A GUIDE TO

deprescribing

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CPS Experts in Medicines
CONSULTANT PHARMACY SERVICES

ANTIHYPERTENSIVES

KEY POINTS

Lowering blood pressure reduces risk of a range of long-term consequences, this benefit is still evident in older patients.

Less aggressive control of blood pressure in older people gives results equivalent to those achieved with more aggressive control.

Low blood pressure may be associated with increased morbidity and mortality in the older person.

Patients being treated for hypertension are more likely to fall if they have proven orthostatic hypotension.

Adverse effects of many antihypertensive agents are likely to be more common in the older person.

Guidelines generally recommend vasodilators (ACE inhibitors, angiotensin receptor blockers, calcium-channel blockers) as first line therapy.

Withdrawal of antihypertensives should be gradual.

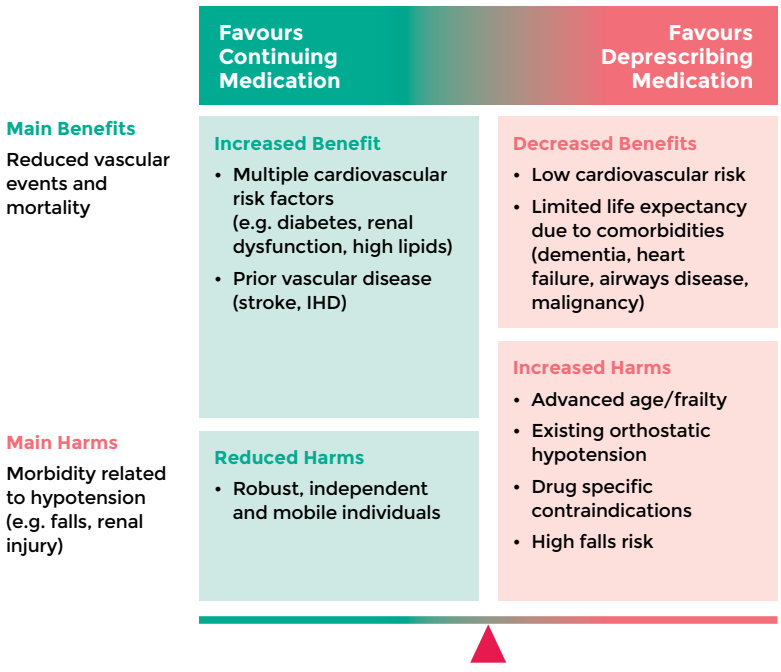
CONTEXT

This guide considers the use of antihypertensive agents in older adults.

RECOMMENDED DEPRESCRIBING STRATEGY

- Many patients are receiving multiple agents that lower blood pressure. Reduction and cessation strategies should focus on one agent at a time.
- Reduction or cessation of antihypertensive agents should be considered:
  - In frail elderly and/or immobile patients
  - In patients with a high falls risk
  - In patients with confirmed orthostatic hypotension (>20mmHg fall in systolic on standing, and/or >10mmHg fall in diastolic on standing)
  - In patients with limited life expectancy e.g. terminal phase

BENEFIT VERSUS HARM



BACKGROUND

Multiple studies have shown increased morbidity and mortality in patients with hypertension, and correspondingly a reduction in morbidity and mortality with appropriate treatment of hypertension. With increasing age, however, the relative benefit of lowering blood pressure is attenuated. In 2002, Lewington et al published data from over 1 million adults from 61 studies on the associations between Systolic Blood Pressure (SBP) and Diastolic Blood Pressure (DBP) and mortality from stroke and coronary heart disease by age. The associations between both SBP and DBP and mortality from stroke, coronary heart disease and other vascular disease were graded and continuous with the lowest risk at SBP of 115 mmHg and DBP of 75 mmHg (lower BP levels were not reported) and the highest risk at SBP of 175 mmHg and DBP of 105 mmHg (higher levels were not reported). However, these associations were weaker in older age (see **Figure 1**).[1]

It should be noted however, that the absolute risks of cardiovascular disease are greater in an older population and that the lower relative risk reduction with treatment may still translate into a higher absolute risk reduction

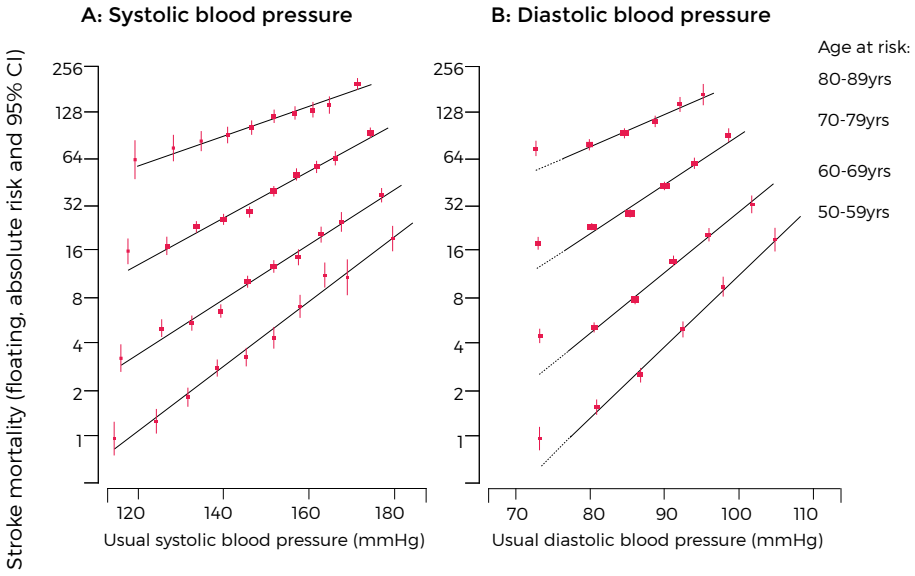


Figure 1: Stroke mortality in each decade of age versus usual blood pressure[1]

EFFICACY

Trials of hypertension management in older people are limited and were reviewed by Fleg et al.[2] in 2011 and Muntner et al.[3] in 2014. Of 12 studies reviewed by Fleg, five showed statistically significant reductions in cardiovascular events. All five studies showed a relative reduction of stroke risk of between 23 and 57%, where starting BP was between 169 and 185mmHg systolic.

Muntner's review included three different studies that compared intensive vs more lenient systolic blood pressure control in older patients:

- A Japanese study of 4418 patients aged 65-84 years compared tight vs lenient control of blood pressure on outcomes. One group achieved 136/75 mmHg on average while the other 146/78 mmHg. Over 2 years of follow-up, there were no differences in the primary composite outcome of cardiovascular disease or renal failure.[4]
- The Valsartan in the Elderly with Isolated Systolic Hypertension (VALISH) study found no difference in a population of patients aged 70-84 years that achieved SBPs of 137mmHg vs 142mmHg in terms of stroke, sudden death or myocardial infarction frequency.[5]
- An Italian study of 1111 patients with a mean age of 67 years randomised patients to tight (<130mmHg) vs moderate (<140mmHg) control of SBP. They showed the composite endpoint of CVD/renal disease after 2 years occurred in 9.4% of patients in the moderate control group compared to 4.8% in the tight control group (ARR 4.6%, Annualised **NNT=44**).[6]

One additional study (HYVET) looked specifically at patients over 80 years of age.[7] This involved randomising patients with a starting SBP of 160mmHg or more to indapamide or a placebo. Perindopril was added to the indapamide if a SBP of 150mmHg was not achieved. They reported positive outcomes for the following endpoints with active treatment compared to placebo after an average 1.8 year follow-up:

- Death from stroke – ARR 0.8%, Annualised **NNT= 225**
- Death from any cause - ARR 2.2%, Annualised **NNT= 81**
- Development of Heart Failure - ARR 1.83%, Annualised **NNT= 97**
- Any cardiovascular event – ARR 2.95%, Annualised **NNT= 61**

THE SPRINT STUDY

In 2015 , the SPRINT research group published the results of a randomised trial comparing intensive to standard blood pressure control.[8] They randomly assigned 9361 non-diabetic people with an SBP of 130mmHg or higher to intensive control (target SBP<120mmHg) or standard treatment (target SBP<140mmHg). The primary outcome was a composite of MI, ACS, stroke, acute CCF or death from cardiovascular causes and occurred overall in 5.2% (243/4678) of the intensively treated patients and 6.8% (319/4683) of the standard treatment patients over 3.26 years (ARR over 1 year= 1.6%, NNT=63).

Approximately 28% of the patients were 75 years old or more (mean age 79.8). Of these 1317 received intensive and 1319 received standard treatment.

The reduction in the composite outcome with intensive treatment remained evident in those aged 75 years and over, with an event rate of 10.9% (144/1319) in the standard treatment arm and 7.7% (101/1317) of the intensive treatment arm over the median follow-up of 3.26 years (ARR=3.2%, NNT= 31). To achieve lower targets in SPRINT a higher number of medications was required; 54.2% of participants in the intensive treatment group required 3 or more antihypertensives compared to 26.5% in the usual care group.[8]

The over 75 year old age group in the SPRINT study were more closely examined in a separate paper.[9] A summary of the overall findings from the SPRINT overall group and the over 75 SPRINT group is shown in **Table 1**.

IMPACT OF FRAILTY

It should be noted that these studies all include relatively fit older patients and that frail older patients may be more sensitive to the impact of antihypertensive treatment and may or may not obtain the same benefit from antihypertensive therapy. Frailty is significantly related to orthostatic intolerance and postural blood pressure drops.[10] A recent large prospective observational analysis by Masoli et al. supports higher blood pressure targets in frail and older adults.[11] Data from 415,980 people >75 years found that SBPs above the 130-139 mmHg reference range were associated with lower mortality risk in those with moderate/severe frailty (Electronic frailty index) and for all adults >85 years. There was increased mortality regardless of frailty level when SBP was <130 mmHg.

Across the population (74-84 years old and >85 years) the risk of stroke, MI, and heart failure was raised with SBP >150 mmHg, however, for people >85 years, systolic blood pressures up to 180mmHg were not associated with an increase in mortality. In these older adults, those that were frail had better mortality outcomes with higher systolic blood pressure (see **Figure 2**). Mean baseline blood pressure was lower as frailty increased, which has been seen in other studies.[12]

	ALL SPRINT SUBJECTS	OVER 75yo SPRINT SUBJECTS
Over 3.26 years	For every 1000 subjects treated to <120mmHg systolic:	For every 1000 patients over 75yo treated to <120mmHg systolic:
	16 less primary events as defined by the authors (**NNT= 62**) › (or 12 less events if combined MI, Stroke or CV Death is used) (**NNT= 83**)	32 less primary events as defined by the authors (**NNT=31**) › (or 26 less events if combined MI, Stroke or CV Death is used) (**NNT= 38**)
	12 less deaths (all causes) (**NNT= 83**)	26 less deaths (all causes) (**NNT= 38**)
	6 less cardiovascular deaths (**NNT= 167**)	8 less cardiovascular deaths (**NNT= 125**)
	53 serious adverse events would occur (**NNH= 19**) › 14 severe hypotensive episodes (**NNH= 71**), › 11 more syncopal episodes (**NNH= 91**), › 10 more electrolyte disorders (**NNH= 10**) and › 18 episodes of renal damage (**NNH= 56**)	92 serious adverse events would occur (**NNH= 11**) › 23 severe hypotensive episodes (**NNH= 43**), › 16 more syncopal episodes (**NNH= 62**), › 25 more electrolyte disorders (**NNH= 40**) and › 28 episodes of renal damage (**NNH= 36**)
Over 1 year	For every 1000 subjects treated to <120mmHg systolic:	For every 1000 patients over 75yo treated to <120mmHg systolic:
	5 less primary events as defined by the authors (**NNT= 200**) › (or 3.7 less events if combined MI, Stroke or CV Death is used) (**NNT= 270**)	10 less primary events as defined by the authors (**NNT=100**) › (or 8 less events if combined MI, Stroke or CV Death is used) (**NNT= 125**)
	3.7 less deaths (all causes) (**NNT= 270**)	8 less deaths (all causes) (**NNT= 125**)
	1.8 less cardiovascular deaths (**NNT= 555**)	2.5 less cardiovascular deaths (**NNT= 400**)
	16 serious adverse events would occur (**NNH= 62**) › 4.3 severe hypotensive episodes (**NNH= 232**), › 3.4 more syncopal episodes (**NNH= 294**), › 3 more electrolyte disorders (**NNH= 333**) and › 5.5 episodes of renal damage (**NNH= 182**)	28 serious adverse events would occur (**NNH= 36**) › 7 severe hypotensive episodes (**NNH= 143**), › 5 more syncopal episodes (**NNH= 200**), › 7.7 more electrolyte disorders (**NNH= 130**) and › 8.6 episodes of renal damage (**NNH= 116**)

Table 1: Summary of SPRINT findings for all subjects combined and those over 75 separately.[8,9]

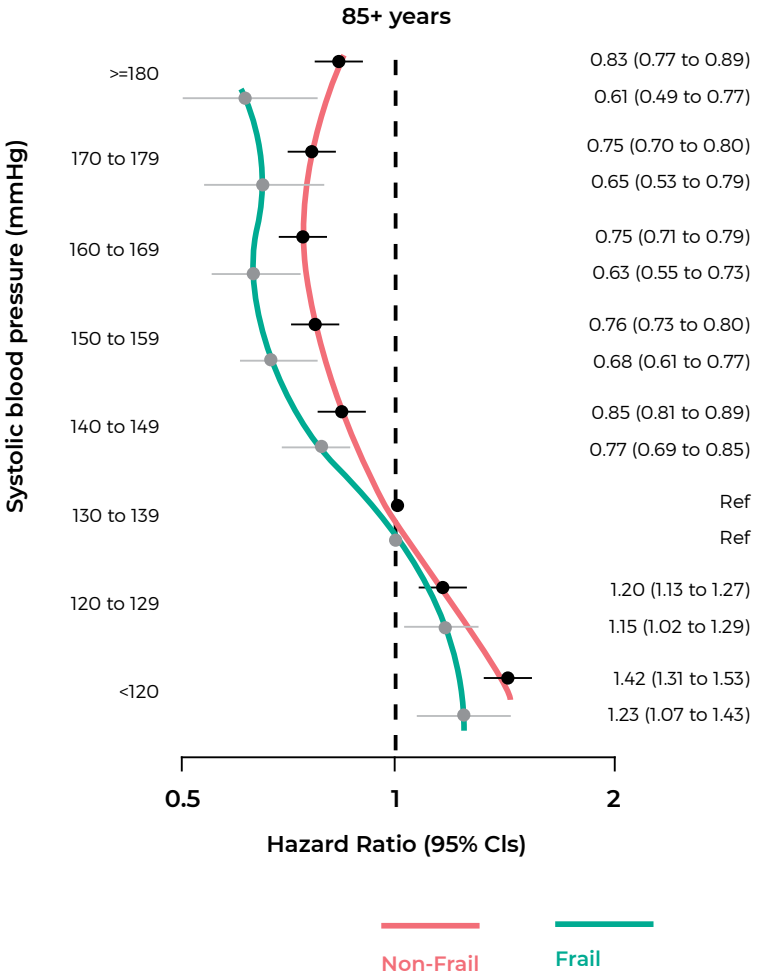


Figure 2: Relationship between all-cause mortality and systolic blood pressure in frail and non-frail adults aged greater than 85 years.[11]

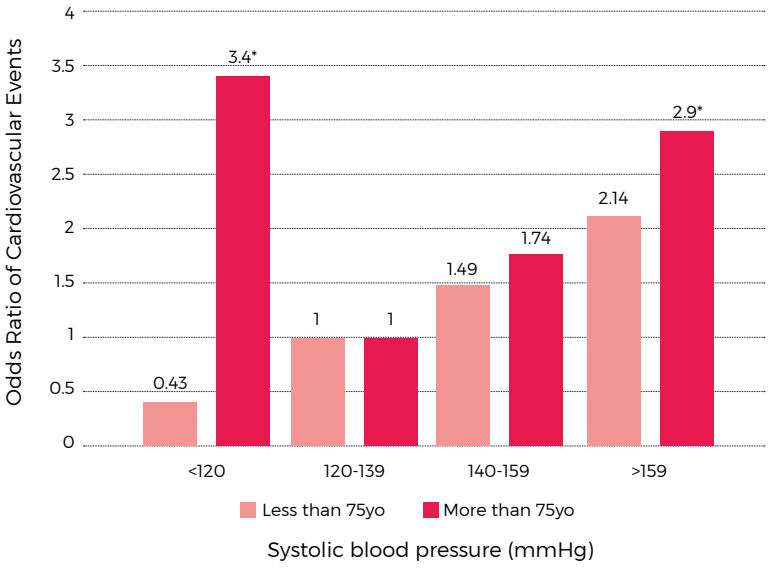


Figure 3: Relation between achieved blood pressure and cardiovascular events (* = statistically significant) [14]

ADVERSE EFFECTS

SUSTAINED HYPOTENSION

Some studies have reported an increased cardiovascular risk at very low systolic or diastolic blood pressures in the elderly.[13,14,15]

Ogihara et al undertook a 3 year follow-up 2164 patients over 60 who regularly attended their clinician and documented cardiovascular morbidity and mortality as well as achieved blood pressure.[14] In the subgroup of patients aged 75 years or more, patients with an achieved systolic BP of <120mmHg had a significantly higher incidence of total cardiovascular events, as did patients with a systolic BP of >160mmHg (see **Figure 3**).

Voko et al. reported a J shaped relationship between incidence of stroke and diastolic (but not systolic) blood pressure in treated hypertensives.[13] In patients receiving treatment for hypertension, diastolic blood pressure of less than 65mmHg was associated with the same stroke risk as patients with a diastolic of >84mmHg, and significantly higher than those with a diastolic of 65-74mmHg.

Taken together, the above studies indicate that low blood pressure may be associated with increased morbidity and mortality in the elderly. It remains unclear whether the low blood pressure is itself an indicator of poor cardiovascular health e.g. comorbid heart failure, which may be responsible for this observation.

ORTHOSTATIC HYPOTENSION

A further potential limiting factor in the treatment of elderly patients is the presence of, or exacerbation by treatment of, orthostatic hypotension. The majority of people with orthostatic hypotension are asymptomatic and may not be routinely identified.

Of note, orthostatic and/or postprandial hypotension is found in up to 20% of elderly patients with isolated systolic hypertension.[16,17,18] Hypertensive older patients with orthostatic hypotension (particularly those with less well controlled hypertension) are more likely to fall than patients without.[19] Indeed, antihypertensive treatment has been associated with a 43% increased risk of hip fractures in the elderly in the first 45 days of treatment.[20] Measurement of sitting and standing blood pressures are essential prior to commencing or modifying antihypertensive therapy. Sitting and standing blood pressure monitoring is also recommended in all symptomatic adults, those >80 years and those with diabetes.[21]

OTHER ADVERSE EFFECTS OF ANTIHYPERTENSIVE AGENTS

There are a wide range of differing antihypertensive agents available with multiple mechanisms of action. As a result, there are a wide range of possible adverse effects beyond hypotension, that may occur with these agents, either alone or as a result of their combination with other agents taken by the patient. Metabolic, cardiac and renal effects are seen with many antihypertensives, with some agents also exhibiting more specific adverse effects. For the vast majority of adverse effects, the elderly and those with limited reserve are more likely to sustain adverse effects. A summary of the complexity in choosing between drug classes for treatment of hypertension has been undertaken.[22] In **Table 3**, the main adverse effects with each class of agents are listed.

DRUG CLASS (COMMON EXAMPLES)	ADVERSE EFFECTS
Thiazide and Loop diuretics (hydrochlorothiazide, indapamide, chlorthalidone, frusemide)	Hypokalaemia, hyponatraenia, hypomagnesaemia
	Volume-depletion
	Renal impairment, hyperuricaemia, gout, lipid alterations, hyperglycaemia, insulin resistance
	NSAIDs reduce thiazide potency
	Erectile dysfunction and possibly impotence
	Reduction of lithium excretion and precipitate lithium toxicity
	Potential to increase fatigue and lethargy
	Pro-diabetogenic potential in combination with Beta Blockers
	Increase of urinary frequency, leg cramps
	Decrease of renal blood flow, creatinine clearance, Glomerular Filtration Rate
Potassium Sparing Diuretics (spironolactone, amiloride)	Hyperkalaemia
Beta Blockers (atenolol, metoprolol)	Sinus bradycardia, fatigue, AV-nodal heart block bronchospasm, aggravation of acute heart failure
	Intermittent claudication, confusion, hyperglycaemia
	Diabetes mellitus
	Drowsiness, lethargy, sleep disturbance, visual hallucinations, depression, blurring of vision, nightmares
	Pulmonary side-effects (increased airway resistance in asthmatics)
	Peripheral vascular side-effects (cold extremities, Raynaud's phenomenon)
	Erectile dysfunction
Angiotensin Converting Enzyme Inhibitors (eg. perindopril, ramipril)	Cough, hyperkalaemia
	Angioneurotic oedema
	Rash, altered taste sensation, renal impairment
Angiotensin Receptor Blockers (eg. candesartan, irbesartan)	Hyperkalaemia, renal impairment
Calcium Channel Blockers Non-dihydropyridines (eg. verapamil, diltiazem)	Rash, sinus bradycardia, heart block, heart failure, constipation (verapamil), gingival hyperplasia
	Ankle oedema, headache
Calcium Channel Blockers Dihydropyridines (eg. amlodipine, nifedipine)	Peripheral oedema, tachycardia
	Aggravation of angina pectoris (short-acting agents)
Direct vasodilators (eg. hydralazine)	Tachycardia, fluid retention
	Angina pectoris
Alpha 1 adrenergic blockers (eg. prazosin)	First-dose hypotension, peripheral oedema, worsening of stress incontinence in women
Alpha-beta adrenergic blockers (eg. carvedilol, labetalol)	Heart block, sinus bradycardia, bronchospasm
Central acting agents (eg. moxonidine, methyldopa)	Sedation, constipation, dry mouth

Table 3: Most common drug-related side effects of antihypertensive classes[22]

DISCONTINUING ANTIHYPERTENSIVES

Recent small trials have looked at the effects of withdrawing antihypertensives. In general, resultant increases in blood pressure have only been modest though there has not been long-term data on clinical outcomes.

A Cochrane Review in 2020 assessed six trials (1073 participants) of withdrawing antihypertensives in people aged 50 years and older where the indication was hypertension and/or primary prevention of cardiovascular disease. Duration and follow-up ranged from 3 to 12 months. No significant effect was found in terms of an increase of the primary endpoints of all-cause mortality (OR 2.08, 95% CI 0.79 to 5.46; low certainty of evidence) or myocardial infarction (OR 1.86, 95% CI 0.19 to 17.98; very low certainty of evidence) when comparing discontinuing and continuing antihypertensives. This was despite a mean SBP increase of 9.75 mmHg (95% CI 7.33 to 12.18).[23]

The OPTIMISE trial looked at removal of one antihypertensive in participants 80 years or older, with SBP <150 mmHg (mean baseline 130 mmHg) and treated with two or more antihypertensives. Compared to the usual care group, a similar majority of participants achieved the primary endpoint, maintaining a SBP <150 mmHg at the 12-week follow-up (87.7% vs 86.4%, RR 0.98, 95% CI 0.92–1.05). Following medication reduction, the increase in SBP was on average 3.4 mmHg (955 CI, 1.1 to 5.8 mmHg). There was no significant difference in serious adverse events or adverse effects, though a limitation on the study was the short duration. Two thirds of the intervention group did not require any regimen alteration following cessation of their antihypertensive, suggesting successful withdrawal may be achievable for many patients.[24]

Of interest, a study of 765 nursing home residents in Norway found that as expected, SBP increased with deprescribing of antihypertensives (average increase of 14mmHg from mean baseline of 128/71mmHg) at four months, however at nine months blood pressures had re-settled at baseline level (mean 134mmHg).[25]

Choice of antihypertensive

A post-hoc analysis of the OPTIMISE trial may help to support decisions on particular medications to target for dose reduction or discontinuation.[26] This found that prescriber interventions, alongside recommendations to follow NICE 2019 Guidelines, resulted predominantly in reduction of high doses of thiazides and calcium-channel blockers and cessation of low doses of beta-blockers (heart failure patients were excluded). Thiazide diuretics as a class provide similar antihypertensive effect across a dose-range (i.e. higher doses do not provide significant increases in blood pressure reduction).[27]

Withdrawal of calcium channel blockers (CCB) at 12 weeks was associated with an increase in SBP (5 mmHg, 95%CI 0–10 mmHg) and reduced SBP control (adjusted RR 0.89, 95%CI 0.80–0.998) compared to usual care. In contrast, withdrawal of beta-blockers (BB) was associated with no change in SBP (–4 mmHg, 95%CI –10 to 2 mmHg) and no difference in SBP control (adjusted RR 1.15, 95%CI 0.96–1.37). The implication is that to maintain SBP control, cessation of BB is preferable to CCB, in populations where there is no compelling indication to continue a BB for other reasons.

Thiazides and beta-blockers are common targets for withdrawal, though in the absence of clear guideline recommendations, this remains up to the clinical judgement of individual prescribers.[28] Considering comorbidities can help guide these decisions, as summarised in **Table 2**.

	ACE Inhibitor or ARB	DHP Calcium Channel Blockers (e.g. amlodipine)	Non-DHP Calcium Channel Blocker (e.g. diltiazem)	Diuretics (Thiazides, Loop)	Beta-blocker	Alpha-blocker
Angina	+	+ / -	+		+	
Myocarial infarction	+		-		+	
Peripheral Arterial Disease					-	
Aortic Stenosis	-	-	-			-
Atrial Fibrillation	+		+		+	
Bradycardia/AV block			-		-	-
Heart Failure	+		-	+	+	
Diabetes	+			-	-	
Chronic kidney disease	+			-		-
Bilateral renal artery stenosis	-	-	-			-
Benign prostatic hyperplasia						+
Urinary incontinence		-	-	-		
Constipation			-			
Gout				-		
Asthma						

\+ = potentially beneficial
\- = potentially harmful

ACE = angiotensin converting enzyme
ARB = angiotensin || receptor blocker
DHP = dihydropyridine

Table 2. Comorbidities that can influence choice of antihypertensive to deprescribe. Adapted from Parekh et al., 2017[29] and Nelson M, 2010.[30]

FACTORS TO CONSIDER

IN FAVOUR OF DEPRESCRIBING

- Lifestyle modification can achieve significant benefit. In patients where lifestyle modification (exercise, salt and sugar restriction, alcohol, weight loss) are possible, these changes can support the reduction and/or cessation of antihypertensive agents. Similarly, this includes minimising medication that can increase blood pressure.
- The benefits of treating hypertension in the >85 age group are unclear; ongoing treatment should be reassessed in light of prognosis, frailty, comorbidities, and quality of life.
- Patients who are frail and have a high risk of falls are more likely to fall as a result of antihypertensive treatment and may not derive the same benefit of treatment as non-frail elderly. Reduction or cessation of antihypertensives should be considered in these patients.
- Predictive factors for successful deprescribing include low/normotensive levels while using antihypertensives, and the use of a sole antihypertensive for therapy.
- Use of agents with less/no evidence of impact on CV clinical outcomes e.g. moxonidine

AGAINST DEPRESCRIBING

- Agents with an antihypertensive effect may have other benefits in patients with other comorbidities and they may be prescribed more specifically for these other purposes. Beta blockers for heart failure, atrial fibrillation or ischaemic heart disease, ACE inhibitors for heart failure or renal protection and prazosin for prostatic symptoms are examples of where cessation of these agents may worsen the underlying condition.
- Following recent vascular events where good control of hypertension may be beneficial and outweigh potential risks and adverse effects.

DISCONTINUATION SYNDROMES

Withdrawal effects may be wide ranging, depending on the specific class of agent and any other conditions being treated. These may include peripheral oedema, tachycardia, rebound hypertension or worsening heart failure or ischaemic heart disease.

As a result, it is recommended that most antihypertensives should be tapered as part of any discontinuation plan

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DECEMBER 2022