, and lower overall costs compared with free drug combinations.[19] Importantly, evidence suggests that upfront combination therapy is associated with reductions in cardiovascular hospitalizations and all-cause mortality.[20] For patients whose blood pressure remains above target, the guideline recommends escalation to triple therapy using the same three complementary first-line drug classes, with spironolactone suggested as the preferred fourth-line agent for managing resistant hypertension.

Summary

The 2025 Hypertension Canada Primary Care Guideline represents a meaningful shift toward simpler, more pragmatic hypertension management. Key updates (including a lower diagnostic threshold of ≥130/80 mmHg, selective initiation of pharmacotherapy, a single blood pressure target of <130 mmHg, and early use of single-pill combination therapy) are grounded in contemporary evidence and designed for real-world primary care practice settings. By integrating these recommendations within the HEARTS framework, the guideline prioritizes standardization, efficiency, and population-level impact while preserving flexibility for individual patient care. Successful implementation has the potential to reverse declining hypertension control rates in Canada and to substantially reduce the burden of cardiovascular disease. For primary care providers, the messages are clear: measure blood pressure accurately, intervene earlier, treat decisively when indicated, and simplify care wherever possible. Hypertension control remains one of the most powerful tools we have to improve long-term health outcomes, and primary care remains at the heart of that effort.

Correspondence

Gregory L. Hundemer, MD, MPH
E-mail: ghundemer@toh.ca

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ABOUT THE AUTHORS

Eviatar Fields, PhD

Dr. Eviatar Fields is a third-year medical student at McGill University. Prior to medical school, he completed his undergraduate degree in psychology at York University, followed by a PhD in neuroscience at McGill University. He has a strong background in basic and clinical research, as well as innovation. His work spans multiple disciplines, with a focus on research translation, evidence synthesis, and patient-centered care. Dr Fields has contributed to numerous peer-reviewed publications, and he has been supported by several funding agencies including CIHR and FRQS. He was recently recognized as the recipient of the 2026 Canadian Urological Association Medical Student Award. He is passionate about patient education and bridging the gap between bench research and clinical care to improve healthcare delivery and patient outcomes.

Affiliations: *Faculty of Medicine, McGill University*

Naeem Bhojani, MD

Dr. Naeem Bhojani is a Montreal-born academic urologist and internationally recognized endourologist with a clinical and research focus on kidney stone disease and BPH. He completed undergraduate degrees in Microbiology and Immunology as well as Physiotherapy at McGill University, followed by medical school and urology residency training at the Université de Montréal. He then undertook a fellowship in BPH and stone disease at Indiana University. Recruited to the Université de Montréal in 2013, he established one of Canada's first comprehensive multidisciplinary kidney stone programs and a novel patientcentered BPH clinic. He currently holds the rank of Full Professor and serves as a clinician-scientist. His research, supported by the CIHR, FRQS and other national funding agencies, focuses on innovation in endourology, patient-reported outcomes and quality of life. He led the development of the CEGSSS, a patient-centered stent symptom questionnaire. Dr. Bhojani has authored more than 290 peer-reviewed publications in the fields of stone disease and BPH. He serves on numerous editorial boards and is an Associate Editor for the *Canadian Urological Association Journal*. His contributions have been recognized with multiple prestigious awards, including the American Urological Association Young Urologist of the Year in 2017, the FRQ-S Clinical Research Scholar Award in 2019, and finally, in 2025 Dr. Bhojani was named the Best Endourologist of the Year by the European Association of Urology.

Affiliations: *Division of Urology, University of Montreal*

Bilal Chughtai, MD

Dr. Bilal Chughtai completed his medical degree followed by residency in urologic surgery at Albany Medical Center. Dr. Chughtai completed his fellowship in Male Voiding Dysfunction, Neuro-Urology, Female Pelvic Medicine Reconstructive Surgery at Memorial Sloan-Kettering Cancer Center and New York Presbyterian Hospital/Weill Cornell Medical College in New York City. Dr. Chughtai is currently Chief of Urology at Plainview and Syosset Hospital who specializes in Female Pelvic Medicine and Reconstructive Surgery and Male Voiding Dysfunction. Dr. Chughtai maintains a deep, longitudinal interest in these highly prevalent and debilitating conditions and has focused both his clinical, research, and teaching activities on these areas. Dr. Chughtai has mentored students, residents, and fellows in clinical and basic science research projects which has allowed his team to be investigators in numerous clinical trials, as well as translational, and outcomes research including National Institute of Health and industry support. Dr. Chughtai has published over 300 peer reviewed articles, authored chapters in several urologic texts, published 4 textbooks, and has presented numerous national and international meetings.

Affiliations: *Division of Urology, Zucker School of Medicine at Hofstra/Northwell*

Dean Elterman, MD

Dr. Dean Elterman is an Associate Professor of Urology at the University of Toronto, sub-specializing in functional urology. He holds the Lang Family Endowed Chair in Urologic Innovation. As an attending urologist at the University Health Network (Toronto), his clinical and research interests include voiding dysfunction, novel technologies for BPH, sacral neuromodulation, male incontinence, and men's health. He trained at the University of Toronto, Memorial Sloan-Kettering Cancer Center and New York Presbyterian/Weill Cornell Medical College. He holds a Master's degree in Clinical Epidemiology and Health Services Research from Cornell. He is the Medical Director of the Sexual Health Clinic at Princess Margaret Cancer Center and Fellowship Director for functional urology. He is the recipient of the 2022 SIU Innovators award, leads numerous clinical trials in BPH and neuromodulation, and has published over 280 peer reviewed manuscripts including the lead author of the Canadian guidelines for Male LUTS/BPH. Dr. Elterman is the Associate Editor of European Urology Focus, and is an Associate Member of both the EAU Section of Section of Functional Urology and the EAU Section of Endourology. He sits on the Board of Directors for the Société Internationale D'Urologie (SIU) as the Innovators Chair, and is the Chief Medical Officer of MedTech Syndicates, a platform which supports clinicians to fund MedTech innovation.

Affiliations: *Division of Urology, Department of Surgery, University of Toronto*

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Andropause: Myths and Management

Eviatar Fields, PhD
Naeem Bhojani, MD
Bilal Chughtai, MD
Dean Elterman, MD

Over the past two decades, prescription rates for testosterone have risen sharply, due to direct-to-consumer advertising and the spread of online misinformation. As a result, the topic of andropause, or aging-associated testosterone deficiency, is increasingly encountered by Canadian primary care physicians. Despite its growing visibility, up to 25% of healthcare practitioners report challenges in accurately diagnosing and managing andropause. In this article, we clarify the common myths around andropause to better inform primary care physicians and their patients. We discuss the Canadian guidelines on diagnosis and management of testosterone deficiency. Finally, we provide insight into the safety of testosterone-replacement therapy. By implementing structured monitoring and shared decision making, primary care physicians can ensure safe, effective, and evidence-based care for aging Canadians.

Introduction

Andropause is a colloquial term used to describe age-associated testosterone deficiency. It is characterized by deficient production of testosterone, leading to three major categories of symptoms: **1)** sexual symptoms including reduced libido, erectile dysfunction, and ejaculatory dysfunction; **2)** cognitive symptoms including fatigue, depression, and insomnia; and **3)** physical symptoms including decreased energy levels, increased visceral fat, and alterations in bone and muscle density.[1]

Testosterone deficiency has been receiving considerable attention in recent years, with a growing proportion of men receiving exogenous testosterone despite the absence of a conclusive diagnosis of hypogonadism.[2,3] Due to considerable misinformation about andropause and the benefits of testosterone-replacement therapy (TRT), coupled with a push for its direct-to-consumer advertising,[4,5] primary care physicians are increasingly facing patient-initiated requests for testosterone testing and treatment. Further, up to 25% of Canadian healthcare practitioners report difficulty with the diagnosis and treatment of testosterone deficiency.[1] Demystifying the diagnosis and management of andropause will thus benefit Canadian physicians and patients alike.

In this article, we will examine some of the common myths surrounding aging-related testosterone decline and discuss its management.

Common Myths

Myth 1: Andropause is simply the male equivalent of menopause.

Reality: Andropause is highly variable and not universally experienced by men.

Unlike menopause, which is universally experienced by women typically around age 50, there is great variability in the clinical and biochemical manifestations of andropause. The onset of andropause is insidious, with a gradual decline in testosterone beginning as early as age 30, with serum testosterone levels decreasing at a rate of 1–2% per year[6] The decline in testosterone levels is influenced by many factors beyond chronological age, including smoking status, glycemic control (HbA1c), body mass index, and other comorbidities.[7]

While menopause is a well-characterized phenomenon resulting in complete loss of fertility, the criteria for andropause is subject to multiple guideline variations and does not follow a definitive path.[8] Indeed, the term andropause is somewhat misleading, as the true 'pausing' of male androgen production can only occur from the

complete loss of testicular function, such as from medical castration. This ambiguity is reflected in the diverse names given to andropause, including hypogonadism or late-onset hypogonadism (LOH) by the Endocrine Society and European Association of Urology, testosterone deficiency by the Canadian and American Urological Associations, or androgen deficiency in the aging male (ADAM).

Myth 2: Andropause is a "silver bullet" diagnosis for the symptoms of aging.

Reality: Andropause is often a symptom of aging rather than a cause.

Aging is associated with functional decline across multiple physiological systems, leading to a broad range of non-specific symptoms. Many of these symptoms—erectile dysfunction, reduced libido, fatigue, reduced muscle and bone mass, cognitive complaints, and weight gain—are loosely associated with reduced testosterone levels. Because of the inverse relationship between testosterone and biological age, patients may be misled to believe that testosterone reduction causes aging.

In practice, these symptoms are multifactorial, and their underlying causes are often difficult to determine. Erectile dysfunction, for example, is often more strongly associated with vascular health and psychosocial factors than with reduced testosterone levels. Comorbid conditions such as obesity and metabolic syndrome, diabetes, and cardiovascular diseases can also exert an influence on sexual function, energy levels, and cognition, independent of androgen status. Indeed, many of the classic signs of low testosterone, including the sexual symptoms of androgen deficiency, show only weak associations with low testosterone in older men.[9] Consequently, a large proportion of men with testosterone deficiency confirmed with bloodwork remain asymptomatic.[10]

Attributing aging-related symptoms to testosterone deficiency risks oversimplification that could lead to inappropriate treatment with TRT, which shows limited benefit in men without confirmed testosterone deficiency.[4] Despite this, testosterone deficiency should remain part of the differential diagnosis, and biochemical evaluation may be appropriate in patients presenting with multiple relevant comorbidities.

Myth 3: Andropause is an inevitable consequence for aging men, and all men should consider TRT.

Reality: Not all men experience symptoms of andropause, and TRT shows limited benefit without confirmed testosterone deficiency.

Despite an annual decline of testosterone levels, most men will maintain normal testosterone levels, and fewer than 25% meet biochemical criteria for testosterone deficiency.[10] Further, only approximately 2% of men are estimated to experience symptoms of andropause,[9] demonstrating the complex interactions between genetics, lifestyle, comorbidities, and medications that play a role in the manifestation of andropause. Rather than an inevitable consequence of aging, it is possible that andropause only impacts a small subset of men.

The goal of TRT is to restore physiological levels of testosterone in men with confirmed testosterone deficiency to alleviate the symptoms of andropause.[11] Clinical trials have shown that TRT can improve sexual, mood, and osteoporotic symptoms in men with testosterone deficiency.[12,13] However, TRT confers limited benefit in men without pathological hypogonadism,[4] and even among men with confirmed testosterone deficiency, improvements in vitality and cognition are modest.[12,13]

Testosterone replacement is not without risk, with commonly reported side effects including polycythemia, acne, fluid retention, gynecomastia, and worsening of sleep apnea.[11] TRT also leads to infertility and is thus contraindicated in patients wishing to remain fertile (see **Box 1**).[13] Despite these risks, off-label prescribing of TRT has substantially increased over the past two decades, largely driven by direct-to-consumer advertising and misinformation about its benefits in aging men without testosterone deficiency.[4,5] Although short trials of TRT (3–6 months) may be warranted for symptomatic men when alternative causes of the symptoms have been excluded, TRT should be used as a tool for the treatment of symptoms of low testosterone, rather than a 'fountain of youth' for aging men: We treat the patient, not the blood-test.

Contraindications for TRT
Allergy or hypersensitivity to testosterone
Male breast cancer (known or suspected)
Desire to preserve fertility
Metastatic or high-risk prostate cancer requiring androgen deprivation therapy
Unstable cardiovascular disease or recent cardiac events within the previous 3–6 months

Box 1. Contraindications for TRT; *courtesy of Eviatar Fields, PhD, Naeem Bhojani, MD, Bilal Chughtai, MD and Dean Elterman, MD*.

Clinical Pearls
Prioritize clinical history: Testosterone testing should not be performed routinely in all men but reserved for those with a positive history and persistent symptoms.
The equivocal range: In men with total testosterone between 8–12nmol/L, always calculate free testosterone because elevated sex hormone-binding globulin (SHBG) (elevated in ageing and liver disease) can mask a functional androgen deficiency.
Circadian testosterone: Always ensure that blood draws for testosterone are obtained between 7:00 and 11:00 AM. Testing outside this window increases the risk of false-positive results due to natural circadian decline.
TRT is not a "Fountain of Youth": Although TRT can help symptomatic patients with confirmed testosterone deficiency, clinical trials demonstrate minimal benefits for vitality, or for asymptomatic men with numerous contraindications, side effects and risks, and should be avoided in asymptomatic men regardless of testosterone levels.

Box 2. Clinical Pearls; *courtesy of Eviatar Fields, PhD, Naeem Bhojani, MD, Bilal Chughtai, MD and Dean Elterman, MD*.

Myth 4: Total testosterone is the definitive metric for diagnosing andropause.

Reality: Although total testosterone is useful as an initial test, it requires clinical correlation, as well as supplementary testing in equivocal cases.

According to Canadian clinical guidelines, the clinical diagnosis of testosterone deficiency is based on several factors (see **Figure 1** and **Box 2**).[1,3] First, as mentioned, an initial diagnosis requires a total testosterone level below 8 nmol/L (230 ng/dL), in conjunction with a positive clinical history and physical examination. However, because total testosterone measures both protein-bound and unbound hormone, patients whose testosterone falls within the borderline range of 8–12 nmol/L are considered equivocal. These patients warrant further biochemical workup to determine whether symptoms are driven by a lack of active, unbound hormone.

Up to 60% of plasma testosterone is bound to sex hormone-binding globulin (SHBG), rendering it biologically inactive, with only approximately 2% of total testosterone being unbound (i.e., free testosterone) and thus bioactive.[14] Variations in SHBG levels will skew the proportion of bound to unbound hormone for a given total testosterone value. Several factors are known to increase SHBG levels, including aging, hyperthyroidism, and liver disease.[14] As a result, many aging patients with testosterone levels within this equivocal range have a 'normal' appearing total testosterone level, despite having reduced free testosterone due to elevated SHBG, which could explain their symptoms. Calculating free testosterone in older men with total testosterone within the equivocal range can prevent clinicians from missing biochemical hypogonadism.

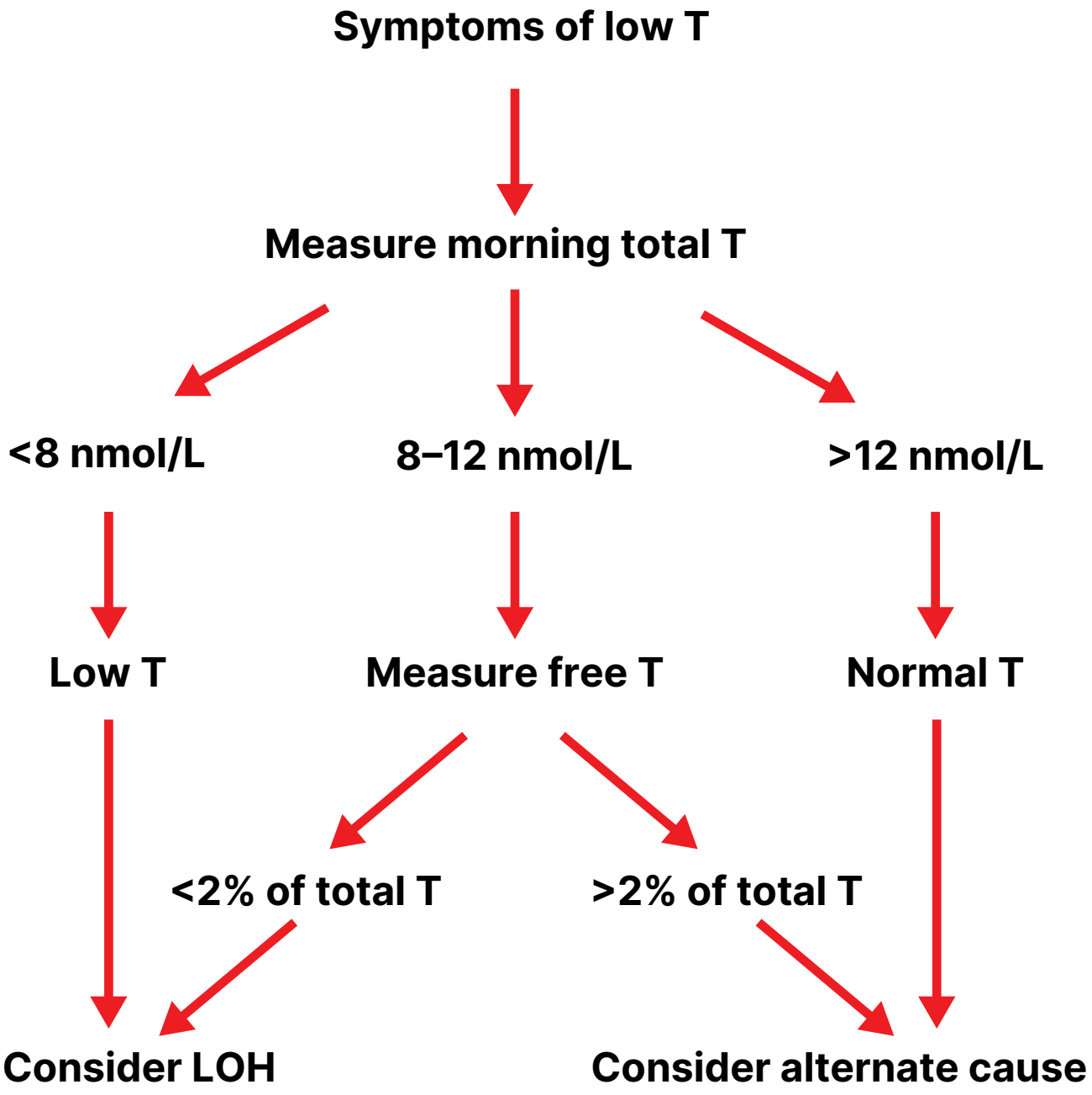

Figure 1. Diagnostic algorithm for men experiencing the symptoms of low testosterone.[1] Men with symptoms of low testosterone (T) in the absence of a cause should undergo measurement of morning total T. If total T falls within the equivocal range (8–12 nmol/L), free T should be calculated. Late-onset hypogonadism (LOH) can be considered in patients with low total T (<8 nmol/L) or with free T comprising less than 2% of total T; *courtesy of Eviatar Fields, PhD, Naeem Bhojani, MD, Bilal Chughtai, MD and Dean Elterman, MD.*

Diagnosis of Andropause

Canadian guidelines emphasize that biochemical evaluation be undertaken in conjunction with a thorough clinical history and physical examination. Thus, routine screening of men with non-specific symptoms such as fatigue, stress, or new-onset sexual dysfunction, is not recommended. Testosterone testing should be considered in men presenting with persistent sexual symptoms, including reduced libido or impaired quality of erections, especially after an inadequate response to phosphodiesterase-5 inhibitors. Other indications include unexplained sarcopenia and obesity in the absence of other metabolic syndrome components.[3]

The diagnostic criteria for andropause are not straightforward; however, the *Canadian Medical Association Journal* (CMAJ) provides official guidelines (**Figure 1**).[3,8] In men with a positive history and physical examination, the initial workup should include measurement of total testosterone levels. This blood sampling must be performed in the morning, ideally between 7:00 and 11:00 AM, to account for the circadian pattern of testosterone secretion and to avoid false-positive results. While Canadian guidelines do not specify a formal diagnostic threshold value for total testosterone, other international guidelines suggest that levels below 8 nmol/L are indicative of testosterone deficiency.[8] Investigation into reversible causes of hypogonadism is strongly recommended. Conversely, investigation for hypogonadism is suggested for men with unexplained anemia or sarcopenia of undetermined origin.[3]

Management of Andropause

Non-pharmacological Management

Non-pharmacological management of andropause should constitute first-line therapy for andropause and revolves around education and lifestyle modification aimed at managing comorbidities. Weight management is among the most impactful modifications that men could make, as adipose tissue forms a bidirectional relationship with testosterone. First, adipose tissue expresses high levels of aromatase, which converts testosterone to estradiol; elevated estradiol, in turn, inhibits hypothalamic gonadotropin release, which reduces testosterone production.[15] Conversely, low testosterone levels also trigger deposition of visceral adipose tissue, forming a vicious cycle that further reduces testosterone levels.[15] Thus, significant loss of visceral fat can increase serum total testosterone levels.

Similarly, exercise has been shown to increase testosterone levels and improve androgen receptor sensitivity.[16] Resistance training provides the best benefit for testosterone production and release,[17] while the added benefit of increased lean mass and management of comorbidities such as obesity and diabetes further improves testosterone levels. Sleep hygiene should also be optimized, as testosterone production follows a circadian pattern, with peak plasma testosterone levels occurring in the morning; unsurprisingly, sleep restriction is known to reduce testosterone levels.[18] Lifestyle modifications targeting other reversible causes of testosterone deficiency—including medications, nutrition, extreme aerobic exercise, obstructive sleep apnea, and acute illness—should also be considered.[1]

Pharmaceutical Options

The current Canadian guidelines for testosterone deficiency strongly recommend TRT in men with biochemical and clinical evidence of testosterone deficiency, provided no contraindications are present.[1,3] The goals of therapy are to relieve symptoms of testosterone deficiency through restoration of the patient's testosterone levels to physiological levels (14–17.5 nmol/L);[8] the guidelines acknowledge biological variability.[3]

Various formulations of TRT are available in Canada, including short-acting injectables, oral formulations, and transdermal delivery systems such as patches, gels, and solutions (see Table 3 in [1]). Selection of the appropriate formulation should be made through shared decision making between the physician and patient, based on the patient's goals and lifestyle. Given that exogenous testosterone suppresses gonadotropin production leading to infertility, alternative therapies, such as human chorionic gonadotropin, aromatase inhibitors, and selective estrogen receptor modulators are indicated in patients wishing to remain fertile.[3,13]

Monitoring and Safety

The safety profile of TRT remains an active area of study, and the risks of treatment should be balanced with the goals of the patient. Current evidence suggests that testosterone treatment does not increase the risk of developing prostate

cancer,[12] although patients with active prostate cancer should consult a urologist prior to initiation of TRT.[1] Similarly, there is no evidence suggesting that TRT worsens symptoms of benign prostatic hyperplasia, and some patients may experience improvements in lower urinary tract symptoms.[1] Furthermore, while earlier studies raised concerns regarding cardiovascular risk, the TRAVERSE trial demonstrated that TRT does not increase the incidence of cardiac events in patients who are at risk of- or have preexisting- cardiovascular disease risk.[19] TRT can thus be provided to men with stable cardiovascular disease if appropriately monitored.[1,3]

Ongoing monitoring for adverse effects is critical to ensure the success and safety of TRT (see Table 4 in [1]). A baseline assessment is recommended prior to the initiation of treatment, followed by follow-up at 3, 6, and 12 months to evaluate symptoms, side effects, prostate specific antigen and serum testosterone levels, with or without assessment of other gonadotropins.[3] Hematocrit levels are important to evaluate at baseline, as well as at 3 and 12 months to monitor for polycythemia. A baseline digital-rectal exam is recommended, with annually assessments thereafter. A review of medications should be conducted in patients who undergo TRT, as testosterone can interact with numerous medications and increase the risk of hepatotoxicity, bleeding, and cardiac or thrombolytic events.[20]

Conclusions

Andropause is a heterogenous and often over-diagnosed condition, largely due to inconsistent guidelines and the non-specific nature of its symptoms. Diagnostic criteria require both biochemical confirmation in conjunction with clinical symptoms. While lifestyle modification remains first-line therapy, selected men with confirmed testosterone deficiency can benefit from TRT, provided that potential risks of testosterone therapy are considered. Ultimately, primary care physicians play a central role in the diagnosis and treatment of andropause.

Correspondence

Dean Elterman, MD
Email: dean.elterman@uhn.ca

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ABOUT THE AUTHOR

Michael Boivin, Rph, CDE, CBE

Michael Boivin is a clinical pharmacist consultant, continuing education developer and president of CommPharm Consulting Inc. In 2009, he left full-time pharmacy practice to pursue a career in continuing education and consulting. He has developed in excess of 500 accredited continuing education activities for pharmacists, family physicians, specialists and allied healthcare professionals. In 2024, he was recognized for his work with a lifetime achievement award from the Ontario Pharmacists' Association.

Affiliations: *CommPharm Consulting, Barrie, Ontario*

Chronic Pain in Primary Care: A Practical Guide to What Works (and What Does Not)

Michael Boivin, Rph, CDE, CBE

Introduction

Chronic non-cancer pain is a leading driver of disability worldwide.[1] It is defined as pain persisting beyond normal tissue healing time (>3 months).[1] Chronic pain is among the most prevalent conditions encountered in primary care.[2] An estimated 7.6 million people, approximately 1 in 5, live with chronic pain.[3] Pain affects many aspects of a person's life, including their ability to work, attend school ,and participate in family and community life.[2]

Although multiple treatment options are available for the management of chronic pain, it is one of the most complex and difficult conditions to treat.[3] Even with optimal treatment, complete alleviation of pain is not a realistic treatment goal.[4] For most patients, the goals focus on minimizing suffering and maximizing functionality.[4] Management is further complicated by the heterogeneity of chronic pain, as its sources can vary and therefore require different treatment options.

Management Strategy Varies Based on Cause of Chronic Pain

Over the past several years, several guidelines for the management of chronic pain have been updated. This article will provide clinicians with a concise update on the management of the most common chronic pain disorders encountered in clinical practice:

1. Osteoarthritis
2. Chronic low back pain
3. Neuropathic pain
4. Fibromyalgia

For each condition, both the non-pharmacological and pharmacological treatment options are reviewed. Patient and provider resources are also provided for each condition.

Osteoarthritis

Osteoarthritis (OA) is a highly prevalent condition. It is estimated that 13.6% of Canadians aged ≥20 years are living with OA.[5] While the affected joints can vary, knee OA accounts for the largest site-specific burden, followed by hand and hip OA.[6] **Table 1** provides treatment options along with patient and provider resources.

Chronic Low Back Pain

Most people will experience low back pain (LBP) at some point in their lifetime.[12] In 2020, approximately 1 in 13 people globally were affected by LBP, representing a 60% increase in cases since 1990.[12] For most patients with acute LBP, the prognosis is excellent with complete resolution of symptoms within 4 to 6 weeks.[13] Unfortunately, those with persistent LBP have a very poor prognosis, and recovery is often unlikely.[13] No curative treatments exist for chronic LBP.[13] The goal is to reduce pain, disability, and the long-term consequences of chronic pain.[13] **Table 2** provides treatment options and includes relevant patient and provider resources.

Neuropathic Pain

Neuropathic pain is estimated to affect between 2% to 3% of the population.[16] Neuropathic pain encompasses a large number of conditions including postherpetic neuralgia, diabetic neuropathy, spinal cord injury, multiple sclerosis, post-stroke pain, tumour compression, and drug/condition induced neuropathy.[17] The prevalence of diabetic neuropathy and postherpetic neuralgia are expected to increase with population aging and rising rates of obesity.[16] **Table 3** outlines treatment options along with patient and provider resources.

Fibromyalgia

Fibromyalgia (FM) is estimated to affect 1% to 5% of the population[20] It is the third most common musculoskeletal condition after LBP and OA.[21] FM is characterized by chronic, generalized pain and is accompanied by a range of somatic and psychological symptoms, such as fatigue, sleep disturbances, and anxiety.[20] Unlike other pain conditions, FM can affect the whole body, from head to toe.[21] The pain is commonly described as being similar to neuropathic pain, with 20–30% of patients reporting paresthesia in their limbs, hands, or trunk.[21] **Table 4** summarizes available treatment options along with patient and provider resources.

What Does this Mean for Your Patients with Chronic Pain?

Managing chronic pain in primary care requires a fundamental shift away from pursuing complete pain relief toward prioritizing functional improvement and reduced suffering. As outlined throughout this article, even with optimal treatment, most patients will continue to experience ongoing symptoms. For the majority of chronic pain conditions, the management approach is multimodal, combining non-pharmacological strategies (e.g., exercise, cognitive behavioural therapy) with targeted pharmacological therapies.

Patients should be counselled to assess a treatment's success based on their ability to return to work, school, and community life, rather than solely on reductions in pain intensity. For primary care clinicians, this means helping patients understand that progress is often gradual and highly individualized. Even when pain does not fully resolve, meaningful improvements in mobility, sleep, mood, and daily functioning are achievable and represent clinically important outcomes.

Osteoarthritis (OA)	
Non-Pharmacological	
Exercise	• **Exercise is foundational to OA management**[7,8] • No single exercise type is universally superior; the key is encouraging patients to be as active as possible • Exercise programs should be tailored to the individual patient
Assistive Devices	• Appropriate footwear, walking aids, and assistive devices can reduce pain and improve function and mobility confidence[7]
Acupuncture / Hot-Cold Therapy	• Evidence supporting these adjunctive therapies is limited, and guideline recommendations remain inconsistent
Nutraceuticals	
Chondroitin	• **NOT recommended** by current guidelines for OA management[8]
Glucosamine	• **NOT recommended** by current guidelines for OA management[8]
Fish Oil / Omega-3	• **NOT recommended** by current guidelines for OA management[8]
Pharmacological	
Topical non-steroidal anti-inflammatory drugs (NSAIDs)	• **First-line** therapy for OA of the hand and knee[6] • Similar efficacy to oral NSAIDs with an excellent safety profile[6]
Oral NSAIDs	• Effective in reducing overall pain in OA, with no strong evidence that any one NSAID is superior to another[9] • Should be used at the lowest dose for the shortest duration due to gastrointestinal, cardiovascular, and renal risks[6] • Diclofenac and COX-2 selective agents carry the highest cardiovascular risk, whereas naproxen has a lower cardiovascular risk[10]
Acetaminophen	• Evidence for meaningful benefit is limited, with small effect sizes reported[11] • Best reserved for patients in whom NSAIDs are contraindicated or not tolerated[11]
Intraarticular Corticosteroids	• Supported for short-term pain relief, particularly in knee OA[8,11] • Guideline recommendations for hip OA are less consistent[8,11] • A maximum of four injections per knee joint per year is recommended[10]
Duloxetine	• Consider in patients with chronic pain amplification, central sensitization, or comorbid mood symptoms[6,10] • Benefits appear modest[6,10]
Opioids	• **Generally NOT recommended** by guidelines due to limited sustained benefit and significant associated risks[6] • For most patients, the risks greatly outweigh the benefits
Resources	
Clinician Resources	• **CEP: Osteoarthritis Tool** • **PEER Simplified Decision Aid: OA Treatment Options in Primary Care**
Patient Resources	• **Arthritis Society of Canada: Exercises for OA of the Hip & Knee** • **Andrea Furlan MD: Twenty Exercises for OA of Hip and Knees (Video)**

Table 1. Osteoarthritis (OA); *courtesy of Michael Boivin, Rph, CDE, CBE.*

Chronic Low Back Pain (LBP)	
Non-Pharmacological	
Exercise	• Moderate-quality evidence suggests small improvements in pain relief and function[14]
Acupuncture	• Evidence for improvement in pain relief is limited, with no demonstrated improvement in function[14]
Massage	• Provides short-term pain relief and improvement in function[14]
Spinal Manipulative Therapy	• Provides short-term improvements in pain and function[12] • Should be used as an adjunct to other therapies rather than as monotherapy[12]
Pharmacological	
Oral Non-steroidal Anti-inflammatory Drugs (NSAIDs)	• Associated with small improvements in pain and function[14] • No differences in efficacy have been demonstrated between individual NSAIDs[14]
Antidepressants (Serotonin–norepinephrine Reuptake Inhibitors [SNRIs] / Tricyclic Antidepressants [TCAs])	• Both duloxetine (an SNRI) and tricyclic antidepressants are associated with clinically meaningful pain reduction (>30%) vs placebo[3,15]
Acetaminophen	• **No significant evidence of benefit** in chronic LBP[13]
Capsaicin	• **No significant evidence of benefit** in chronic LBP[13]
Muscle Relaxants	• **No significant evidence of benefit** in chronic LBP[13]
Opioids	• Strong opioids associated with small short-term improvements in pain and function[14] • For most patients, the risks, including dependence and tolerance, generally far outweigh the benefits[3]
Resources	
Clinician Resources	• **CORE Back Tool** • **C-TOP Tool: Comparing Treatment Options for Low Back Pain**
Patient Resources	• **RAC-LBP: Exercise Videos for Low Back Pain**

Table 2. Chronic Low Back Pain (LBP); *courtesy of Michael Boivin, Rph, CDE, CBE.*

Neuropathic Pain	
Non-Pharmacological	
Exercise	• Evidence for the reduction of neuropathic pain is inconsistent[18]
TENS	• Limited evidence demonstrating pain reduction[19] • Tolerability is excellent[19]
Cognitive Behavioural Therapy (CBT) / Psychotherapy	• Evidence for pain intensity reduction in neuropathic pain is limited[19]
Acupuncture	• Overall efficacy for neuropathic pain remains limited[18,19]
Pharmacological	
Gabapentinoids (First-Line)	• Demonstrates efficacy in peripheral neuropathy[16] • **Pregabalin:** Provides analgesic benefit in chronic central neuropathic pain after spinal cord injury and offers secondary benefits (improved sleep, reduced anxiety) in central post-stroke pain[16]
Serotonin–norepinephrine Reuptake Inhibitors (SNRIs) (First-Line)	• Demonstrates efficacy in post-herpetic neuralgia[16] • Both duloxetine and venlafaxine are recommended; duloxetine is preferred based on a stronger level of evidence[19]
Tricyclic Antidepressants (TCAs) (First-Line)	• Commonly recommended first-line therapy although evidence is less robust than that for gabapentinoids/SNRIs[16,17,19] • The PEER guideline does not recommend use for chronic neuropathic pain[3] • Associated with important adverse effects and safety considerations, especially in older adults[17,19]
Opioids (Second-Line)	• **Tramadol:** A weak opioid receptor agonist with weak SNRI activity; associated with benefit in diabetic neuropathy and mixed neuropathic pain syndromes[16] • Other opioids are more effective than placebo, but for most patients, the risks outweigh the benefits[16]
Cannabinoids (Third-Line)	• Trial evidence demonstrating significant improvements in neuropathic pain is limited[19] • The risk profile tends to be lower than that of opioids for most patients[16] • Patients should avoid smoking cannabis and discontinue if there is no clear benefit[16]
Resources	
Clinician Resources	• C-TOP Tool: Comparing Treatment Options for Neuropathic Pain • Pharmacologic Management of Chronic Neuropathic Pain (CFP)

Table 3. Neuropathic Pain; *courtesy of Michael Boivin, Rph, CDE, CBE.*

Fibromyalgia (FM)	
Non-Pharmacological	
Patient Education	• **Education is the first step** in FM management[20] • Reassure patients that FM is a legitimate pathological condition and that their symptoms and suffering are real[21] • FM is not a progressive disease and not caused by peripheral tissue damage[21] • Encourage good sleep hygiene, relaxation techniques, and stress reduction strategies
Exercise	• **These approaches are essential** and strongly recommended by guidelines[20,22] • Moderate-quality evidence supports aerobic, resistance, and stretching exercises, which provide small improvements in quality-of-life, pain, and function[23] • Aquatic therapy has been shown to reduce pain and fatigue[20]
Cognitive Behavioural Therapy (CBT)	• **Considered the most effective psychological therapy** for FM[20] • Demonstrates superiority over other psychological treatments for short-term reductions in pain intensity[20] • Also improves sleep quality and duration[20]
Acupuncture	• Evidence remains limited; however, some trials report a higher proportion of patients achieving a 30% pain reduction[22] • Could be considered for patients not responding to or unwilling to pursue other options
Pharmacological	
Tricyclic antidepressants (TCAs) (Amitriptyline)	• Increases the proportion of patients achieving a ≥30% pain reduction[22] • Associated with a modest effect on sleep, small reductions in fatigue[22] • Benefits appear independent of mood effects, with effective doses (25 mg/day) lower than those used for depression[20,22] • Safety concerns limit use in older adults
Gabapentinoids	• **Pregabalin:** Increases the proportion of patients achieving a ≥30% pain reduction vs placebo, with small benefits for fatigue and sleep[22] • Gabapentin: Insufficient evidence to determine efficacy in FM[23]
Serotonin–norepinephrine reuptake inhibitors (SNRIs)	• **Duloxetine and milnacipran** are both more effective than placebo for reducing FM pain[21] • Associated with small improvements in sleep and disability, with no effect on fatigue[22]
Muscle Relaxants	• **Cyclobenzaprine:** Structurally related to TCAs; improves pain and quality-of-life (especially sleep) but not fatigue[21] • Adverse effects are significant, with a meta-analysis reporting high rates of treatment-emergent effects (85%) and only 71% study completion[22]
Analgesics (acetaminophen [APAP], non-steroidal anti-inflammatory drugs (NSAIDs), Opioids)	• **Limited role** for opioids, NSAIDs, or acetaminophen in FM[21] • These agents do not target central sensitization and have not been shown to reduce pain in FM[23]
Resources	
Clinician Resources	• Primary Care Pathway: Fibromyalgia • CEP: Fibromyalgia Tool • AAFP: Fibromyalgia – Diagnosis and Management
Patient Resources	• Arthritis Society of Canada: What is Fibromyalgia? • Dr. Andrea Furlan: What is Fibromyalgia? (Video)

Table 4. Fibromyalgia (FM); *courtesy of Michael Boivin, Rph, CDE, CBE.*

Correspondence

Michael Boivin, Rph, CDE, CBE
Email: mike@commpharm.com

Financial Disclosures

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ABOUT THE AUTHOR

Zale Mednick, MD

Dr. Zale Mednick is a board-certified Ophthalmologist in Canada and in the United States. He completed his medical school and subsequent Ophthalmology residency at Queen's University. Following residency, he completed his Fellowship training in Cornea and Refractive Surgery at the University of Toronto. Dr. Mednick is currently on staff at Humber River Hospital, North York General Hospital, and the Bochner Eye Institute. Dr. Mednick produces and hosts several podcasts, including the award-winning Preconceived podcast, and the Ophthalmology-focused Blind Spot podcast.

Affiliations: *North York General Hospital, Toronto, ON*
Humber River Hospital, Toronto, ON
Bochner Eye Institute, Toronto, ON

Refractive Surgery Primer for Primary Care

Zale Mednick, MD

Introduction

When a patient expresses a desire to become 'glasses-free,' the conversation that follows can quickly become overwhelming. Patients often have a specific procedure in mind (most commonly laser-assisted *in-situ* keratomileusis [LASIK]) and can become frustrated if this is not the primary option recommended. From the physician's perspective, it can be challenging to simplify a vast array of options without confusing the patient. In this article, I aim to offer a simplified approach for navigating the many contemporary refractive surgery options. The goal is to provide both patients and physicians with an overview of the currently available treatment modalities and to elucidate the multiple variables that influence the surgical decision-making process, guiding the selection of the most appropriate surgery for each patient (**Table 1**).

What are the Main Variables to Consider?

When a patient presents for a refractive surgery consultation, two primary factors must be considered: efficacy and safety.

Efficacy

The primary question is which procedure is most effective to achieve the patient's goal of reducing or eliminating the need for glasses. Certain glasses prescriptions are more amenable to corneal procedures such as LASIK or photorefractive keratectomy (PRK), while others are better treated with lens-based procedures such as implantable Collamer lenses (ICL) or refractive lens exchange (RLE).[1-3] Key considerations include whether the patient is myopic (nearsighted) or hyperopic (farsighted). In cases where the glasses prescription is low or very high, even initially successful procedures may lose effectiveness over time, resulting in a return to dependence on glasses. For patients seeking to become glasses-free, it is therefore essential to select the procedure most likely to achieve that goal.[1,2]

Safety

While rare, the main complication refractive surgeons seek to avoid is corneal ectasia.[4,5] Corneal ectasia occurs when the cornea decompensates after excessive

Corneal Procedures	Indications	Advantages	Disadvantages
LASIK	• Normal corneas	• Quick recovery • Minimal pain • Treats a wide range of prescriptions	• Higher ectasia risk than with other corneal procedures • Increased dryness risk compared with other corneal procedures
PRK	• Normal corneas • Thinner corneas	• Lower ectasia risk than with LASIK	• Slower healing, longer recovery time • More postoperative pain than with LASIK
SMILE	• Normal corneas • Thinner corneas	• A good option for patients with concerns about dryness	• Less long-term data than with LASIK and PRK • Not indicated for hyperopia
Lens Procedures			
ICL	• Abnormal corneas • Very high prescriptions	• Can correct significantly higher prescriptions compared to the corneal procedures	• Additional risks of any intraocular procedure
RLE	• Over the age of 55 • Not a candidate for other refractive options	• More appropriate for older patients who are already presbyopic	• Additional risks of any intraocular procedure • Possible dependence on reading glasses

Table 1. Refractory surgery options; *courtesy of Zale Mednick, MD.*

Abbreviations: ICL: implantable Collamer lenses; **LASIK:** laser-assisted in-situ keratomileusis; **PRK:** photorefractive keratectomy; **RLE:** refractive lens exchange; **SMILE:** small incision lenticule extraction

weakening, resulting in outward bulging. When ectasia develops, the cornea transitions from a spherical shape to a more 'cone-like' shape. Ectasia can present as a primary condition, known as keratoconus, or as a secondary condition in patients who have undergone certain refractive surgeries.[4,5]

During the preoperative assessment, one of the main goals of the ophthalmologist is to screen for patients whose corneas may be at risk of such decompensation.[5,6] The risk of ectasia is a particular concern when the surgery in consideration involves reshaping the cornea, as is the case with LASIK, PRK, and small incision lenticule extraction (SMILE).[4,5] Each of these procedures involves thinning and structurally weakening the cornea. Tissue is being ablated or removed from the cornea, resulting in a mechanically weakened structure.[5] When corneal integrity is compromised, the tissue becomes more susceptible to bulging and the development of ectasia.

A variety of diagnostic tests can help us identify corneas at risk for ectasia; however, two of the most fundamental criteria are whether the cornea is too thin at baseline, and a history of eye rubbing.[6,7] When a cornea is thin before surgery, there is a limited margin of corneal tissue that can be safely removed during the refractive procedure. Furthermore, reduced corneal thickness may reflect an underlying weakness that has not yet manifested clinically as ectasia. Eye rubbing represents a significant modifiable risk factor, as it is strongly associated with both keratoconus progression and post-operative corneal instability.[6,7] Even in cases in which a preoperative cornea appears normal, chronic eye rubbing after surgery may contribute to ectatic changes in susceptible individuals.[6]

Cornea-based Refractive Surgeries

Laser-Assisted In-Situ Keratomileusis

When most people think of refractive surgery, they typically are referring to LASIK, and for good reason. LASIK is one of the most extensively studied elective surgical procedures in medicine, with over 25 years of outcome data.[1,2,8]

The average cornea measures approximately 540 microns in thickness.[5] In the first step of the LASIK procedure, a femtosecond laser is used to create a corneal flap measuring approximately 90–120 microns in thickness.[8] In the second step, the flap is lifted to expose the underlying cornea, which is subsequently ablated using an excimer laser according to the patient's refractive error.[1,2] The flap is then repositioned without sutures, and adheres to the underlying cornea via natural hydrostatic forces.

LASIK has demonstrated high levels of efficacy, predictability, and patient satisfaction.[1,2,8] Visual recovery is rapid, with many patients achieving uncorrected visual acuity of 20/20 or better shortly after surgery.[1,2] Post-operative discomfort is typically mild and transient, and refractive predictability is particularly strong in cases of low to moderate myopia.[1,2] Taken together, these advantages make LASIK the preferred option for most patients who are eligible for the procedure.

Photorefractive Keratectomy

PRK predates LASIK and remains a well-established procedure supported by long-term safety data.[3,9] In PRK, the corneal epithelium is removed, allowing the excimer laser to directly ablate the anterior stroma.[3,9] Because no corneal flap is created, corneal biomechanical integrity may be better preserved in certain patients, potentially reducing the risk of ectasia compared to LASIK.[5]

Because the epithelium is removed—essentially causing a corneal abrasion—visual recovery following PRK is slower than with LASIK, and early post-operative discomfort is more pronounced.[3,9] A bandage contact lens is typically used during re-epithelialization. Despite these differences in recovery, long-term visual outcomes are comparable to those of LASIK in appropriately selected patients.[1,3]

PRK is associated with a small risk of corneal haze, particularly in cases involving higher refractive corrections.[9,10] The intraoperative application of mitomycin C has significantly reduced the incidence of clinically significant haze.[10] However, both refractive regression and haze are more common in higher myopic treatments.[9,10]

Given the post-operative pain and lengthier healing process, PRK is typically reserved for patients who are not candidates for LASIK.

Small Incision Lenticule Extraction

SMILE is a newer refractive surgery technique that has been in clinical use for more than a decade. It is more commonly performed in Europe, where it was developed, as well as in Asia. While SMILE is increasingly adopted among surgeons in North America, LASIK remains the primary refractive surgery option offered to patients by most ophthalmologists.

SMILE is a flapless refractive procedure in which a femtosecond laser creates an intrastromal lenticule that is removed through a small incision.[11] By avoiding flap creation, SMILE may preserve a greater proportion of anterior corneal nerves, potentially reducing early post-operative dry eye symptoms compared to LASIK.[11,12]

Studies suggest similar refractive efficacy and safety profiles between SMILE and LASIK for treating myopia.[11] Some studies demonstrate faster recovery of corneal sensitivity and tear film parameters after SMILE; however, whether this translates to long-term differences in dryness remains an area of ongoing debate.[12] Currently, SMILE is primarily indicated for myopia and myopic astigmatism and is not approved for hyperopic correction in most regions.[11]

As longer-term data emerges, SMILE may gain broader acceptance and may even overtake LASIK as the procedure of choice. For now, however, LASIK remains the more commonly performed option in North America.

Lens-based Procedures

Implantable Collamer Lenses

In some cases, neither LASIK, PRK, nor SMILE are appropriate treatment options. For patients with high refractive errors or thin corneas unsuitable for corneal laser surgery, phakic intraocular lenses (IOL) such as the ICLs offer an effective alternative.[13] ICLs are implanted within the eye, positioned posterior to the iris and anterior to the crystalline lens.

The benefit of ICLs is that they expand the range of patients eligible for treatment while avoiding any corneal ectasia risk. ICLs can correct a wide range of myopia and hyperopia, often exceeding the effective treatment range of corneal laser procedures.[13] Multiple studies have demonstrated excellent visual outcomes, high predictability, and good long-term stability with ICL.[13] Whereas LASIK typically corrects up to -10.00 Diopters of myopia and +4.00 Diopters of hyperopia, ICLs can address a much wider range, from approximately -3.00 to -18.00 Diopters of myopia and +3.00 and +10.00 Diopters of hyperopia. While such extreme prescriptions are uncommon, these patients often derive the greatest benefit from refractive surgery.

The limitation of ICLs, however, is that implantation is an intraocular procedure. In corneal procedures, which do not involve entering the eye, there is no real concern about endophthalmitis, glaucoma, or cataract formation. Because ICL implantation is an intraocular procedure, it carries risks inherent to intraocular surgery, including infection, cataract formation, increased intraocular pressure, and endothelial cell loss.[13] Advances in lens design, particularly the introduction of models with central ports, have reduced the risk of pupillary block and improved safety profiles.

Refractive Lens Exchange

In some patients, even ICL implantation is not an option. Safe ICL placement requires a certain amount of intraocular space, and in smaller eyes, there may be inadequate room to accommodate the additional lens. In such patients, an RLE can be considered. RLE is essentially cataract surgery performed for refractive purposes,[14] in which the crystalline lens is removed and replaced with an IOL to correct refractive error.

RLE is generally considered in older patients, particularly those approaching presbyopia or early cataract formation.[14,15] While modern multifocal and extended depth-of-focus IOLs provide an improved range of vision, they do not fully replicate the accommodative capacity of the natural crystalline lens.[15] Thus, RLE is generally not offered to younger patients, such as those in their 20s or 30s.

As an intraocular procedure, RLE carries the same risks associated with cataract surgery, including retinal detachment (particularly in high myopes), endophthalmitis, cystoid macular edema, and posterior capsule opacification.[14]

Summary

Modern refractive surgery offers a range of effective options that can be tailored to a patient's refractive error, corneal anatomy, age, and visual goals.[1-3] LASIK and PRK remain preferred procedures with extensive long-term safety and efficacy data.[1-3,8,9] SMILE offers a flapless alternative with potentially reduced early dry eye symptoms.[11,12] ICLs expand treatment options for patients with high refractive errors or thin corneas,[13] while RLE is generally reserved for older patients and those approaching presbyopia.[14,15]

Patients should be reassured that many of these procedures are indeed highly safe and are supported by long-term data. At the same time, they should be counselled that all surgical procedures carry inherent risks, and even seemingly 'routine cases' can have unexpected complications. Although LASIK has a reputation for being a straightforward procedure, it remains surgery. Patients should therefore carefully consider their motivations for seeking independence from glasses and whether the benefits outweigh the risks.

Correspondence

Zale Mednick, MD
Email: zalemednick@gmail.com

Financial Disclosures

Z.M.: Thea Pharma Canada

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ABOUT THE AUTHOR

Dilpriya K. Mangat, MD, FRCPC, MBA

Dr. Dilpriya K. Mangat is an Internal Medicine and Geriatric Medicine specialist based in Calgary, Alberta. She is the Founder and CEO of NexCerebrum Brain Health and Memory Wellness, a clinic dedicated to advancing early detection, prevention, and personalized care in cognitive health. Dr. Mangat is a Clinical Associate Professor at the University of Calgary and is affiliated with the Hotchkiss Brain Institute. Her work focuses on integrating clinical care with emerging biomarkers, brain health optimization, and innovative models of cognitive longevity. She is also an active speaker and contributor to national conversations on brain health, dementia prevention, and the evolving "brain economy."

Affiliations: *Founder & CEO, NexCerebrum Brain Health and Memory Wellness*
Clinical Associate Professor, University of Calgary, Calgary, AB
Affiliated Member, Hotchkiss Brain Institute
Staff Physician, Alberta Health Services (Rockyview General Hospital & Seniors Health)

Parkinson's Disease in Primary Care: A Practical Canadian Primer

Dilpriya K. Mangat, MD, FRCPC, MBA

Executive Summary

Parkinson's disease (PD) is a progressive neurodegenerative disorder that is increasingly encountered in Canadian primary care. National surveillance data estimate that over 110,000 Canadians are living with Parkinsonism, with incidence rising steadily as the population ages.[1] Approximately 80% of new diagnoses occur after the age of 65 years, making PD highly relevant to geriatric-focused primary care practice. For family physicians, PD represents not only a diagnostic challenge but also a longitudinal care responsibility that intersects with multimorbidity, polypharmacy, fall risk, and caregiver burden.

The diagnosis of PD remains clinical. It requires recognition of Parkinsonism, defined as bradykinesia in combination with rest tremor and/or rigidity, and on an assessment of whether the clinical features support idiopathic PD versus alternative etiologies such as drug-induced, vascular, or atypical Parkinsonism.[2] In primary care, a structured approach that incorporates a focused history, bedside motor examination, and screening for non-motor symptoms can significantly improve diagnostic accuracy.[3]

Management begins with patient education, early initiation of exercise, and symptom-guided pharmacotherapy. Levodopa remains the most effective treatment for motor symptoms and should not be delayed once functional impairment emerges.[4] Primary care clinicians are well positioned to initiate therapy, monitor therapeutic response, and coordinate multidisciplinary care, particularly in regions where access to neurology is limited.

Epidemiology and Clinical Relevance

PD represents one of the fastest growing neurological conditions globally, and Canada reflects this trend. Data from the Canadian Chronic Disease Surveillance System demonstrate a steady increase in the number of individuals living

with Parkinsonism over the past decade, driven largely by demographic shifts and improved survival.[1] While PD accounts for the majority of these cases, the category of Parkinsonism also includes vascular, drug-induced, and atypical syndromes, adding to the diagnostic complexity faced in primary care.

For clinicians, the significance of PD extends beyond its prevalence to its impact. Patients frequently present with subtle early symptoms that may be attributed to aging, stress, or comorbid conditions. As the disease progresses, it affects mobility, cognition, mood, and autonomic function, requiring a comprehensive and longitudinal care approach. Primary care clinicians are uniquely positioned to identify early changes, initiate appropriate investigations, and provide continuity of care as the disease progresses.

Diagnosis: A Clinical Framework

Confirming Parkinsonism

The first step in diagnosing PD is confirming the presence of Parkinsonism (**Figure 1**). According to the Movement Disorder Society clinical diagnostic criteria, Parkinsonism is defined by bradykinesia in combination with either rest tremor or rigidity.[2] Bradykinesia is more than slowness; it is characterized by a progressive reduction in speed or amplitude during repetitive movements.

In clinical practice, this can be assessed using simple bedside manoeuvres. Finger tapping is particularly useful, as patients with PD demonstrate a clear decrement in amplitude over successive taps. Rapid alternating movements and heel tapping can provide additional confirmation. Rigidity may be appreciated as increased resistance to passive movement and may have a cogwheel quality. When present, rest tremor is typically asymmetric and most evident when the affected limb is relaxed.

Gait assessment also provides valuable information. Patients may exhibit reduced arm swing, a stooped posture, shuffling steps, and difficulty with turning. Taken together, these findings support the diagnosis of Parkinsonism and guide further evaluation.

Clinical History

A detailed history is essential for distinguishing PD from other causes of Parkinsonism. Patients often report subtle functional changes that precede development of obvious motor symptoms, such as difficulty with fine motor tasks including buttoning clothing, changes in handwriting (micrographia), or reduced dexterity.

Non-motor symptoms frequently precede the onset of motor features and can provide important diagnostic clues. Common early manifestations include constipation, reduced sense of smell, REM sleep behaviour disorder, and mood changes such as depression or anxiety.[5] Identifying these features can increase diagnostic confidence and support earlier recognition.

A thorough medication history is particularly important. Dopamine receptor–blocking agents, including antipsychotics and certain antiemetics such as metoclopramide, can cause drug-induced Parkinsonism. Although this condition may mimic PD, it often presents with more symmetric symptoms and may improve after discontinuation of the offending agent.[6]

Differential Diagnosis

Distinguishing idiopathic PD from other causes of Parkinsonism is a critical task in primary care (**Table 1**).

Atypical Parkinsonian syndromes, such as progressive supranuclear palsy and multiple system atrophy, should be considered when red flags are present. These include early falls, rapid disease progression, prominent autonomic dysfunction, oculomotor abnormalities, and poor response to levodopa.[7] These features warrant early referral to a specialist.

Vascular Parkinsonism typically presents with lower-body predominance, gait instability, and a history of cerebrovascular disease. Tremor is less common, and response to levodopa is often limited.[8]

Essential tremor is another common mimic. Unlike PD, essential tremor is primarily an action tremor, often bilateral, and not associated with bradykinesia or rigidity.[9]

Role of Imaging

PD remains a clinical diagnosis, and routine neuroimaging is not required.[4] However, imaging may be useful in selected cases where diagnostic uncertainty exists or alternative pathology is suspected.

Dopamine transporter imaging (DaT-SPECT) can help differentiate degenerative Parkinsonism from conditions such as essential tremor or drug-induced Parkinsonism. However, it does not reliably distinguish PD from atypical Parkinsonian syndromes and is not widely accessible across

Stepwise approach to diagnosis and early management of Parkinson's disease in primary care.

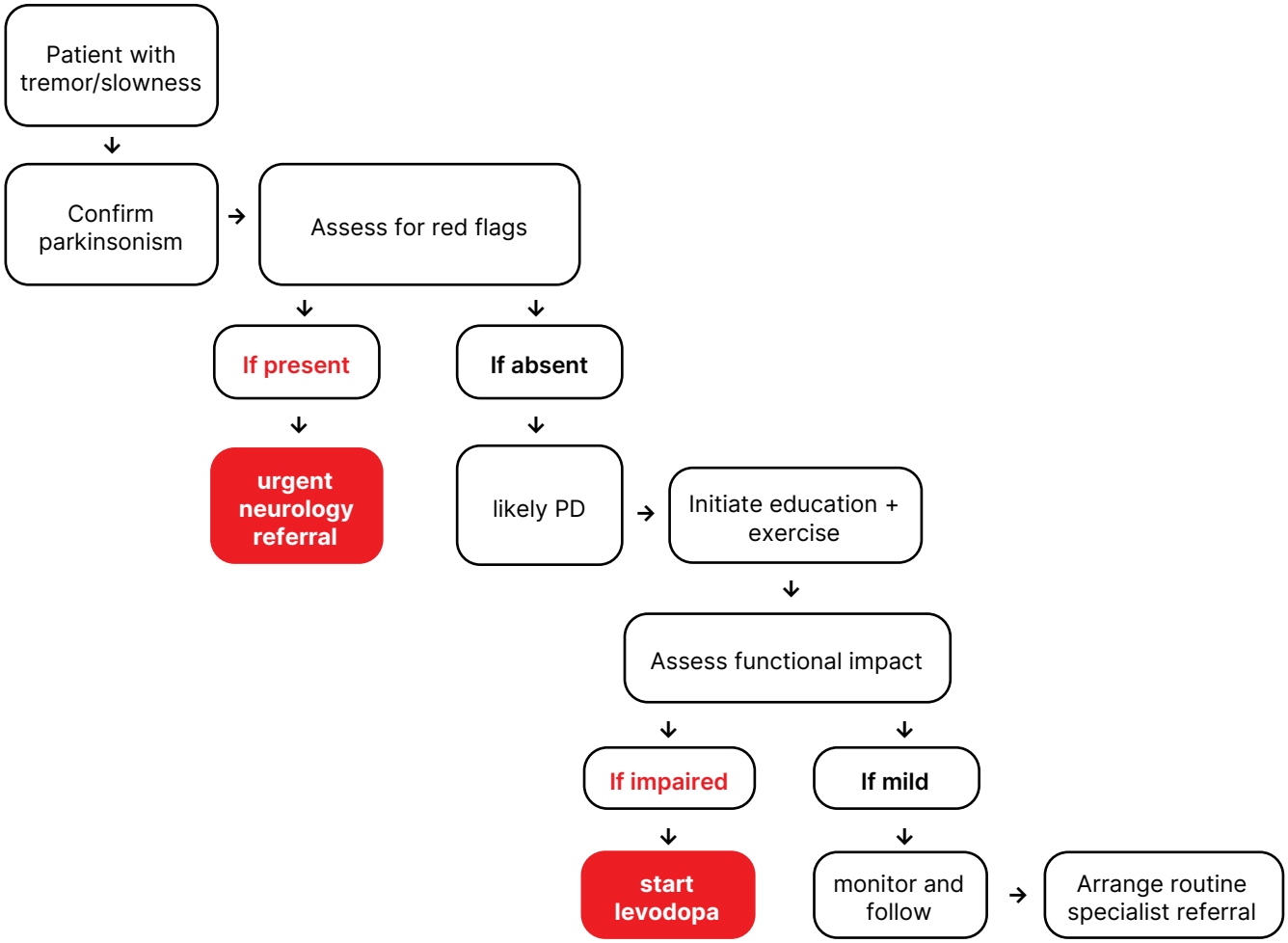

Figure 1. Primary Care Approach to Suspected Parkinson's Disease; *courtesy of Dilpriya K. Mangat, MD, FRCPC, MBA.*

Feature	Parkinson Disease	Atypical Parkinsonism	Drug-Induced	Vascular	Essential Tremor
Onset	Asymmetric	Often rapid/symmetric	Symmetric	Lower-body	Bilateral
Tremor	Rest tremor	Often absent	Variable	Rare	Action tremor
Bradykinesia	Present with decrement	Present + atypical signs	Present	Present	Absent
Gait	Late instability	Early falls	Variable	Early gait disorder	Usually normal
Levodopa response	Good	Poor	Variable	Poor	None

Table 1. Key Clinical Features in the Differential Diagnosis of Parkinsonism; *courtesy of Dilpriya K. Mangat, MD, FRCPC, MBA.*

Canada.[10] As such, clinical judgment remains the cornerstone of diagnosis in most settings.

Non-Motor Symptoms

Non-motor symptoms are a hallmark of PD and contribute significantly to patient morbidity. These symptoms often precede motor manifestations and may include sleep disturbances, mood disorders, cognitive impairment, and autonomic dysfunction.[5]

Primary care clinicians should routinely screen for these non-motor symptoms. A cognitive assessment can be performed using the Montreal Cognitive Assessment (MoCA), which has demonstrated utility in detecting early cognitive impairment in PD.[11] The Non-Motor Symptoms Questionnaire (NMSQuest) provides a practical tool for identifying a broad range of non-motor symptoms,[12] while the SCOPA-AUT questionnaire can help assess autonomic dysfunction.[13]

Recognizing and managing non-motor symptoms is essential, as these features often have a greater impact on quality of life than motor symptoms alone.

Initial Management in Primary Care

Education and Counselling

Effective management begins with patient education. Patients should understand that PD is a chronic, progressive condition with variable progression. Counselling should address medication adherence, fall prevention, driving safety, and the importance of maintaining physical activity.

A critical safety point is that dopaminergic medications should not be stopped abruptly, as this can lead to severe clinical deterioration and potentially life-threatening complications.[4]

Exercise and Rehabilitation

Exercise is a cornerstone of PD management and should be initiated early. Evidence supports improvements in motor function, balance, and overall disease severity with regular physical activity.[4]

Referral to physiotherapy, occupational therapy, and speech-language pathology should occur early in the disease course. These interventions help maintain function, reduce fall risk, and support communication and swallowing difficulties.

Pharmacotherapy

Levodopa remains the most effective treatment for motor symptoms and should not be delayed once symptoms interfere with daily function.[4] Evidence from the PD MED trial demonstrated that levodopa provides superior long-term quality-of-life outcomes compared to levodopa-sparing strategies.[14]

Treatment is typically initiated at a low dose and titrated gradually based on clinical response. Common side effects include nausea, orthostatic hypotension, hallucinations, and dyskinesias. These risks are particularly relevant in older adults and require careful monitoring.

Dopamine agonists may be considered in selected patients but are associated with higher rates of adverse effects, including hallucinations, sleep disturbances, and impulse control disorders. Their use should be approached with caution, especially in patients over 70 years of age.[4] See **Table 2** for a summary of pharmacologic management options for Parkinson's disease in primary care.

Motor Fluctuations and Medication-Related Complications

As PD progresses and dopaminergic therapy continues, many patients develop motor fluctuations and treatment-related complications. These are important for primary care clinicians to recognize, as they directly impact function, safety, and quality of life.[4,15]

Dyskinesias are involuntary, non-purposeful movements that may range from mild (e.g., subtle ankle or finger movements) to severe generalized writhing. They are most commonly associated with peak levodopa levels and are therefore often referred to as "peak-dose dyskinesias." While not always distressing to patients, dyskinesias may interfere with function or increase fall risk in more advanced disease.[4]

Motor fluctuations are commonly described using "on-off" terminology. During "on" periods, patients experience adequate symptom control and improved mobility. During "off" periods, motor symptoms re-emerge, often with reduced mobility and functional impairment. A common pattern is "wearing off," where symptoms return before the next scheduled medication dose, reflecting a shortening duration of levodopa's effect over time.[14,15]

Medication/Class	Mechanism	Role in Care	Starting Dose	Key Adverse Effects	Clinical Notes
Levodopa/Carbidopa (Sinemet®) **or Levodopa/ Benserazide** (Prolopa®)	Converted to dopamine in the brain	First-line for motor symptoms	½ tablet (100/25) TID	Dyskinesia, nausea, orthostasis, hallucinations	Most effective therapy; do not delay when function impacted; protein may reduce absorption
Dopamine Agonists (Pramipexole, Ropinirole, Rotigotine)	Mimics dopamine activity	Alternative or adjunct	Pramipexole 0.125 mg TID	Sleep attacks, hallucinations, edema, impulse control disorders	Use cautiously in older adults (>70); monitor behaviour changes
Amantadine	Enhances dopamine release, NMDA antagonist	Early symptoms or dyskinesia management	100 mg daily	Confusion, hallucinations, edema, livedo reticularis	Useful for dyskinesia reduction; caution in cognitive impairment
COMT Inhibitors (Entacapone)	Inhibits peripheral levodopa breakdown	Adjunct for wearing-off	Given with levodopa doses	Dyskinesia, diarrhea	Extends levodopa effect; used when response shortens
Levodopa/Carbidopa/ Entacapone (Stalevo®)	Combined levodopa + COMT inhibition	Wearing-off management	Equivalent levodopa dose	Dyskinesia	Simplifies regimen in fluctuating patients
Levodopa Intestinal Gel (Duodopa®)	Continuous intestinal levodopa delivery	Advanced PD	Specialist initiated	Device complications, dyskinesia	Not primary care initiated
MAO-B Inhibitors (Rasagiline, Selegiline)	Reduces dopamine breakdown	Mild symptoms or adjunct	Rasagiline 0.5–1 mg daily	Dyskinesia, insomnia	Watch drug interactions (SSRIs, sympathomimetics)
Anticholinergics (Trihexyphenidyl)	Reduces cholinergic activity	Tremor-predominant PD (younger patients)	Low dose titration	Confusion, delirium, dry mouth	Avoid in older adults
Domperidone *(supportive)*	Peripheral dopamine blockade	Treat levodopa-induced nausea	10 mg TID PRN	QT prolongation	Use cautiously; avoid high doses

Table 2. Pharmacologic Management of Parkinson's Disease in Primary Care; *courtesy of Dilpriya Mangat, MD, FRCPC, MBA*.

Abbreviations: COMT: catechol-O-methyltransferase; **NMDA:** N-methyl-D-aspartate; **PD:** Parkinson's disease; **PRN:** as needed; **SSRIs:** selective serotonin reuptake inhibitors; **TID:** three times a day

Recognition of these patterns in primary care is important, as they often signal the need for medication adjustment or specialist input.[4]

Referral and Multidisciplinary Care

Referral to a neurologist or movement disorders specialist is indicated when there is diagnostic uncertainty, the presence of red flags, or a suboptimal response to therapy. Early referral is particularly important in cases of atypical presentation or rapid disease progression.[7]

Primary care clinicians should also facilitate access to multidisciplinary care. Allied health professionals play a critical role in maintaining function and quality of life. Community resources, including Parkinson Canada, provide valuable support for patients and caregivers.

Conclusion

PD is a common and complex condition that requires a thoughtful and structured approach in primary care. Early recognition of Parkinsonism, careful differentiation from alternative diagnoses, and timely initiation of treatment can significantly improve patient outcomes. Primary care clinicians play a central role in managing both motor and non-motor symptoms, coordinating care, and supporting patients and families throughout the disease trajectory.

Correspondence

Dilpriya K. Mangat, MD, FRCPC, MBA
Email: dilpriya.mangat@nexcerebrum.com

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