BMC Nephrology

REVIEW

Open Access

Management of blood pressure in adults, children and young people on dialysis: UK kidney association clinical practice guideline

Tim Doulton[1], Mariyam Adam[2], Katie Durman[3], Stanley L. S. Fan[4], Anna Forbes[5,6], Ed Jenkinson[7], Charlotte Mallindine[8], Kieran McCafferty[3,4], Manish D. Sinha[9,10], Douglas J. Stewart[11], Pauline Swift[12] and Indranil Dasgupta[13,14,15*]

Abstract

People with end stage kidney disease receiving dialysis have a very risk of cardiovascular disease and mortality. Hypertension is a modifiable risk factor for cardiovascular disease and mortality, affecting around 90% of dialysis patients. Conversely, intradialytic hypotension in haemodialysis patients is associated with debilitating symptoms as well as the risk of cardiovascular disease and mortality. Blood pressure management in dialysis patients involves accurate measurement of blood pressure, setting a target blood pressure, adequate fluid volume management, lifestyle changes, modification of dialysis and dialysate, and the use of medication with proven efficacy in these patients. However, globally there is no published contemporary clinical practice guideline to assist health care professionals to manage blood pressure in dialysis patients. This is mainly because of a dearth of good quality evidence to inform guideline recommendations. Here, we present a comprehensive guideline to manage blood pressure in dialysis patients based on a thorough systematic review of literature. The guideline development committee comprised a multidisciplinary group of nephrology healthcare professionals and a patient. The evidence underpinning the guidance is often of moderate to low quality, influencing the strength of the recommendations. Therefore, the committee have come up with a list of research recommendations with a view to informing future guidelines. It is envisaged that this guideline will improve the care and outcomes of dialysis patients in the UK and elsewhere.

Clinical trial number

Not applicable.

Keywords Blood pressure, Haemodialysis, Peritoneal dialysis, Guideline, Lifestyle, Evidence, Dialysate, Dry weight, Medication, Children and youn people

*Correspondence:
Indranil Dasgupta
indranil.dasgupta@uhb.nhs.uk

Full list of author information is available at the end of the article

Background

The UK adult population receiving dialysis continues to increase. Between 2020 and 2021, although the annual increase was 1.5%, and slightly lower compared with 2-2.5% seen annually prior to the COVID-19 pandemic. The average age of adults receiving dialysis is older (66.1 years for HD and 63.3 years for PD) with a history of longer renal replacement therapy (median duration 3.2 years) when compared with published registry data from 2010 [1, 2].

Cardiovascular (CV) disease remains one of the most significant causes of mortality in adults receiving dialysis, with an incidence of over 20%, and death from a primary CV disease cause is more likely in those less than 65 years of age [2]. In children and young people (CYP) with end stage kidney disease (ESKD), similar trends are seen, with the current childhood dialysis population in the UK being the largest on record. CYP are waiting longer than before to receive a kidney transplant and CV disease remains the most common cause of morbidity and mortality for those receiving dialysis [2, 3].

Hypertension is one of the commonest modifiable causes of CV disease in adults receiving dialysis [4–7]. In CYP on maintenance dialysis too, hypertension is the strongest risk factor for left ventricular hypertrophy (LVH), the most evaluated surrogate marker of CV abnormality in this population [8]. Data from adults on long term dialysis additionally highlight increased CV risk from lower blood pressure (BP) and declining BP over time [4, 5].

This guideline has been developed with a focus on BP management, acknowledging the central role of body fluid status in adults and CYP receiving dialysis [6, 7]. Existing guidance does not focus on the systematic evaluation of the evidence base for BP management in dialysis-dependent adults and CYP and, as such, highlights the need for specific guidelines [1, 6].

The authors of this report include a broad range of healthcare professionals with experience in kidney disease, including dietitians, pharmacists and both adult and paediatric kidney doctors, as well as a patient representative, who have worked together to review the evidence for management of BP in adults and CYP who are receiving dialysis for ESKD.

The main themes in this document include evaluation of the published evidence for: (i) measurement of BP; (ii) BP targets; (iii) lifestyle modifications; (iv) dialysis prescription modifications; (v) antihypertensive management to aid with BP control; (vi) dry weight optimisation in adults, and (vii) for all these issues in CYP receiving dialysis. Our overall aim with this guidance is to ensure a consistent and standardised approach to the management of BP in patients receiving dialysis across the UK, whilst striving to improve quality of care and reduce disparities in outcomes.

Methodology

Literature sources and search terms

The review process for this guideline was in accordance with the PRISMA statement [9]. Several databases were searched (including PubMed, EMBASE, Ovid MEDLINE, Cochrane and CINAHL) to obtain articles that met eligibility for the literature review. Articles included were those with a publication date from 1st January 2000 to 31st December 2022 published in the English language. Full details of the PICO search tool, with all included databases and search strategies, are available in Appendix B.

Inclusion and exclusion criteria

Detailed inclusion criteria, according to the PICO search tool, are available in Appendix B. Exclusion criteria were studies relating to: (i) acute haemodialysis, (ii) acute peritoneal dialysis, (iii) continuous veno-venous haemofiltration and other acute kidney replacement modalities, and (iv) non-systemic hypertension in any specialised vascular bed (e.g. pulmonary, intracranial).

Study selection

All articles identified from the literature search were allocated to a predefined topic group by lead authors AF and TD. The seven topic groups were developed along the main themes highlighted previously. Within each topic group, articles were screened by at least two authors. Any discrepancies in whether an article met inclusion criteria were dealt with by mutual agreement between the authors allocated to that topic group, and TD or ID if consensus could not be met. Authors for each topic group are listed in Appendix B.

Data extraction and quality appraisal

For articles where there was a consensus opinion on inclusion, data extracted were: study aim, study design, method of BP assessment, follow-up period, sample size, population (country and kidney replacement therapy modality), primary analysis, and major results. These data are summarised in the Evidence Tables (Appendix C) and findings were used to support the rationale for the recommendations of this guideline. The recommendations and supporting rationale were reviewed by all authors and by key stakeholders prior to publication of the guidelines.

Evidence grading

We followed the principles set out in the UK Kidney Association's "Clinical Practice Guideline Development Manual" and grade evidence according to a

two-tier grading system (see Table 1). We use the term "recommend" within the guideline text where Recommendations are based on Grade 1 evidence, and the term "suggest" for those based on Grade 2 evidence. We also made ungraded 'Research recommendations', which help define ongoing areas of clinical uncertainty, and we offer 'Audit measures', to define how to demonstrate effective implementation of recommendations.

Summary of clinical practice guidelines

Measurement of blood pressure

1. We recommend interdialytic ambulatory blood pressure monitoring (ABPM) as the gold standard to diagnose hypertension in people on haemodialysis. (1C)
2. We suggest using either home blood pressure measurement (HBPM) or standardised out-of-dialysis unit clinic BP measurement to monitor BP and guide treatment for in-centre haemodialysis. (2C)
3. We suggest clinicians use routine dialysis unit BP measurements to inform safety of delivering haemodialysis rather than to inform hypertension management decisions, because of the imprecision of routine dialysis unit BP measurement compared to standardised out-of-dialysis-unit BP measurement and interdialytic ABPM. (2C)
4. We suggest using either HBPM or standardised clinic BP measurements to monitor BP and guide treatment in patients receiving home-based dialysis (home haemodialysis and peritoneal dialysis). (2C)

Table 1 UK kidney association's grading system for recommendations' strength and evidence quality

Level of evidence	Evidence quality
• Grade 1 recommendation is a strong recommendation to do (or not do) something, where the benefits clearly outweigh the risks (or vice versa) for most, if not all, patients (i.e. "recommendations"). • Grade 2 recommendation is a weaker recommendation, where the risks and benefits are more closely balanced or are more uncertain (i.e. "suggestions").	• Grade A evidence means high-quality evidence that comes from consistent results from well-performed randomised controlled trials, or overwhelming evidence of some other sort. • Grade B evidence means moderate-quality evidence from randomised trials that suffer from serious flaws in conduct, inconsistency, indirectness, imprecise estimates, reporting bias, or some combination of these limitations, or from other study designs with special strength. • Grade C evidence means low-quality evidence from observational studies, or from controlled trials with several very serious limitations. • Grade D evidence is based only on case studies or expert opinion.

Blood pressure targets

5. We suggest, for those on haemodialysis where *non-standardised* in-centre BP measurements are used, aiming for:
   a. pre-dialysis systolic BP between 140 and 165 mmHg (2B) and pre-dialysis diastolic BP between 60 and 100 mmHg. (2C)
   b. post-dialysis systolic BP between 120 and 140 mmHg and post-dialysis diastolic BP of ≥ 70 mmHg (2C).
6. We suggest aiming for the lower end of the systolic BP ranges in recommendation 5a, unless this results in an increased frequency of intradialytic hypotension (IDH) and/or in those with a prior history of frequent IDH (2D).
7. We suggest that factors such as age and comorbidities may be used to determine an individual patient's target BP range. For younger people or those with fewer co-morbidities, a *lower* systolic BP range than suggested in recommendation 5a can be considered. (2C)
8. We suggest that clinic BP should be < 140/90 in people on peritoneal dialysis. (2C)

Lifestyle modification

9. We recommend salt reduction to a maximum intake of 5 g daily. (1B)
10. We suggest that fluid restriction, together with salt reduction, should be advised. Fluid restriction should be individualized considering urine output, fluid gains between dialysis and ultrafiltration. (2D)
11. We suggest that exercise should be considered as a strategy to reduce BP in those receiving haemodialysis. (2D)
12. We suggest that a combination of aerobic and resistance exercise at least 3 times per week of moderate to vigorous intensity, either during or in between dialysis, would be most likely to reduce BP in haemodialysis patients. (2B)
13. We suggest that haemodialysis units consider adoption of strategies to support patient adherence to lifestyle changes. (2D)

Dialysis and dialysate

14. We suggest that extended dialysis hours should be considered for individuals who fail to achieve adequate BP control or experience IDH during the standard thrice weekly dialysis if the resources are available. (2A)

15. We suggest that lowering dialysate temperature can reduce incidence of IDH in patients prone to this condition. (2A)
16. We suggest that online haemodiafiltration (HDF) could be trialled in patients experiencing symptomatic IDH to improve CV tolerance of treatment. (2B)
17. We suggest that HDF should only be considered once alternative causes for IDH have been addressed and where patients have failed to respond to other methods. (2C)
18. We suggest that HDF should *not* be used as a treatment strategy to control BP in patients who are hypertensive. (2C)
19. We recommend that bicarbonate-based dialysate should be used rather than acetate-based solutions to reduce IDH risk. (1B)
20. We suggest that low magnesium dialysate concentrations (≤ 0.25mmol/L) be avoided, particularly if dialysate calcium is 1.25mmol/L, in patients at risk of IDH. (2D)
21. We suggest the use of Icodextrin can be useful to control BP when used in conjunction with effective setting and probing of target weight in people who are on peritoneal dialysis. (2D)

Dry weight optimization

22. We suggest that patients on dialysis (both haemo- and peritoneal dialysis) should avoid significant over or underhydration. (2A)
23. We suggest that dialysis patients should be assessed regularly in a systematic manner for fluid volume status to guide alterations to their dry/target weight and ultrafiltration volume. (2C)
24. Multiple technologies are available to aid fluid volume management in dialysis patients including continuous blood volume monitoring, inferior venacaval diameter measurement, lung ultrasound and bioimpedance spectroscopy. There is inadequate evidence to recommend one method as superior to another or clinical assessment of fluid volume status. (2C)

Medication

25. We recommend BP lowering medication to reduce all-cause and CV mortality in adult dialysis patients. (1B)
26. We suggest β-blockers (βBs) as first line and calcium channel blockers (CCBs) as second line BP lowering medication in adults on haemodialysis, based upon BP lowering efficacy. (2B)
27. We suggest ACE inhibitors (ACEi) as third line BP lowering medication in adults on haemodialysis, based upon BP lowering efficacy and enhanced risk of hypotension and discontinuation compared to βBs and CCBs. (2B)
28. We suggest ACEis or angiotensin receptor blockers (ARBs) as first line BP lowering medication in people on peritoneal dialysis, based upon evidence that these classes of antihypertensive may slow loss of residual kidney function. (2B)
29. We suggest mineralocorticoid receptor antagonists (MRAs), combined with careful monitoring of plasma potassium levels, may be considered in those with difficult to control BP. (2B)
30. We suggest advising people on haemodialysis *against* the practice of omitting BP lowering medications prior to dialysis sessions. For those in whom BP lowering medication is implicated as contributing to IDH we suggest advising consistent evening dosing instead. (2D)
31. We suggest that, where β-blockers are used, those with low dialysability are generally preferred in those receiving haemodialysis. (2C)
32. We suggest that use of BP lowering medication with prolonged half-lives (e.g. atenolol, amlodipine, lisinopril or enalapril) in people on haemodialysis could be considered in those who are non-adherent to medication when combined with dosing at the end of the dialysis session. (2D)
33. We suggest that L-carnitine and/or oral midodrine may be considered as part of a multi-faceted approach to management of IDH although data supporting usage is limited. (2C)

Children & young people (CYP)

34. We suggest that when measuring BP in CYP on dialysis, the clinical setting and assessment method should be standardised. (2C)
35. We suggest that the best accepted practice for diagnosing hypertension in CYP on dialysis is with 24-hour ABPM. This should be performed at least annually once children reach a height of 120 cm. (2B)
36. We suggest if ABPM is not feasible, standardised in-centre BP measurements and/or home BP monitoring (HBPM), should be used to assess BP control. (2D)
37. We suggest for CYP receiving in-centre haemodialysis, BP should be monitored at every dialysis session (including pre-, intra- and post-dialysis measurements), to aid with assessment of required fluid removal. This should be done in conjunction with weight measurement and clinical evaluation of fluid status. (2C)

38. We suggest for CYP on dialysis, BP should be targeted to < 90th percentile for age, height and sex on non-dialysis days. (2D)
39. We suggest there is inadequate evidence to provide target BP ranges for HBPM in CYP on dialysis although this method may be used as an adjunct to in-centre measurements. (2D)
40. We suggest for CYP on dialysis, baseline and annual echocardiography should be performed to assess for morphological left ventricular changes that may indicate hypertension-mediated organ damage. (2C)
41. WE suggest for CYP with elevated BP on dialysis, salt intake should not exceed the age-related upper limit of recommended daily intake (RDI) although nutritional requirements should be regularly reviewed by dietetic colleagues. (2B)
42. We suggest antihypertensive medications should be considered if BP remains uncontrolled despite lifestyle interventions (e.g. fluid and salt restriction) and optimised fluid removal, particularly in the context of underlying target organ damage. (2C)
43. We suggest when prescribing antihypertensive medications for CYP on dialysis, there is insufficient evidence to support the first-line use of any specific single agent or drug class. Both patient-specific factors, including the pharmacokinetics and dialysability of a drug (Appendix D), should be considered with the support of pharmacist colleagues. (2D)

Practice point

1. We recognise that in adult dialysis patients there may be circumstances in which a clinician and/or patient prefers to base BP management decisions on out-of-office (HBPM or ABPM) or standardized clinic BP readings. Whilst we have not suggested a target BP range based on HBPM due to a paucity of evidence, we note that observational studies utilising HBPM or standardised clinic BP readings generally demonstrate a linear relationship between BP and adverse outcomes, and that a systolic BP approximately ≤ 130 mmHg appears to be associated with lowest risk of all-cause and/or CV mortality.

Rationale for clinical practice guidelines

Measurement of blood pressure

There is no universal agreement on the measurements that should be used to diagnose hypertension and monitor BP in people on dialysis, even though they have their BP checked more frequently than any other group of patients. There is absence of randomised control trial (RCT) data comparing various BP monitoring techniques and their effects on long-term outcomes. However, there are emerging themes from the current research that must be incorporated into clinical practice for people receiving haemodialysis. The evidence is even more scarce in people receiving peritoneal dialysis. Therefore, recommendations made here are based on either very low-grade evidence or expert opinion.

Diagnosis

For the minority of patients who may not already have hypertension diagnosed prior to starting dialysis, we recommend using ABPM as gold-standard to diagnose hypertension. Fagugli et al. compared 48-hour ABPM that included a dialysis day and found that 24-hour APBM either on interdialytic period or dialysis day is not different from 48-hour ABPM. Prevalence of hypertension diagnosed in this study (SBP > 140 mmHg) was 80% compared to 61.7% diagnosed by predialysis SBP of > 140 mmHg [10].

For people on haemodialysis, BP measured outside the dialysis unit has more prognostic value [4, 11] and both studies suggest ambulatory BP is more closely associated with the risk of all-cause mortality than home BP. Alborzi et al. studied this in a cohort of 150 people on haemodialysis and found that every 1 standard deviation (SD) increase in (SBP 22.3, DBP 13.8 mmHg) was associated with 50% increase in mortality when ambulatory BP was used, whereas home BP was associated with 35% (SBP) and 40% (DBP) elevation in risk respectively [11]. There was no association seen with dialysis unit SBP or DBP. Agarwal et al. report similar findings in a larger cohort of a similar population and demonstrated an increased risk of all-cause mortality with increasing SBP using both ABPM and HBPM but not with dialysis unit BP [4].

There is very limited data on using HBPM to diagnose hypertension in dialysis patients. Agarwal found one week averaged systolic BP of 150 mmHg or post-dialysis standardized BP of 122 mmHg has both high sensitivity and specificity to predict hypertension diagnosed by ABPM [12].

Monitoring

Routine pre- and post-dialysis BP are poor estimates of average BP measured by ABPM. In a meta-analysis of 692 haemodialysis patients, SD of the difference of the pooled observations between ambulatory SBP and predialysis SBP was 16.7 mmHg with wide limits of agreement of 41.7 mmHg to − 25.2 mmHg [13]. In addition to inaccuracy, dialysis unit BP doesn't have any prognostic value as demonstrated in a cohort of 326 maintenance haemodialysis patients, where a strong relationship was observed for mortality over an average of 32 months with increasing quartiles of BP measured by 44 h ABPM and HBPM but not for dialysis unit BP [4]. Therefore, we suggest that dialysis unit BP should only be used to guide

Table 2 The relative merits and demerits of different BP measurement techniques in haemodialysis patients

Different BP measurements in HD patients– relative merits and demerits

BP measurement technique	Advantages	Disadvantages
Ambulatory monitoring (ABPM)	▪ Good reproducibility ▪ Good prognostic accuracy **Gold Standard**	▪ Low patient acceptability ▪ Expensive, ▪ Complex to utilise in routine practice
Home monitoring (HBPM)	▪ Reproducible ▪ Good prognostic accuracy ▪ Widely available ▪ Relatively inexpensive ▪ Empowers patients	▪ Need motivated patients ▪ Noncompliance ▪ Needs training
Standardised out-of-centre	▪ Linear relationship with outcomes ▪ Easy to utilise in practice	▪ Clinic attendance on a non-HD day ▪ Prognostic accuracy lower than ABPM and HBPM
Standardised peri-dialytic	▪ More accurate than routine BP ▪ Better prognostic value than routine BP - especially post-HD	▪ Time constraint ▪ Difficult to utilise in a busy dialysis unit
Routine peri-dialytic	▪ Readily available	▪ Imprecise ▪ Confounded by various healthcare and patient related factors ▪ 'U' or 'J' shaped relationship with outcomes ▪ Poor reproducibility

+ Prognostic accuracy -

Ease of utilisation + -

safety of dialysis sessions rather than to inform long-term management of elevated BP and CV risk. This raises an important question as to which other measurements might be more appropriate to use for long term management of hypertension in people on dialysis.

As outlined above ABPM seems to have most evidence and is used in studies as a gold standard for comparison of BP measurement methodologies. It is, however, not feasible to use ABPM routinely on patients, whereas HBPM and standardised clinic BP measurement are two alternatives. There is evidence that out-of-unit BP measurements including standardised clinic BP [14] and HBPM and ABPM [4] show a linear relationship to mortality. Further, there is a linear relationship between out-of-unit BP readings and CV risk [14].

There are some studies using HBPM to guide long term management of hypertension and these show that it is feasible, safe and acceptable to people on dialysis [15] and it more closely reflects changes in interdialytic ABPM when compared to standardised pre- and post-dialysis BP measured in-centre [16]. A small RCT of 96 people on haemodialysis showed that treatment decisions based on HBPM resulted in better BP control as determined by ABPM as the gold standard, compared to routine pre-dialysis BP [17]. Although these differences in BP control did not translate into lower left ventricular mass index in the HBPM cohort, follow-up was short at 6 months. The frequency of HBPM measurements differs between studies. However most suggest twice a day BP monitoring for 4 consecutive days [18, 19]. Perhaps more importantly, however, it is not yet clear what the target BP should be when utilising home BP readings in people on dialysis.

Dialysis unit BP measurements are an inaccurate estimate of interdialytic BP [13]. While every effort should be made to standardise BP measurement in the dialysis unit, it will be logistically challenging to implement in routine care. Furthermore, there is no evidence to guide what the target BP should be for standardised pre- and post-dialysis BP measurements. On the other hand, there is evidence that HBPM is more accurate [12, 16] and better predicts CV events and mortality [4, 11]. Therefore, a focus for future research should be how we can incorporate HBPM and standardised out-of-unit BP measurements into clinical practice. Table 2 summarises the advantages and disadvantages of different BP readings available for dialysis patients.

Blood pressure targets

Numerous observational studies have described a 'U' or 'J' shaped relationship between pre-dialysis systolic BP and outcomes in people on haemodialysis where only low and, usually, very high BPs are associated with excess all-cause or CV mortality. Such studies have typically utilised 'usual' non-standardised BP measurements and there is poor agreement between 'usual' BP readings

and those taken in standardised conditions [20]. Besides a likelihood that ascertainment of 'usual' BP readings doesn't adhere to best practice [21] other factors account for the 'U' or 'J' shaped relationship which contrasts with the linear relationship between BP and adverse outcomes seen in the general hypertensive population and in haemodialysis patients where other measurement methods (standardised, HBPM, or ABPM) are utilised. For example, in the CRIC study standardised BP (mean of 3 seated BP measurements) taken away from the dialysis unit is linearly related to mortality, whereas pre-dialysis measurements retain a U-shaped association within the same cohort of patients [14]. There are likely to be several explanations for these discrepant relationships, not least that pre-dialysis BP reflects an individual's physiological ability to tolerate volume loading. Importantly, people on haemodialysis differ from the general hypertensive population in another important regard: a tendency to suffer IDH which affects ~ 30% of dialysis sessions. Accumulating evidence suggests that IDH and ultrafiltration volumes are independently predictive of myocardial stunning which, in turn, is predictive of left ventricular systolic dysfunction and myocardial fibrosis [22], vascular access thrombosis [23] and mortality [24].

A single feasibility and safety study randomised 126 hypertensive haemodialysis patients to a "standard" BP target range of 155–165 mmHg or an "intensive" range of 110 to 140 mmHg, measured in a *standardised* manner in the immediate pre-dialysis period [25]. Of seven prespecified feasibility objectives [26], two were achieved (mean separation of BP between arms by > 10 mmHg; 75% participants providing minimum required number of standardised unit BP measurements), two were not achieved (IDH in intensive arm not > 20% higher than standard arm; ≥66% of required HBPM/ABPM measurements) and three were not reported. Additionally, 55% of the 281 participants that consented did not progress to randomization, predominantly due to not achieving SBP 155 mmHg despite back-titration of medications ($n = 65$, 23% of those consented) or participant-initiated withdrawal (45, 17%). Nonetheless the authors judged that a full scale RCT would be feasible, presumably with modifications. Importantly, and despite not being powered for definitive conclusions, a number of safety signals emerged around intensive BP lowering: those in the intensive arm experienced a non-significant three-fold increase in vascular access thrombosis (incidence rate ratio, IRR 3.09) and a non-significant IRR of 1.61 for hospitalisation.

Besides this study, the remaining data to guide BP targets is predominantly derived from observational cohort studies utilising non-standardised pre- and post-dialysis BP measurements described above. These studies fairly consistently show worse outcomes where pre-haemodialysis BP is below ~ 140 mmHg. A facility-level analysis of DOPPS data designed to minimise effects of unmeasured patient-level confounding found lowest mortality in those with a pre-haemodialysis BP of 130 to 159 mmHg [27]. Other studies have found lowest risk of CV events with a pre-haemodialysis systolic BP range of 140 to 170 mmHg [5]; lowest risk for all-cause mortality at 165 mmHg and for CV mortality at 157 mmHg [28] and lowest risk for all-cause mortality at 152 mmHg and CV events at 143 mmHg [29]. Considering the totality of data available, we suggest for haemodialysis units or individual clinicians wishing to base BP targets on pre-haemodialysis measurements, that a systolic BP range of approximately 140 to 165 mmHg appears to be associated with the fewest short- and long-term adverse outcomes.

Data on pre-dialysis diastolic and post-dialysis BP are more limited. In the Robinson study, facility-level pre-dialysis DBP between 60 and 99 mmHg and post-dialysis SBP between 120 and 139 mmHg were associated with lowest mortality [27]. The same analysis identified post-dialysis DBP < 70 mmHg to be associated with increased mortality with no corresponding upper limit. Hannedouche did not find a pre-dialysis DBP level that was indicative of minimal risk for all-cause mortality. For CV mortality, a DBP of 90 mmHg was found to be associated with lowest CV mortality, although 95% confidence intervals for hazard ratio were wide, and depart from 1.0 at a DBP of approximately 70 mmHg [28]. Post-dialysis BP readings were not analysed.

Limited observational data suggest that demographic characteristics such as age [30], co-morbidities including diabetes [31], atrial fibrillation and heart failure [32] and biomarkers, e.g. troponin I and NT-proBNP [33], influence the nature of the relationship between BP and outcomes, such that in younger patients without co-morbidity and normal biomarkers only higher SBP tends to be associated with adverse outcomes, leading us to suggest that aiming for lower BP targets may be acceptable in this population.

Studies using HBPM, ABPM and standardised clinic BP readings generally demonstrate a positive linear relationship between BP and outcomes [4, 5, 11]. For example, a study of 326 predominantly African-American haemodialysis patients dialysing in Indiana, US found an average home SBP of 120–130 mmHg to be associated with lowest all-cause mortality [4]. Similarly the CRIC study investigators found in 377 haemodialysis patients that, compared to those with a standardised SBP < 128 mmHg, adjusted HR for CV events was 2.14 (95% CI 1.17 to 3.9) and 2.9 (95% CI 1.55 to 5.42) for those with SBP 128 to 145 and > 145 mmHg respectively (5). A similar relationship was demonstrated in a recent Brazilian study in 2,672 haemodialysis patients, where BP was measured in the inter-dialytic period albeit probably not in

a standardized manner. Compared to a reference SBP of ≥ 171 mmHg, incidence of CV events was reduced in those with SBP 101–110 (HR 0.65, 95% CI 0.46–0.90), 111–120 (HR 0.66, CI 0.49–0.89), 121–130 (HR 0.75, CI 0.57–0.98), and 131–140 mmHg (HR 0.76, CI 0.6–0.97) [34].

However, it is important to recognise important limitations of these studies. Firstly, they were conducted in small, often single-centre, populations that might be unrepresentative of the wider UK dialysis population; and, secondly, some excluded participants with important CV co-morbidities such as atrial fibrillation [4, 11]. In a European study using ABPM in 344 haemodialysis patients, a *U-shaped* relationship between ambulatory SBP and CV and all-cause mortality was observed in the whole study cohort, whereas when ~ 30% with either atrial fibrillation or heart failure were excluded a positive linear relationship between BP and outcomes was observed [32]. These contradictory findings emphasize the inherent difficulties in using observational data to guide clinical practice even when using 'gold-standard' BP measurement methodologies, and the need for well-designed interventional trials to define appropriate BP targets in those on dialysis.

In the peritoneal dialysis population there are few data to guide BP targets with the ISPD recommended target of < 140/90 mmHg being extrapolated from the general and CKD population [35]. One large prospective cohort study undertaken in China found a U-shaped relationship between usual clinic SBP – presumably non-standardized – and all-cause and CV mortality, with an SBP range of 119–141 being associated with lowest hazard ratio for adverse outcomes [36]. An analysis of UK Renal Registry data in peritoneal dialysis suggested that higher BP is associated with reduced mortality in the first 12 months following commencement of renal replacement therapy (RRT), except in the subgroup listed for transplantation within six months of starting RRT [37]. Assuming, as the authors suggest, that early transplant listing is a proxy for minimal comorbidity, it is suggested that we should aim for lower BP targets in less comorbid peritoneal dialysis patients although no specific BP targets are identified by this study.

Lifestyle modification

Salt intake

Volume expansion, net positive sodium balance, renin-angiotensin-aldosterone system (RAAS) and sympathetic nervous system activation contribute to high BP in people on dialysis [38]. Lifestyle interventions that attenuate these effects may have some impact on BP control.

Volume expansion by salt and water is thought to be a major contributor to hypertension in people on dialysis [39, 40]. Higher dietary sodium intake is independently associated with greater mortality in people on haemodialysis [41]. Reduction in salt intake lowers BP in the general population, in people with hypertension of all ethnicities, in people with and without diabetes and in people with chronic kidney disease (CKD) [42–46]. A low salt diet may be particularly beneficial in people on dialysis because they are largely dependent on the dialysis process to remove excess sodium and water. Studies in the 1990s explored dietary salt reduction together with dialysis interventions to achieve optimal dry weight and BP control [47–49]. The studies reported to date, however, are poorly designed, uncontrolled and underpowered to show a BP difference following dietary interventions. A meta-analysis of randomized controlled trials in 91 haemodialysis patients has shown that a mean difference in salt intake of 5 g/day was associated with a reduction in BP of 8/4 mmHg (95% CI 4.8 to 12/2.2 to 6.6) [50]. A subsequent systematic review and meta-analysis of salt reduction in all stages of CKD included 5 studies in haemodialysis patients and found reducing salt intake reduced systolic BP by 6.32 mmHg (95% CI -11.04 to -1.60) and diastolic BP by 3.46 mm Hg (95% CI -6.39 to -0.54) [51].

Fluid restriction

Volume overload is a risk factor for mortality amongst dialysis patients [52]. Efforts to achieve ideal target weight are discussed in other sections of this guideline, some of which will involve salt and water restriction, increased ultrafiltration and longer dialysis times to facilitate achieving euvolaemia. There is an acceptance that fluid restriction requires concomitant salt intake reduction, but evidence for fluid restriction alone lacks any contemporary evidence base [53].

Other dietary approaches

There is good evidence in the general population that a diet high in fruits and vegetables such as the Dietary Approaches to Stop Hypertension (DASH) diet can help to lower BP [44, 54]. However, trials in people on dialysis are lacking. An observational cross-sectional study in 2022 assessed the diets of 583 individuals on haemodialysis and categorized according to adherence to the DASH diet [55]. Higher adherence to the DASH diet was associated with lower serum potassium levels, although no difference in BP was observed between the groups (personal communication with author). This study allays concerns that a DASH diet may lead to hyperkalaemia and paves the way for further research.

Several authors have investigated diets rich in polyphenols. A systematic review of three trials found a reduction in DBP but not SBP [56]. Another small study has reported a reduction in SBP and DBP with pomegranate juice [57]. Due the small number of trials with low

numbers the evidence is not strong enough to make a recommendation.

Although there is a lack of evidence in the dialysis population, we suggest that other lifestyle measures to control BP are adopted including maintenance of ideal body weight, excessive intake of alcohol, coffee and caffeine rich foods and drinks should be discouraged as per advice for the general population with hypertension. We also suggest that specialist renal dietitians are best placed to provide dietary advice to those on dialysis. Patients on dialysis have complex dietary requirements and renal dietitians can provide individualized, holistic advice.

Exercise

A number of systematic reviews and meta-analyses have considered whether exercise reduces BP in dialysis. Most studies have focused on intra-dialytic exercise.

In a 2019 meta-analysis of intradialytic exercise trials that included BP as an outcome, there was a significant reduction in SBP of 4.87 mmHg (95% CI -9.2 to -0.5, $p = 0.03$) and DBP of 4.11 mmHg (95% CI -6.5 to -1.72, $p = 0.0007$) [58]. In another meta-analysis of intradialytic exercise, the effect of aerobic exercise and combined exercise (aerobic and strength) were analysed separately. The authors found a significant reduction in SBP of 10.07 mmHg (95% CI --16.36 to – 3.78) with aerobic exercise and a reduction in DBP of 5.76 mmHg (95% CI -2.7 to -8.83) with combined exercise, but there was no reduction in SBP with combined exercise or DBP with aerobic exercise [59].

Other meta-analyses have explored the effects of all types of exercise, intradialytic exercise, exercise on non-dialysis days, aerobic and combined strength and aerobic, on BP in dialysis populations. One included 16 studies of at least 8 weeks and found that only combined training reduced BP. SBP was reduced by 9 mmHg (95% CI -13 to -4) and DBP by 5 mmHg (95% CI -6 to -3) [60]. The other considered whether the intensity of exercise was important. Moderate to vigorous exercise was found to be most effective at reducing SBP by 8.8 mmHg (95% CI -17 to -1.6) and DBP by 4.9 mmHg (95% CI – 9.9 to – 0.4) [61]. Both meta-analyses suggest that combined training was the most effective.

The most comprehensive report on exercise in dialysis populations is a 2022 Cochrane systematic review of RCTs and quasi-RCTs of any structured exercise programs of eight weeks or more in adults undergoing maintenance dialysis compared to no exercise or sham exercise [62]. The authors report uncertainty as to whether exercise training reduces the risk of death and no studies reported CV events. They found that exercise training was likely to improve functional capacity and depressive symptoms with some degree of certainty. The effects of exercise on BP were analysed in smaller meta-analyses, which considered different types of exercise regimens separately. There was a significant reduction in SBP 8.69 mmHg (95% CI -13.69 to -3.69) and DBP 4.45 mmHg (95% CI -5.98 to -2.91)- with combined exercise, but authors consider the results are of very low certainty due to the high risk of bias, the short duration of the interventions and follow-up and the low number of participants in the included studies. There was no significant reduction in BP with aerobic exercise alone. We are unable to make any recommendations for exercise reducing BP in PD patients due to the lack of evidence.

Self-care engagement

Achieving BP targets is notoriously difficult for a combination of reasons. Some small studies have reported recently that explore the impact of interventions on adherence to diet, fluid and BP medications [63]. An educative nursing intervention in 118 participants recruited from 6 haemodialysis units in the USA found that BP education sessions, with home BP monitoring twice daily, diarising salt and fluid intake weekly for a 12-week period vs. standard care with BP monitoring and medication adjustment by health care providers on a weekly basis in the haemodialysis unit significantly decreased both SBP and DBP in the treatment group. Another non-controlled study has reported a greater likelihood of reduction in SBP in a cohort of 58 haemodialysis patients, who received counselling from a pharmacist on adherence to BP medications [64]. We considered that these studies are not sufficient to make a recommendation on the role of supportive care interventions in BP management of dialysis patients, but measures to support adherence to treatment goals is possibly an area for further research.

Dialysis and dialysate

Studies of extended duration of haemodialysis (beyond the conventional 3 to 5 h three times a week) generally support improved BP control and or reduced BP medication burden, with inconsistent findings on left ventricular mass measurements [65–68].

More recently, however, extended duration HD has been shown to be associated with lower mortality. This is true whether the haemodialysis session is extended, or the frequency of haemodialysis is increased [69, 70]. The Frequent Hemodialysis Network (FHN) Daily Trial reported that 2 months of a frequent haemodialysis regimen lowered pre-dialysis SBP by 7.7 mm Hg (95% CI: –11.9 to – 3.5) and DBP by 3.9 mm Hg (95% CI – 6.5 to – 1.3) [71] whilst the FHN Nocturnal Trial reported a reduction in systolic SBP from baseline of 7.9 ± 18.4 mm Hg in the nocturnal cohort at 12 months [72]. Short daily and nocturnal schedules also reduce the per-session probability of IDH by between 20 and 68%, while well-being and shorter recovery times are observed in those

having extended hours HD, possibly mediated through a combination of optimizing sodium and volume status or enhanced solute clearance [68, 72].

Whilst there is good evidence to support extending haemodialysis for improved BP control, either by longer sessional hours or increased frequency of sessions, universal adoption of practice is unlikely due to the high cost of providing this service. The NightLife study is ongoing and will evaluate the cost and clinical effectiveness, including effect on BP, of thrice-weekly extended in-centre nocturnal haemodialysis versus daytime haemodialysis [73].

It has also been reported that HDF, compared to haemodialysis, improves cardiovascular stability during treatment sessions and that it reduces the frequency of IDH [74–76]. These findings, however, are not consistent across all studies. Two meta-analyses on this topic have reached differing conclusions on the intervention effect on IDH, but neither study reports a significant effect on BP outcomes [77, 78]. These inconsistent findings may be explained by failure to achieve sufficient convective volumes, different haemodialysis modalities (low-flux vs. high-flux) and HDF techniques, participant demographics and access type. With the view that there was still clinical equipoise as to the potential benefits of high dose HDF compared to conventional high-flux haemodialysis, the CONVINCE and H4RT trials were conceived to address the issue. CONVINCE has recently reported on the primary outcome; a reduction in death from any cause in the HDF group (hazard ratio, 0.77; 95% CI, 0.65 to 0.93), although risk of death from CVD was similar in HDF and high-flux haemodialysis treatment arms [79]. At present, there remains insufficient evidence to recommend the widespread implementation of HDF on the grounds of cardiovascular benefit. H4RT trial outcome data is awaited [80].

Active BP lowering in haemodialysis patients can lead to increased frequency of IDH. Large observational studies have demonstrated an association between frequency of IDH and mortality [81–83]. It is therefore prudent that steps are taken to minimize IDH especially in IDH prone patients. Comparative studies have shown that haemodialysis tolerance, which includes IDH, is better when using bicarbonate rather than acetate dialysate [69, 70]. However, we note that in an RCT [83] there appeared to be a dissociation between IDH reduction (reduced with temperature lowering) and mortality (no effect).

Different strategies for changing dialysate temperature have been studied including biofeedback and different degrees of temperature lowering. A systematic review [84] of 11 RCTs concluded that lowering dialysate temperature reduced the rate of IDH by 70% (95% CI 49–89%). However, more recent studies including a multi-centre RCT enrolling 73 patients for 12 months [85] and a cluster randomised study [86] did not demonstrate an advantage of setting dialysate temperature − 0.5 °C below body temperature vs. 37 °C. On other hand, a recent large observational study using the Fresenius NephroCare Eclid database [83] did show that in case-mix, facility-level adjusted incident haemodialysis patients, a 0.5 °C reduction in dialysate temperature was associated with a 33% risk reduction of IDH. It is possible that patient selection (patients at high risk of IDH [83] vs. patients at lower risk of IDH [85, 86]) accounts for the different outcomes.

Results of studies examining effect of changing dialysate sodium concentration on BP and IDH have been conflicting and covered in recent systematic reviews [87, 88]. Whilst studies have shown that lowering dialysate sodium reduces pre-dialysis mean arterial pressure (MAP) by 3.6 mmHg (95% CI -5.7 to -1.7) and post-dialysis MAP by 3.3 mmHg (95% CI -1.7 to -4.8) [89–92], there is also weak evidence that lowering dialysate sodium may increase rate of IDH in IDH-prone patients [89, 90]. The effect of varying dialysate sodium (sodium profiling) during a dialysis session has also been examined. Different profiles of sodium reduction were the subject of a meta-analysis [93]. Stepwise rather than linear reduction of sodium concentrations was found to reduce IDH. A single centre RCT [94] also confirmed that profiling reduced IDH although, contrary to the meta-analysis, the authors reduced dialysate sodium concentrations linearly. This study demonstrated similar benefit on IDH by dialysate cooling but the benefits were not additive. Thee DOPPS cohort study demonstrated that routine use of sodium profiling was associated with higher all-cause mortality [95]. Given the theoretical risk of "sodium loading" leading to increased dialytic weight gain, we do not feel there is enough evidence to recommend sodium profiling. Sodium profiling may have a role in IDH prone patients but there is insufficient evidence to recommend as routine care.

The correction of chronic metabolic acidosis is an important goal of dialysis and dialysate bicarbonate should be optimised for mineral bone disorder and nutrition. However, acid-base balance might have an acute effect on BP and therefore IDH. We acknowledge that DOPPS [96] have suggested a reduction of the dialysate bicarbonate concentration can be considered for patients with significant peri- and post-dialysis alkalaemia with frequent IDH unresponsive to classical management protocols. However, we considered that the evidence based on three studies was weak [97–99].

There are few studies examining the effect of adjusting dialysate magnesium [100, 101] on BP and IDH. A single study of 14 patients [100] suggests that low dialysate concentration of magnesium (0.25mmol/L) be avoided with dialysate calcium concentration of 1.25mmol/L due to

increased risk of IDH. We considered that the evidence base too weak to make a recommendation. A study has examined the effect of dialysate calcium concentration on IDH risk [98] but we suggest that dialysate calcium should be adjusted in accordance for optimal management of mineral bone disorder.

There is evidence that long intraperitoneal dwells of 7.5% icodextrin can achieve greater ultrafiltration than 2.27% glucose dwells [102]. Sodium removal in peritoneal dialysis is dependent on convection so it is to be expected that using icodextrin can improve fluid status [103] and hence help to attain dry weight in people on peritoneal dialysis. Icodextrin can achieve greater UF during long dwells in patients who are classified to have fast peritoneal solute transfer rates (PSTR) without the need for the higher concentration of glucose containing peritoneal dialysis fluids.

Dry weight optimization

Optimum fluid volume management in people on dialysis is important for both patient experience and outcomes. Both volume overload and depletion are associated with poor outcomes [104–108]. Therefore, accurate 'dry' or 'target' weight assessment is critically important. The clinical assessment of target weight is based on symptoms, skin turgor, peripheral oedema, jugular venous pressure, BP measurement and lung auscultation. It is often a 'guesstimate'. Several technologies are now available to aid fluid volume assessment including blood volume monitoring, inferior vena caval diameter measurement, lung ultrasound scan and bio-impedance spectroscopy (BIS).

A large, international, observational study and small single centre RCTs suggest that protocolised clinical assessment of volume status in people on haemodialysis may be associated with better clinical and patient reported outcomes [95, 109, 110]. This is further supported by the recently published BISTRO trial demonstrating that standardised clinical assessment of fluid volume status is equivalent to standardised clinical assessment augmented by BIS in maintaining residual kidney function in people on haemodialysis [111].

Of the technologies available to assist fluid volume management, BIS is most extensively studied. However, when compared with routine clinical assessment of target weight, none of these technologies have provided consistent benefit in terms of clinical outcomes [105, 112–130]. These include two recently published, well-conducted, RCTs (LUST [131] and BISTRO [111]). Furthermore, some of these found increased adverse patient reported outcomes in the technology assisted fluid management group.

Hypertension is a common consequence of fluid overload in dialysis patients. Several observational studies and small trials demonstrate improvement in BP control in both people on peritoneal and haemodialysis [109, 113, 116, 119, 122, 123, 130, 132]. However, there are also other studies that fail to show the BP lowering effect of active fluid volume management. Importantly, the BISTRO trial did not demonstrate difference in BP control between protocolised fluid volume management compared with BIS added to protocolised fluid volume management in people on haemodialysis [111]. Therefore, there is lack of consistent and firm evidence to support any strong recommendations for fluid volume management to control BP in dialysis patients.

Medication

BP lowering with medication has been demonstrated to reduce all-cause and CV mortality in people on dialysis [133]. The pooled reduction in BP was −4.5/-2.3 mmHg and BP lowering was associated with relative risks (RR) of CV events (RR 0.71, 95% CI 0.55–0.92; $p = 0.009$), all-cause mortality (RR 0.80, 0.66–0.96; $p = 0.014$), and CV mortality (RR 0.71, 0.50–0.99; $p = 0.044$) respectively compared with the control group. Eight RCTs were included in this meta-analysis of which 7 were in haemodialysis populations and only 4 explicitly included participants with elevated BP. Therefore, there is little direct evidence of effects of BP lowering medication on outcomes in people on peritoneal dialysis. BP-lowering medications used in the intervention arm of included trials was heterogenous: ARBs in three, ACEi in two, βBs in two and CCBs in one. A further meta-analysis published at the same time was not included in our evidence synthesis since it included the same original publications, did not provide a pooled estimate of BP reduction, but it did reach very similar conclusions regarding event rates [134].

We identified three RCTs of BP lowering medication in dialysis patients with a primary or secondary outcome relevant to our search criteria that have been published since this 2009 meta-analyses [135–137]. These three trials were not included in the network meta-analysis described below as these were all 'treat to target' trials. One of these trials compared ACEi vs. non-renin-angiotensin system inhibition (RASi) and a second compared ARB vs. non-RAS. Both trials achieved similar BPs in intervention and comparator arms as per their respective designs, but found no difference in their respective primary composites end-point of CV mortality, non-fatal stroke or myocardial infarction (plus heart failure admission in the first trial) [136, 137]. A third trial compared a βB to ACEi in predominantly African-American haemodialysis patients [135]. Despite a 'treat to target' design with a goal home BP of ≤140/90 mmHg, in post-hoc analysis there was a slightly lower home BP ($p = 0.037$) in the atenolol arm; on 44-hr ABPM, there was numerically

lower BP in the atenolol arm (-3.6/3 mmHg). This trial was terminated early due to an excess of CV events in the ACEi group, with no difference found in primary outcome of change in left ventricular mass index.

We were unable to draw firm conclusions regarding choice of antihypertensive drug class in dialysis patients due to lack of consistent evidence of reduction in all-cause, CV mortality or other relevant endpoints favouring any particular class. We have therefore based several of our suggestions on the findings of a recent network meta-analysis that assessed comparative BP lowering efficacy of antihypertensive medications in people on haemodialysis [138]. Mineralocorticoid receptor antagonists (MRAs) and βBs were most effective at reducing systolic BP (SBP), compared to both placebo (MRAs: -10.8 mmHg; βBs: -8.7 mmHg) and other classes (e.g. MRAs – 6.4 mmHg vs. ACEi and βBs -4.4 mmHg vs. ACEi). CCBs and ACEi lowered BP compared to placebo by -4.6 mmHg and – 4.3 mmHg respectively. Additionally, the βB vs. placebo comparison provided a high GRADE (Grading of Recommendations Assessment, Development and Evaluation) confidence rating for effect estimate whereas other comparisons in this meta-analysis varied from moderate (e.g. CCB and ACEi vs. placebo; CCB and βB vs. ACEi) to low or very low confidence ratings.

Hypotension and discontinuation due to adverse effects were more common with ACEi (RR for hypotension and discontinuation were 6.62 and 1.77 vs. control). For these reasons, and in the absence of a clear cardioprotective class effect of RAS blockade in dialysis patients, we have suggested use of ACEi as third line in those on haemodialysis. In those on peritoneal dialysis there is evidence from small RCTs that ACEi or ARBs preserve residual renal function and urine output compared to control groups, despite similar reductions in BP over 12 to 24 months [139, 140] with a mean difference in GFR compared to controls of + 0.93 mL/min/1.73 m^2 (95% CI 0.11–1.75) for ACEi and + 1.11 mL/min/1.73 m^2 (95% CI 0.38–1.83) for ARB in pooled analyses [141, 142], leading us to suggest these classes as first line antihypertensives in people on peritoneal dialysis.

Although MRAs are most efficacious at lowering SBP, higher rates of discontinuation due to adverse effects (RR 3.35) were observed for this class, with a numerically increased risk for hyperkalaemia (RR 1.63, 95% CI 0.75 to 3.57). Additionally, the confidence rating for the effect estimate of MRA vs. comparators, including placebo, was either low or very low. Finally, although three meta-analyses [143–145] confirm BP-lowering efficacy of MRAs and impressive reductions in pooled estimates of CV and all-cause mortality (RR around 0.4 for all-cause mortality), concerns exist around the quality of some of the original trials of MRAs. Two large global RCTs of MRAs in haemodialysis populations (ACHIEVE and ALCHEMIST) are expected to complete recruitment shortly, and these are anticipated to define the efficacy and safety of MRAs with greater confidence. For these reasons, and until publication of ACHIEVE and ALCHEMIST studies, we recommend that MRAs are considered for use only in those dialysis patients with more problematic hypertension.

Alpha-blockers (αBs), ARBs and renin inhibitors were not found to lower BP more than placebo, although confidence intervals for the former two classes were wide (-6.7 mmHg, 95% CI -14.1 to 0.7 for αB vs. placebo; -3.0 mmHg, 95% CI -8.7 to 2.6 for ARB vs. placebo) and only indirect comparison between αBs with placebo was available. Discontinuation due to adverse effects was also higher for ARBs (RR 1.57).

There are significant differences between antihypertensives in the extent to which they are removed by dialysis (see Appendix D). Theoretically, choice of antihypertensives based on dialysability may affect BP lowering efficacy, enhance BP variability and, in the case of drugs that reduce cardiac events by other mechanisms e.g. antiarrhythmic effects of βBs, dialysability may confer a more immediate effect on risk of CV events. There exists some lower quality evidence to support this hypothesis: a large propensity-matched retrospective cohort study from a Canadian haemodialysis population ($n = 6588$) found increased risk of all-cause (RR 1.4) and CV (RR 1.2) mortality in those receiving high (atenolol, acebutolol, metoprolol) vs. low dialysability (bisoprolol, propanolol) βBs [146]. Conversely, in non-adherent patients, it may be helpful to administer antihypertensives under direct supervision at the end of a dialysis session, particularly if using highly dialysed antihypertensives (e.g. atenolol, lisinopril, enalapril) that have prolonged half-lives in people on haemodialysis. Given the continuous nature of peritoneal dialysis, and paucity of relevant evidence in this population, we do not make any recommendations about in-class choice of BP lowering medication in those on peritoneal dialysis.

Anecdotal evidence suggests that dialysis patients may omit prescribed antihypertensives prior to haemodialysis sessions, either on advice of healthcare professionals or by their own decision [147]. We were unable to find evidence to support this practice. However, it is reasonable to assume that, where a person takes one or more antihypertensive medications early in the morning on 4 days of the week and 6 or more hours later than this on the remaining 3 days, this may increase BP variability. Observational studies have established increased short- and long-term BP variability as independent predictors of all-cause and CV mortality and major adverse CV events in people on haemodialysis [148]. Considering that the TIME study found that evening dosing of antihypertensive medication did not differ from morning dosing in terms of major CV outcomes in the general

population [149], we suggest that clinicians discourage omission of antihypertensives prior to dialysis sessions and instead encourage consistent night-time dosing of antihypertensive medication in those patients for whom such medication is thought to be contributory to IDH. We acknowledge that the efficacy of this approach should be tested in a clinical trial.

We found limited evidence to support pharmacological approaches to management of IDH. Evidence to support the use of midodrine is inconclusive. A meta-analysis, published in 2004, included 10 studies with 117 participants and reported nadir BP 13.3/5.9 mmHg higher in those receiving midodrine, but no consistent benefit in terms of symptom reduction [150]. All included studies had significant methodological flaws (e.g. none were of parallel group design) and were subjective to substantial risk of bias. A retrospective cohort study of 3083 patients, albeit not necessarily with confirmed IDH and subject to confounding by indication, found an adjusted incidence rate ratio of 1.37, 1.31 and 1.41 for all-cause mortality, all-cause hospitalization and hospitalization for CV causes respectively for those prescribed midodrine vs. controls [151].

Evidence to support use of L-carnitine supplementation is similarly inconclusive. A recent meta-analysis (8 studies, 224 participants) of 6 to 24 weeks duration using either cross-over or parallel group design [152]. Of the included studies, only two were judged to be at low overall risk of bias. Compared to controls, participants allocated to L-carnitine supplementation had a pooled odds ratio for incidence of IDH of 0.26 (95% CI 0.1–0.72). Subgroup analysis suggested that only oral, as opposed to intravenous, supplementation was effective and that a minimum weekly dose of 4,200 mg is required. Conversely, a recent Cochrane review (3 RCTs, 128 participants) found insufficient evidence that L-carnitine prevented IDH (RR 0.76, 95% CI 0.34–1.69; low certainty evidence) [153].

Children and young people (CYP)

Background

Hypertension (defined as SBP and DBP ≥ 95th percentile for age, height and sex) is highly prevalent in CYP on dialysis [154–156]. However, lack of BP measurement standardisation for CYP on dialysis makes interpretation of values difficult. Details regarding standardisation of BP measurement are available from clinical practice guidelines [157, 158]. Although there is lack of data demonstrating an association between hypertension in CYP on dialysis and increased incidence of CV events or mortality, several studies have reported correlation with proxy markers of CV morbidity, such as LVH and carotid intima media thickness (cIMT) [8, 159–161]. Improved BP control in children on haemodialysis, with BP maintained < 90th percentile, has been demonstrated to reduce left ventricular mass (LVM) [160]. As such, echocardiography should be conducted at regular intervals to screen for serial morphological changes.

Blood pressure measurement, treatment thresholds

Current evidence for the management of BP in CYP with CKD *not* on dialysis is to lower MAP to ≤ 50th percentile to slow CKD progression [158, 162] and to reverse adverse cardiac remodelling [163]. For patients on dialysis, particularly those with residual urine output, targeting BP to this level increases the risk of extreme BP variability and IDH. IDH is defined by the Paediatric Continuous Renal Replacement Therapy (PCRRT) working group as SBP < 5th percentile for age along with the presence of clinical symptoms [164]. As for adults, episodes of IDH in CYP affect dialysis adequacy, increase the risk of myocardial stunning, and may lead to long-term adverse clinical outcomes [164, 165]. For this reason, targeting BP to ≤ 50th percentile in CYP on dialysis is not generally recommended. Consensus recommendations for minimising the risk of IDH in CYP were published in 2019 [164].

ABPM is considered the gold standard for measuring BP in CYP, including those on dialysis [18, 166]. However, normative data do not exist for those < 120 cm in height or < 5 years of age. Furthermore, achieving compliance in younger patients may not be possible. Compared with in-centre haemodialysis measurements, ABPM enhances the predictability for identifying BP as a risk factor for target organ damage [167, 168]. Our suggestion is that an ABPM monitor should be fitted at the end of a mid-week haemodialysis session to allow for standardisation of measurement. Data on the use of HBPM are lacking in CYP on dialysis, but it may be a useful adjunct alongside ABPM to provide measurements that are less affected by the white coat phenomenon (for both patients on haemodialysis and peritoneal dialysis), by pre-dialysis fluid overload, and by BP fluctuations secondary to acute fluid removal.

This guideline suggests targeting BP to < 90th percentile for age, height and sex as a measure of minimising long-term risk of hypertension-mediated organ damage. However, our recommendations remain weak as data are not available to suggest that accepting or targeting higher BP values, such as in the range of the 90th -95th percentiles, confers worse prognosis in CYP on dialysis. The evidence base for recommendations in CYP on peritoneal dialysis are even weaker owing to a lack of published research in this area. One small study, involving 87 children, demonstrated improved preservation of residual kidney function in patients on peritoneal dialysis when SBP and DBP were maintained ≤ 95th percentile [169].

Management of hypertension and treatment targets

Within the confines of our literature search, no RCTs analysing CYP on dialysis in groups according to BP target were identified. The current evidence related to BP control in CYP is derived from several studies that have looked at BP as either a primary or secondary outcome measure following:

- Commencement of antihypertensive therapies [170, 171].
- Implementation of a blood volume monitoring algorithm to guide ultrafiltration [172, 173].
- Implementation of a haematocrit-guided ultrafiltration algorithm to guide ultrafiltration [174].
- Implementation of a bioimpedance analysis algorithm to guide ultrafiltration [175].
- Assessment of interdialytic weight gain (IDWG) and categorisation into groups according to percentage increase in IDWG [176, 177].
- Commencement of HDF compared with standard haemodialysis [178, 179].

Guidance on the upper limit of recommended daily intake (RDI) for salt is provided by the 2008 KDOQI Guideline for Nutrition in Children with CKD [180]. Age-related RDI ranges are: 3.8 g (1–3 years); 4.8 g (4–8 years); 5.6 g (9–13 years); 5.8 g (14–18 years). This guidance is supported by studies in adults demonstrating that limiting salt intake in hypertensive dialysis patients allows for optimised volume status and BP control [48, 181, 182]. A meta-analysis of paediatric trials demonstrated that salt reduction of 42% was associated with a significant reduction in both SBP and DBP in hypertensive CYP without CKD [183]. One study identified a positive simplified sodium balance (i.e. the difference between daily sodium intake and daily urinary sodium losses) to be an independent predictor of IDWG in CYP on both haemodialysis and peritoneal dialysis, although there was no correlation with SBP and DBP standard deviation scores [184]. However, salt intake remains important for growth in CYP on dialysis, particularly for those who are polyuric [185] and where dialysis prescription and modality may increase sodium removal and put the patient at risk of hyponatraemia [186]. The support of dietetic colleagues is therefore vital to develop an individualised approach for each patient.

No single antihypertensive agent, or drug class, has been demonstrated to be more effective, or have an improved safety profile, in CYP on dialysis. Data on antihypertensive therapies remain limited to those with CKD stages 2–4. Prospective analysis of 478 CYP enrolled in the Chronic Kidney Disease in Children (CKiD) study demonstrated renin-angiotensin system (RAS) antagonists were associated with reduced odds of developing LVH compared with other antihypertensive agents, although this did not reach statistical significance [187]. Data from the International Pediatric Peritoneal Dialysis Network (IPPN) registry showed lower incidence of LVH in those on RAS antagonists [159]. Alongside its antihypertensive effect, ramipril has been demonstrated to improve serum levels of inflammatory mediators and biomarkers of endothelial dysfunction compared with placebo in CYP on dialysis [171]. A clinical practice questionnaire, conducted in the US, demonstrated that dihydropyridine-CCBs and angiotensin converting enzyme inhibitors (ACEis) were the most commonly prescribed antihypertensives in CYP on both haemodialysis and peritoneal dialysis [188]. In terms of the mechanism of action, RAS antagonists are likely to be of limited benefit in anephric patients. Although there is a theoretical risk of reduced urinary excretion of potassium, or potassium accumulation in those with anuria, adult data suggest that RAS antagonists in those on maintenance haemodialysis are not associated with an increased incidence of hyperkalaemia [189]. As per the adult pharmacological recommendations in this guideline, we suggest a consistent evening dosing schedule for antihypertensives where they are implicated as a contributory factor to the development of IDH and when dialysis sessions are delivered during the day. Additionally, pharmacokinetic profiles need to be considered when determining dosing schedules as drugs that are readily dialysed by either haemodialysis and peritoneal dialysis may be unsuitable or may need to be administered after dialysis sessions rather than before.

Summary of audit measures

1. Proportion of adult dialysis patients having ABPM for diagnosis of *de novo* hypertension.
2. Proportion of adult dialysis patients using HBPM or standardized out-of-dialysis unit BP measurements to monitor treatment of hypertension.
3. Proportion of adult dialysis patients who have received advice and support to reduce salt intake to < 5 g/day.
4. Proportion of haemodialysis sessions where patients (adults and CYP) experience symptomatic IDH as defined UK Renal Association [164, 190].
5. Proportion of patients (adults and CYP) who have routine (e.g. minimum 3 monthly) assessment of dry weight.
6. Proportion of hypertensive adult haemodialysis patients prescribed β-blockers.
7. Proportion of hypertensive adult peritoneal dialysis patients prescribed ACEis or ARBs.
8. Proportion of CYP on dialysis undergoing annual ABPM assessment.

9. Proportion of hypertensive CYP with LVH.
10. Proportion of CYP on dialysis on antihypertensive medications and, in those taking antihypertensives, the number of agents used.

Summary of research recommendations

1. Pragmatic RCT comparing management of hypertension using routine (including 'usual' dialysis unit BP in in-centre haemodialysis patients) BP, HBPM and ABPM in dialysis patients.
2. Pragmatic RCT in both haemodialysis and peritoneal dialysis to determine optimal BP target ranges, preferably utilising standardised office BP or home BP monitoring as the primary method for measuring BP.
3. RCT of multiple lifestyle interventions (including dietary salt and fluid reduction, exercise and psychosocial support for medication adherence) on BP and symptomatic IDH in people on dialysis.
4. RCT of protocolised fluid volume management to improve CV outcomes in people receiving haemodialysis.
5. RCT to determine whether routine evening/night-time dosing of antihypertensive medication reduces incidence of symptomatic intra-dialytic hypotension.
6. RCT to determine effectiveness of midodrine and/or L-carnitine in preventing symptomatic IDH and reducing hospitalizations, and whether these treatments are well tolerated and safe.
7. RCT in CYP on dialysis to determine whether those in whom BP is targeted to < 90th centile have a lower risk of LVH compared with those in whom BP is permitted ≥ 90th centile.
8. Study in CYP on dialysis to determine whether HBPM values correlate with data from ABPM assessment.

Abbreviations

ABPM	Ambulatory blood pressure monitoring
ACEi	Angiotensin converting enzyme inhibitors
ARB	Angiotensin receptor blocker
BIS	Bioimpedance spectroscopy
βB	Beta blocker
BP	Blood pressure
CCB	Calcium channel blocker
CI	Confidence interval
CKD	Chronic kidney disease
CV	Cardiovascular
CYP	Children and young people
DASH	Dietary Approaches to Stop Hypertension
DBP	Diastolic blood pressure
DOPPS	Dialysis Observation and Practice Pattern Study
ESKD	End stage kidney disease
FHN	Frequent Hemodialysis Network
GRADE	Grading of Recommendations Assessment, Development and Evaluation
HBPM	home blood pressure monitoring
HD	Haemodialysis
HDF	Haemodiafiltration
IDH	Intradialytic hypotension
KDOQI	Kidney Disease Outcomes Quality Initiative
LVH	Left ventricular hypertrophy
LVM	Left ventricular mass
MAP	Mean arterial pressure
NT-proBNP	N-terminal-pro hormone BNP
MRA	Mineralocorticoid receptor antagonist
PCCRT	Paediatric Continuous Renal Replacement Therapy
PD	Peritoneal dialysis
PICO	Patient, intervention, comparison, outcome
RAAS	Renin-angiotensin-aldosterone system
RCT	Randomised controlled trial
RR	Risk reduction ratio
SBP	Systolic blood pressure

Supplementary Information

The online version contains supplementary material available at https://doi.org/10.1186/s12882-025-04451-2.

Supplementary Material 1

Supplementary Material 2

Acknowledgements

The authors would like to thank Mr Mark Kerr, Clinical Librarian, East Kent Hospitals University NHS Foundation Trust, for his considerable support in the design, refinement and execution of the search strategies.

Author contributions

All authors contributed to the development of the guidelines, contributed to writing the manuscript, read and approved the final manuscript.

Funding

Not applicable.

Data availability

No datasets were generated or analysed during the current study.

Declarations

Ethics approval and consent to participate

Not applicable.

Consent for publication

All authors have provided consent for publication.

Competing interests

The authors declare no competing interests.

Author details

[1]East Kent Hospitals University NHS Foundation Trust, Ashford, UK
[2]Liverpool University Hospitals NHS Foundation Trust, Liverpool, UK
[3]Barts Health NHS Trust, London, UK
[4]Queen Mary University of London, London, UK
[5]University Hospital Lewisham, London, UK
[6]Nuffield Department of Primary Care Health Sciences, University of Oxford, Oxford, UK
[7]Haemodialysis Patient & Lay Representative, Northampton, UK
[8]East and North Hertfordshire NHS Trust, Stevenage, UK
[9]Evelina London Children's Hospital, Guy's & St Thomas's Foundation Hospitals NHS Trust, London, UK
[10]British Heart Foundation Centre, King's College London, London, UK
[11]NHS Greater Glasgow and Clyde, Glasgow, UK
[12]Epsom and St Helier University Hospitals NHS Trust, London, UK
[13]Heartlands Hospital, Birmingham, UK
[14]Warwick Medical School, University of Warwick, Warwick, UK
[15]School of Health Sciences, University of Birmingham, Birmingham, United Kingdom

Received: 19 June 2025 / Accepted: 2 September 2025
Published online: 26 September 2025

References

1. Ashby D, Borman N, Burton J, Corbett R, Davenport A, Farrington K, et al. Renal association clinical practice guideline on haemodialysis. BMC Nephrol. 2019;20(1):379.
2. Registry UR. UK Renal Registry 25th Annual Report – data to 31/12/2021, Bristol, UK2023. Available from: https://ukkidney.org/audit-research/annual-report
3. Mitsnefes MM. Cardiovascular disease in children with chronic kidney disease. J Am Soc Nephrol. 2012;23(4):578–85.
4. Agarwal R. Blood pressure and mortality among Hemodialysis patients. Hypertension. 2010;55(3):762–8.
5. Bansal N, McCulloch CE, Lin F, Alper A, Anderson AH, Cuevas M, et al. Blood pressure and risk of cardiovascular events in patients on chronic hemodialysis: the CRIC study (Chronic renal insufficiency Cohort). Hypertension. 2017;70(2):435–43.
6. Flythe JE, Chang TI, Gallagher MP, Lindley E, Madero M, Sarafidis PA, et al. Blood pressure and volume management in dialysis: conclusions from a kidney disease: improving global outcomes (KDIGO) controversies conference. Kidney Int. 2020;97(5):861–76.
7. Kramer A, Stel VS, Tizard J, Verrina E, Rönnholm K, Pálsson R, et al. Characteristics and survival of young adults who started renal replacement therapy during childhood. Nephrol Dial Transpl. 2009;24(3):926–33.
8. Merrill K, Galbiati S, Mitsnefes M, Investigators N. Left ventricular hypertrophy in pediatric patients on maintenance dialysis: a report of the North American pediatric renal transplant cooperative study (NAPRTCS). Pediatr Nephrol. 2023;38(6):1925–33.
9. Liberati A, Altman DG, Tetzlaff J, Mulrow C, Gøtzsche PC, Ioannidis JP, et al. The PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate healthcare interventions: explanation and elaboration. BMJ. 2009;339:b2700.
10. Fagugli RM, Ricciardi D, Rossi D, De Gaetano A, Taglioni C. Blood pressure assessment in haemodialysis patients: comparison between pre-dialysis blood pressure and ambulatory blood pressure measurement. Nephrol (Carlton). 2009;14(3):283–90.
11. Alborzi P, Patel N, Agarwal R. Home blood pressures are of greater prognostic value than Hemodialysis unit recordings. Clin J Am Soc Nephrol. 2007;2(6):1228–34.
12. Agarwal R, Andersen MJ, Bishu K, Saha C. Home blood pressure monitoring improves the diagnosis of hypertension in Hemodialysis patients. Kidney Int. 2006;69(5):900–6.
13. Agarwal R, Peixoto AJ, Santos SF, Zoccali C. Pre- and postdialysis blood pressures are imprecise estimates of interdialytic ambulatory blood pressure. Clin J Am Soc Nephrol. 2006;1(3):389–98.
14. Bansal N, McCulloch CE, Rahman M, Kusek JW, Anderson AH, Xie D, et al. Blood pressure and risk of all-cause mortality in advanced chronic kidney disease and hemodialysis: the chronic renal insufficiency cohort study. Hypertension. 2015;65(1):93–100.
15. Bansal N, Glidden DV, Mehrotra R, Townsend RR, Cohen J, Linke L, et al. Treating home versus predialysis blood pressure among In-Center Hemodialysis patients: A pilot randomized trial. Am J Kidney Dis. 2021;77(1):12–22.
16. Agarwal R, Satyan S, Alborzi P, Light RP, Tegegne GG, Mazengia HS, et al. Home blood pressure measurements for managing hypertension in Hemodialysis patients. Am J Nephrol. 2009;30(2):126–34.
17. da Silva GV, de Barros S, Abensur H, Ortega KC, Mion D Jr., Cochrane Renal Group Prospective Trial Register CRG. Home blood pressure monitoring in blood pressure control among haemodialysis patients: an open randomized clinical trial. Nephrol Dial Transpl. 2009;24(12):3805–11.
18. Agarwal R, Flynn J, Pogue V, Rahman M, Reisin E, Weir MR. Assessment and management of hypertension in patients on Dialysis. J Am Soc Nephrol. 2014;25(8):1630–46.
19. Cohen DL, Huan Y, Townsend RR. Home blood pressure monitoring in CKD. Am J Kidney Dis. 2014;63(5):835–42.
20. Rahman M, Griffin V, Kumar A, Manzoor F, Wright JT Jr., Smith MC. A comparison of standardized versus usual blood pressure measurements in Hemodialysis patients. Am J Kidney Dis. 2002;39(6):1226–30.
21. Pappaccogli M, Van der Labriola L, Pruijm M, Vogt L, Halimi JM, et al. Is blood pressure measured correctly in Dialysis centres? Physicians' and patients' views. Nephrol Dial Transpl. 2019;34(9):1612–5.
22. Burton JO, Jefferies HJ, Selby NM, McIntyre CW. Hemodialysis-induced cardiac injury: determinants and associated outcomes. Clin J Am Soc Nephrol. 2009;4(5):914–20.
23. Chang TI, Paik J, Greene T, Desai M, Bech F, Cheung AK, et al. Intradialytic hypotension and vascular access thrombosis. J Am Soc Nephrol. 2011;22(8):1526–33.
24. Flythe JE, Xue H, Lynch KE, Curhan GC, Brunelli SM. Association of mortality risk with various definitions of intradialytic hypotension. J Am Soc Nephrol. 2015;26(3):724–34.
25. Miskulin DC, Gassman J, Schrader R, Gul A, Jhamb M, Ploth DW, et al. BP in dialysis: results of a pilot study. J Am Soc Nephrol. 2018;29(1):307–16.
26. Gul A, Miskulin D, Gassman J, Harford A, Horowitz B, Chen J, et al. Design of the blood pressure goals in Dialysis pilot study. Am J Med Sci. 2014;347(2):125–30.
27. Robinson BM, Tong L, Zhang J, Wolfe RA, Goodkin DA, Greenwood RN, et al. Blood pressure levels and mortality risk among Hemodialysis patients in the Dialysis outcomes and practice patterns study. Kidney Int. 2012;82(5):570–80.
28. Hannedouche T, Roth H, Krummel T, London GM, Jean G, Bouchet JL, et al. Multiphasic effects of blood pressure on survival in Hemodialysis patients. Kidney Int. 2016;90(3):674–84.
29. Hara M, Tanaka S, Taniguchi M, Fujisaki K, Torisu K, Masutani K, et al. Prognostic value of pre-dialysis blood pressure and risk threshold on clinical outcomes in Hemodialysis patients: the Q-Cohort study. Medicine. 2018;97(51):e13485.
30. Jhee JH, Park J, Kim H, Kee YK, Park JT, Han SH, et al. The optimal blood pressure target in different Dialysis populations. Sci Rep. 2018;8(1):14123.
31. Myers OB, Adams C, Rohrscheib MR, Servilla KS, Miskulin D, Bedrick EJ, et al. Age, race, diabetes, blood pressure, and mortality among Hemodialysis patients. J Am Soc Nephrol. 2010;21(11):1970–8.
32. Mayer CC, Matschkal J, Sarafidis PA, Hagmair S, Lorenz G, Angermann S, et al. Association of ambulatory blood pressure with All-Cause and cardiovascular mortality in Hemodialysis patients: effects of heart failure and atrial fibrillation. J Am Soc Nephrol. 2018;29(9):2409–17.
33. Shafi T, Zager PG, Sozio SM, Grams ME, Jaar BG, Christenson RH, et al. Troponin I and NT-proBNP and the association of systolic blood pressure with outcomes in incident Hemodialysis patients: the choices for healthy outcomes in caring for ESRD (CHOICE) study. Am J Kidney Dis. 2014;64(3):443–51.
34. De Lima JJG, Gowdak LHW, Reusing JO Jr, David-Neto E, Bortolotto LA. Interdialytic blood pressure and risk of cardiovascular events and death in Hemodialysis patients. High Blood Press Cardiovasc Prev. 2023;30(3):235–41.
35. Wang AY, Brimble KS, Brunier G, Holt SG, Jha V, Johnson DW, et al. ISPD cardiovascular and metabolic guidelines in adult peritoneal Dialysis patients part I - Assessment and management of various cardiovascular risk factors. Perit Dial Int. 2015;35(4):379–87.
36. Xie X, Lv D, Zheng H, Zhang X, Han F, Chen J. The associations of blood pressure parameters with all-cause and cardiovascular mortality in peritoneal Dialysis patients: a cohort study in China. J Hypertens. 2020;38(11):2252–60.
37. Udayaraj UP, Steenkamp R, Caskey FJ, Rogers C, Nitsch D, Ansell D, et al. Blood pressure and mortality risk on peritoneal Dialysis. Am J Kidney Dis. 2009;53(1):70–8.
38. Bansal N, Artinian NT, Bakris G, Chang T, Cohen J, Flythe J, et al. Hypertension in patients treated with In-Center maintenance hemodialysis: current evidence and future opportunities: A scientific statement from the American heart association. Hypertension. 2023;80(6):e112–22.
39. Argilés À, Lorho R, Servel M-F, Chong G, Kerr PG, Mourad G. Seasonal modifications in blood pressure are mainly related to interdialytic body weight gain in Dialysis patients. Kidney Int. 2004;65 5:1795–801.
40. Nongnuch A, Campbell N, Stern E, El-Kateb S, Fuentes L, Davenport A. Increased postdialysis systolic blood pressure is associated with extracellular overhydration in Hemodialysis outpatients. Kidney Int. 2015;87(2):452–7.
41. Mc Causland FR, Waikar SS, Brunelli SM. Increased dietary sodium is independently associated with greater mortality among prevalent Hemodialysis patients. Kidney Int. 2012;82(2):204–11.
42. He FJ, Li J, Macgregor GA. Effect of longer-term modest salt reduction on blood pressure. Cochrane Database Syst Rev. 2013;2013(4):Cd004937.
43. McMahon EJ, Campbell KL, Bauer JD, Mudge DW. Altered dietary salt intake for people with chronic kidney disease. Cochrane Database Syst Rev. 2015;(2):Cd010070.
44. Sacks FM, Svetkey LP, Vollmer WM, Appel LJ, Bray GA, Harsha D, et al. Effects on blood pressure of reduced dietary sodium and the dietary approaches to

stop hypertension (DASH) diet. DASH-Sodium collaborative research group. N Engl J Med. 2001;344(1):3–10.
45. Suckling RJ, He FJ, Macgregor GA. Altered dietary salt intake for preventing and treating diabetic kidney disease. Cochrane Database Syst Rev. 2010;(12):Cd006763.
46. Swift PA, Markandu ND, Sagnella GA, He FJ, MacGregor GA. Modest salt reduction reduces blood pressure and urine protein excretion in black hypertensives: a randomized control trial. Hypertension. 2005;46(2):308–12.
47. Chazot C, Charra B, Laurent G, Didier C, Vo Van C, Terrat JC, et al. Interdialysis blood pressure control by long haemodialysis sessions. Nephrol Dial Transpl. 1995;10(6):831–7.
48. Krautzig S, Janssen U, Koch KM, Granolleras C, Shaldon S. Dietary salt restriction and reduction of dialysate sodium to control hypertension in maintenance haemodialysis patients. Nephrol Dial Transpl. 1998;13(3):552–3.
49. Ozkahya M, Ok E, Cirit M, Aydin S, Akçiçek F, Başçi A, et al. Regression of left ventricular hypertrophy in haemodialysis patients by ultrafiltration and reduced salt intake without antihypertensive drugs. Nephrol Dial Transpl. 1998;13(6):1489–93.
50. Cole NI, Swift PA, He FJ, MacGregor GA, Suckling RJ. The effect of dietary salt on blood pressure in individuals receiving chronic dialysis: a systematic review and meta-analysis of randomised controlled trials. J Hum Hypertens. 2019;33(4):319–26.
51. McMahon EJ, Campbell KL, Bauer JD, Mudge DW, Kelly JT. Altered dietary salt intake for people with chronic kidney disease. Cochrane Database Syst Rev. 2021;6(6):Cd010070.
52. Agarwal R. Hypervolemia is associated with increased mortality among Hemodialysis patients. Hypertension. 2010;56(3):512–7.
53. Tomson CR. Advising Dialysis patients to restrict fluid intake without restricting sodium intake is not based on evidence and is a waste of time. Nephrol Dial Transpl. 2001;16(8):1538–42.
54. Conlin PR. The dietary approaches to stop hypertension (DASH) clinical trial: implications for lifestyle modifications in the treatment of hypertensive patients. Cardiol Rev. 1999;7(5):284–8.
55. Garagarza C, Valente A, Caetano C, Ramos I, Sebastião J, Pinto M et al. Potassium intake-(un)expected non-predictor of higher serum potassium levels in hemodialysis DASH diet consumers. Nutrients. 2022;14(10).
56. Marx W, Kelly J, Marshall S, Nakos S, Campbell K, Itsiopoulos C. The effect of polyphenol-rich interventions on cardiovascular risk factors in haemodialysis: A systematic review and Meta-Analysis. Nutrients. 2017;9(12).
57. Barati Boldaji R, Akhlaghi M, Sagheb MM, Esmaeilinezhad Z. Pomegranate juice improves cardiometabolic risk factors, biomarkers of oxidative stress and inflammation in Hemodialysis patients: a randomized crossover trial. J Sci Food Agric. 2020;100(2):846–54.
58. Pu J, Jiang Z, Wu W, Li L, Zhang L, Li Y, et al. Efficacy and safety of intradialytic exercise in haemodialysis patients: a systematic review and meta-analysis. BMJ Open. 2019;9(1):e020633.
59. Ferrari F, Helal L, Dipp T, Soares D, Soldatelli Â, Mills AL, et al. Intradialytic training in patients with end-stage renal disease: a systematic review and meta-analysis of randomized clinical trials assessing the effects of five different training interventions. J Nephrol. 2020;33(2):251–66.
60. Scapini KB, Bohlke M, Moraes OA, Rodrigues CG, Inácio JF, Sbruzzi G, et al. Combined training is the most effective training modality to improve aerobic capacity and blood pressure control in people requiring haemodialysis for end-stage renal disease: systematic review and network meta-analysis. J Physiother. 2019;65(1):4–15.
61. Song Y, Chen L, Wang M, He Q, Xue J, Jiang H. The optimal exercise modality and intensity for Hemodialysis patients incorporating bayesian network meta-analysis and systematic review. Front Physiol. 2022;13:945465.
62. Bernier-Jean A, Beruni NA, Bondonno NP, Williams G, Teixeira-Pinto A, Craig JC, et al. Exercise training for adults undergoing maintenance Dialysis. Cochrane Database Syst Rev. 2022;1(1):Cd014653.
63. Kauric-Klein Z, Peters RM, Yarandi HN. Self-Efficacy and blood pressure Self-Care behaviors in patients on chronic Hemodialysis. West J Nurs Res. 2017;39(7):886–905.
64. Daud R, Zulkarnain BS, Amu IV. Providing counseling through home pharmacy care (HPC) for Hemodialysis patients with hypertension in Lowering blood pressure. J Basic Clin Physiol Pharmacol. 2021;32(4):459–65.
65. Culleton BF, Walsh M, Klarenbach SW, Mortis G, Scott-Douglas N, Quinn RR, et al. Effect of frequent nocturnal Hemodialysis vs conventional Hemodialysis on left ventricular mass and quality of life: a randomized controlled trial. JAMA. 2007;298(11):1291–9.
66. Fagugli RM, Reboldi G, Quintaliani G, Pasini P, Ciao G, Cicconi B, et al. Short daily Hemodialysis: blood pressure control and left ventricular mass reduction in hypertensive Hemodialysis patients. Am J Kidney Dis. 2001;38(2):371–6.
67. Jardine MJ, Zuo L, Gray NA, de Zoysa JR, Chan CT, Gallagher MP, et al. A trial of extending Hemodialysis hours and quality of life. J Am Soc Nephrol. 2017;28(6):1898–911.
68. Zimmerman DL, Ruzicka M, Hebert P, Fergusson D, Touyz RM, Burns KD. Short daily versus conventional Hemodialysis for hypertensive patients: a randomized cross-over study. PLoS ONE. 2014;9(5):e97135.
69. Chertow GM, Levin NW, Beck GJ, Daugirdas JT, Eggers PW, Kliger AS, et al. Long-Term effects of frequent In-Center Hemodialysis. J Am Soc Nephrol. 2016;27(6):1830–6.
70. Ok E, Duman S, Asci G, Tumuklu M, Onen Sertoz O, Kayikcioglu M, et al. Comparison of 4- and 8-h Dialysis sessions in thrice-weekly in-centre haemodialysis: a prospective, case-controlled study. Nephrol Dial Transpl. 2011;26(4):1287–96.
71. Kotanko P, Garg AX, Depner T, Pierratos A, Chan CT, Levin NW, et al. Effects of frequent Hemodialysis on blood pressure: results from the randomized frequent Hemodialysis network trials. Hemodial Int. 2015;19(3):386–401.
72. Rocco MV, Lockridge RS Jr., Beck GJ, Eggers PW, Gassman JJ, Greene T, et al. The effects of frequent nocturnal home hemodialysis: the frequent Hemodialysis network nocturnal trial. Kidney Int. 2011;80(10):1080–91.
73. Hull KL, Bramham K, Brookes CL, Cluley V, Conefrey C, Cooper NJ, et al. The nightlife study - the clinical and cost-effectiveness of thrice-weekly, extended, in-centre nocturnal haemodialysis versus daytime haemodialysis using a mixed methods approach: study protocol for a randomised controlled trial. Trials. 2023;24(1):522.
74. Lin CL, Huang CC, Chang CT, Wu MS, Hung CC, Chien CC, et al. Clinical improvement by increased frequency of on-line hemodialfiltration. Ren Fail. 2001;23(2):193–206.
75. Maduell F, Moreso F, Pons M, Ramos R, Mora-Macia J, Carreras J, et al. High-efficiency postdilution online hemodiafiltration reduces all-cause mortality in Hemodialysis patients. J Am Soc Nephrol. 2013;24(3):487–97.
76. Morena M, Jaussent A, Chalabi L, Leray-Moragues H, Chenine L, Debure A, et al. Treatment tolerance and patient-reported outcomes favor online hemodiafiltration compared to high-flux Hemodialysis in the elderly. Kidney Int. 2017;91(6):1495–509.
77. Nistor I, Palmer SC, Craig JC, Saglimbene V, Vecchio M, Covic A et al. Haemodiafiltration, haemofiltration and haemodialysis for end-stage kidney disease. Cochrane Database Syst Rev. 2015;(5):CD006258.
78. Wang AY, Ninomiya T, Al-Kahwa A, Perkovic V, Gallagher MP, Hawley C, et al. Effect of hemodiafiltration or hemofiltration compared with Hemodialysis on mortality and cardiovascular disease in chronic kidney failure: a systematic review and meta-analysis of randomized trials. Am J Kidney Dis. 2014;63(6):968–78.
79. Blankestijn PJ, Vernooij RWM, Hockham C, Strippoli GFM, Canaud B, Hegbrant J, et al. Effect of hemodiafiltration or Hemodialysis on mortality in kidney failure. N Engl J Med. 2023;389(8):700–9.
80. Caskey FJ, Procter S, MacNeill SJ, Wade J, Taylor J, Rooshenas L, et al. The high-volume haemodiafiltration vs high-flux haemodialysis registry trial (H4RT): a multi-centre, unblinded, randomised, parallel-group, superiority study to compare the effectiveness and cost-effectiveness of high-volume haemodiafiltration and high-flux haemodialysis in people with kidney failure on maintenance Dialysis using linkage to routine healthcare databases for outcomes. Trials. 2022;23(1):532.
81. Stefansson BV, Brunelli SM, Cabrera C, Rosenbaum D, Anum E, Ramakrishnan K, et al. Intradialytic hypotension and risk of cardiovascular disease. Clin J Am Soc Nephrol. 2014;9(12):2124–32.
82. Yu J, Liu Z, Shen B, Teng J, Zou J, Ding X. Intradialytic hypotension as an independent risk factor for Long-Term mortality in maintaining Hemodialysis patients: A 5-Year Follow-Up cohort study. Blood Purif. 2018;45(4):320–6.
83. Zoccali C, Tripepi G, Neri L, Savoia M, Baro Salvador ME, Ponce P, et al. Effectiveness of cold HD for the prevention of HD hypotension and mortality in the general HD population. Nephrol Dial Transpl. 2023;38(7):1700–6.
84. Mustafa RA, Bdair F, Akl EA, Garg AX, Thiessen-Philbrook H, Salameh H, et al. Effect of Lowering the dialysate temperature in chronic hemodialysis: A systematic review and Meta-Analysis. Clin J Am Soc Nephrol. 2016;11(3):442–57.
85. Odudu A, Eldehni MT, McCann GP, McIntyre CW. Randomized controlled trial of individualized dialysate cooling for cardiac protection in Hemodialysis patients. Clin J Am Soc Nephrol. 2015;10(8):1408–17.

86. My_Temp. Personalised cooler dialysate for patients receiving maintenance haemodialysis (MyTEMP): a pragmatic, cluster-randomised trial. Lancet. 2022;400(10364):1693–703.
87. Basile C, Pisano A, Lisi P, Rossi L, Lomonte C, Bolignano D. High versus low dialysate sodium concentration in chronic haemodialysis patients: a systematic review of 23 studies. Nephrol Dial Transpl. 2016;31(4):548–63.
88. Dunlop JL, Vandal AC, Marshall MR. Low dialysate sodium levels for chronic haemodialysis. Cochrane Database Syst Rev. 2019;1(1):Cd011204.
89. Akdag S, Akyol A, Cakmak HA, Tosu AR, Asker M, Yaman M, et al. The effect of low-sodium dialysate on ambulatory blood pressure measurement parameters in patients undergoing Hemodialysis. Ther Clin Risk Manag. 2015;11:1829–35.
90. Causland FRM, Ravi KS, Curtis KA, Kibbelaar ZA, Short SAP, Singh AT, et al. A randomized controlled trial of two dialysate sodium concentrations in hospitalized Hemodialysis patients. Nephrol Dial Transpl. 2022;37(7):1340–7.
91. Inrig JK, Molina C, D'Silva K, Kim C, Van Buren P, Allen JD, et al. Effect of low versus high dialysate sodium concentration on blood pressure and endothelial-derived vasoregulators during hemodialysis: a randomized crossover study. Am J Kidney Dis. 2015;65(3):464–73.
92. Davenport A. Audit of the effect of dialysate sodium concentration on interdialytic weight gains and blood pressure control in chronic haemodialysis patients. Nephron Clin Pract. 2006;104(3):c120–5.
93. Dunne N. A meta-analysis of sodium profiling techniques and the impact on intradialytic hypotension. Hemodial Int. 2017;21(3):312–22.
94. Ebrahimi H, Safavi M, Saeidi MH, Emamian MH. Effects of sodium concentration and dialysate temperature changes on blood pressure in Hemodialysis patients: A randomized, Triple-Blind crossover clinical trial. Ther Apher Dial. 2017;21(2):117–25.
95. Dasgupta I, Thomas GN, Clarke J, Sitch A, Martin J, Bieber B, et al. Associations between Hemodialysis facility practices to manage fluid volume and intradialytic hypotension and patient outcomes. Clin J Am Soc Nephrol. 2019;14(3):385–93.
96. Tentori F, Karaboyas A, Robinson BM, Morgenstern H, Zhang J, Sen A, et al. Association of dialysate bicarbonate concentration with mortality in the Dialysis outcomes and practice patterns study (DOPPS). Am J Kidney Dis. 2013;62(4):738–46.
97. Viegas M, Candido C, Felgueiras J, Clemente J, Barros S, Farbota R, et al. Dialysate bicarbonate variation in maintenance hemodiafiltration patients: impact on serum bicarbonate, intradialytic hypotension and interdialytic weight gain. Hemodial Int. 2017;21(3):385–92.
98. Gabutti L, Bianchi G, Soldini D, Marone C, Burnier M. Haemodynamic consequences of changing bicarbonate and calcium concentrations in haemodialysis fluids. Nephrol Dial Transpl. 2009;24(3):973–81.
99. Gabutti L, Ferrari N, Giudici G, Mombelli G, Marone C. Unexpected haemodynamic instability associated with standard bicarbonate haemodialysis. Nephrol Dial Transpl. 2003;18(11):2369–76.
100. Kyriazis J, Kalogeropoulou K, Bilirakis L, Smirnioudis N, Pikounis V, Stamatiadis D, et al. Dialysate magnesium level and blood pressure. Kidney Int. 2004;66(3):1221–31.
101. Del Giorno R, Lavorato Hadjeres S, Stefanelli K, Allegra G, Zapparoli C, Predrag L, et al. Consequences of Supraphysiological dialysate magnesium on arterial stiffness, hemodynamic profile, and endothelial function in hemodialysis: A randomized crossover study followed by a Non-Controlled Follow-Up phase. Adv Ther. 2020;37(12):4848–65.
102. Woodrow G, Oldroyd B, Stables G, Gibson J, Turney JH, Brownjohn AM. Effects of icodextrin in automated peritoneal Dialysis on blood pressure and bioelectrical impedance analysis. Nephrol Dial Transpl. 2000;15(6):862–6.
103. Davies SJ, Woodrow G, Donovan K, Plum J, Williams P, Johansson AC, et al. Icodextrin improves the fluid status of peritoneal Dialysis patients: results of a double-blind randomized controlled trial. J Am Soc Nephrol. 2003;14(9):2338–44.
104. Flythe JE, Kshirsagar AV, Falk RJ, Brunelli SM. Associations of posthemodialysis weights above and below target weight with All-Cause and cardiovascular mortality. Clin J Am Soc Nephrol. 2015;10(5):808–16.
105. Dekker M, Konings C, Canaud B, Carioni P, Guinsburg A, Madero M, et al. Pre-dialysis fluid status, pre-dialysis systolic blood pressure and outcome in prevalent haemodialysis patients: results of an international cohort study on behalf of the MONDO initiative. Nephrol Dial Transpl. 2018;33(11):2027–34.
106. Zoccali C, Moissl U, Chazot C, Mallamaci F, Tripepi G, Arkossy O, et al. Chronic fluid overload and mortality in ESRD. J Am Soc Nephrol. 2017;28(8):2491–7.
107. Onofriescu M, Siriopol D, Voroneanu L, Hogas S, Nistor I, Apetrii M, et al. Overhydration, cardiac function and survival in Hemodialysis patients. PLoS ONE. 2015;10(8):e0135691.
108. Hecking M, Moissl U, Genser B, Rayner H, Dasgupta I, Stuard S, et al. Greater fluid overload and lower interdialytic weight gain are independently associated with mortality in a large international Hemodialysis population. Nephrol Dial Transpl. 2018;33(10):1832–42.
109. Agarwal R, Alborzi P, Satyan S, Light RP. Dry-weight reduction in hypertensive Hemodialysis patients (DRIP): a randomized, controlled trial. Hypertension. 2009;53(3):500–7.
110. Agarwal R. Treating hypertension in Hemodialysis improves symptoms seemingly unrelated to volume excess. Nephrol Dial Transpl. 2016;31(1):142–9.
111. Davies SJ, Coyle D, Lindley EJ, Keane D, Belcher J, Caskey FJ, et al. Bio-impedance spectroscopy added to a fluid management protocol does not improve preservation of residual kidney function in incident Hemodialysis patients in a randomized controlled trial. Kidney Int. 2023;104(3):587–98.
112. Barth C, Boer W, Garzoni D, Kuenzi T, Ries W, Schaefer R, et al. Characteristics of hypotension-prone haemodialysis patients: is there a critical relative blood volume? Nephrol Dial Transpl. 2003;18(7):1353–60.
113. Castellano S, Palomares I, Molina M, Pérez-García R, Aljama P, Ramos R, et al. Clinical, analytical and bioimpedance characteristics of persistently overhydrated haemodialysis patients. Nefrologia. 2014;34(6):716–23.
114. Chang ST, Chen CL, Chen CC, Hung KC. Clinical events occurrence and the changes of quality of life in chronic haemodialysis patients with dry weight determined by echocardiographic method. Int J Clin Pract. 2004;58(12):1101–7.
115. Huan-Sheng C, Yeong-Chang C, Ming-Hsing H, Fan-Lieh T, Chu-Cheng L, Tsai-Kun W, et al. Application of bioimpedance spectroscopy in Asian Dialysis patients (ABISAD-III): a randomized controlled trial for clinical outcomes. Int Urol Nephrol. 2016;48(11):1897–909.
116. Hur E, Usta M, Toz H, Asci G, Wabel P, Kahvecioglu S, et al. Effect of fluid management guided by bioimpedance spectroscopy on cardiovascular parameters in Hemodialysis patients: a randomized controlled trial. Am J Kidney Dis. 2013;61(6):957–65.
117. Kayatas M, Ozdemir N, Muderrisoglu H, Ulucam M, Turan M, Hizel N. Comparison of the non-invasive methods estimating dry weight in Hemodialysis patients. Ren Fail. 2006;28(3):217–22.
118. Leung KCW, Quinn RR, Ravani P, Duff H, MacRae JM. Randomized crossover trial of blood volume Monitoring-Guided ultrafiltration biofeedback to reduce intradialytic hypotensive episodes with Hemodialysis. Clin J Am Soc Nephrol. 2017;12(11):1831–40.
119. Luo YJ, Lu XH, Woods F, Wang T. Volume control in peritoneal Dialysis patients guided by bioimpedance spectroscopy assessment. Blood Purif. 2011;31(4):296–302.
120. McIntyre CW, Lambie SH, Fluck RJ. Biofeedback controlled Hemodialysis (BF-HD) reduces symptoms and increases both hemodynamic tolerability and Dialysis adequacy in non-hypotension prone stable patients. Clin Nephrol. 2003;60(2):105–12.
121. Nesrallah GE, Suri RS, Thiessen-Philbrook H, Heidenheim P, Lindsay RM. Can extracellular fluid volume expansion in Hemodialysis patients be safely reduced using the hemocontrol biofeedback algorithm? A randomized trial. ASAIO J. 2008;54(3):270–4.
122. Onofriescu M, Mardare NG, Segall L, Voroneanu L, Cusai C, Hogas S, et al. Randomized trial of bioelectrical impedance analysis versus clinical criteria for guiding ultrafiltration in Hemodialysis patients: effects on blood pressure, hydration status, and arterial stiffness. Int Urol Nephrol. 2012;44(2):583–91.
123. Patel HV, Annigeri RA, Kowdle PC, Rao BS, Seshadri R, Balasubramanian S, et al. Bioimpedance Spectroscopy-Guided ultrafiltration normalizes hydration and reduces intradialytic adverse events in Hemodialysis patients. Indian J Nephrol. 2019;29(1):1–7.
124. Raza SH, Hashmi MN, Elairon P, Riolo AH, Hejaili F, Al-Sayyari AA. Improvement in dialysis-related adverse events with use of body composition monitoring. Saudi J Kidney Dis Transpl. 2018;29(3):518–23.
125. Santoro A, Mancini E, Basile C, Amoroso L, Di Giulio S, Usberti M, et al. Blood volume controlled Hemodialysis in hypotension-prone patients: a randomized, multicenter controlled trial. Kidney Int. 2002;62(3):1034–45.
126. Selby NM, Lambie SH, Camici PG, Baker CS, McIntyre CW. Occurrence of regional left ventricular dysfunction in patients undergoing standard and biofeedback Dialysis. Am J Kidney Dis. 2006;47(5):830–41.
127. Siriopol D, Onofriescu M, Voroneanu L, Apetrii M, Nistor I, Hogas S, et al. Dry weight assessment by combined ultrasound and bioimpedance monitoring

in low cardiovascular risk Hemodialysis patients: a randomized controlled trial. Int Urol Nephrol. 2017;49(1):143–53.
128. Yoon HE, Kwon YJ, Shin SJ, Lee SY, Lee S, Kim SH, et al. Bioimpedance spectroscopy-guided fluid management in peritoneal Dialysis patients with residual kidney function: A randomized controlled trial. Nephrol (Carlton). 2019;24(12):1279–89.
129. Tian N, Yang X, Guo Q, Zhou Q, Yi C, Lin J, et al. Bioimpedance guided fluid management in peritoneal dialysis: A randomized controlled trial. Clin J Am Soc Nephrol. 2020;15(5):685–94.
130. Tangvoraphonkchai K, Davenport A. Changes in extracellular water with Hemodialysis and fall in systolic blood pressure. Int J Artif Organs. 2022;45(2):140–5.
131. Zoccali C, Torino C, Mallamaci F, Sarafidis P, Papagianni A, Ekart R, et al. A randomized multicenter trial on a lung ultrasound-guided treatment strategy in patients on chronic Hemodialysis with high cardiovascular risk. Kidney Int. 2021;100(6):1325–33.
132. Loutradis C, Sarafidis PA, Ekart R, Papadopoulos C, Sachpekidis V, Alexandrou ME, et al. The effect of dry-weight reduction guided by lung ultrasound on ambulatory blood pressure in Hemodialysis patients: a randomized controlled trial. Kidney Int. 2019;95(6):1505–13.
133. Heerspink HJ, Ninomiya T, Zoungas S, de Zeeuw D, Grobbee DE, Jardine MJ, et al. Effect of Lowering blood pressure on cardiovascular events and mortality in patients on dialysis: a systematic review and meta-analysis of randomised controlled trials. Lancet. 2009;373(9668):1009–15.
134. Agarwal R, Sinha AD. Cardiovascular protection with antihypertensive drugs in Dialysis patients: systematic review and meta-analysis. Hypertension. 2009;53(5):860–6.
135. Agarwal R, Sinha AD, Pappas MK, Abraham TN, Tegegne GG. Hypertension in Hemodialysis patients treated with Atenolol or lisinopril: a randomized controlled trial. Nephrol Dial Transpl. 2014;29(3):672–81.
136. Iseki K, Arima H, Kohagura K, Komiya I, Ueda S, Tokuyama K, et al. Effects of angiotensin receptor Blockade (ARB) on mortality and cardiovascular outcomes in patients with long-term haemodialysis: a randomized controlled trial. Nephrol Dial Transpl. 2013;28(6):1579–89.
137. Ruggenenti P, Podestà MA, Trillini M, Perna A, Peracchi T, Rubis N, et al. Ramipril and cardiovascular outcomes in patients on maintenance hemodialysis: the ARCADIA multicenter randomized controlled trial. Clin J Am Soc Nephrol. 2021;16(4):575–87.
138. Shaman AM, Smyth B, Arnott C, Palmer SC, Mihailidou AS, Jardine MJ, et al. Comparative efficacy and safety of BP-Lowering pharmacotherapy in patients undergoing maintenance dialysis: A network Meta-Analysis of randomized, controlled trials. Clin J Am Soc Nephrol. 2020;15(8):1129–38.
139. Li PK, Chow KM, Wong TY, Leung CB, Szeto CC. Effects of an angiotensin-converting enzyme inhibitor on residual renal function in patients receiving peritoneal dialysis. A randomized, controlled study. Ann Intern Med. 2003;139(2):105–12.
140. Suzuki H, Kanno Y, Sugahara S, Okada H, Nakamoto H. Effects of an angiotensin II receptor blocker, valsartan, on residual renal function in patients on CAPD. Am J Kidney Dis. 2004;43(6):1056–64.
141. Liu Y, Ma X, Zheng J, Jia J, Yan T. Effects of angiotensin-converting enzyme inhibitors and angiotensin receptor blockers on cardiovascular events and residual renal function in Dialysis patients: a meta-analysis of randomised controlled trials. BMC Nephrol. 2017;18(1):206.
142. Zhang L, Zeng X, Fu P, Wu HM. Angiotensin-converting enzyme inhibitors and angiotensin receptor blockers for preserving residual kidney function in peritoneal Dialysis patients. Cochrane Database Syst Rev. 2014;2014(6):Cd009120.
143. Gou WJ, Zhou FW, Providencia R, Wang B, Zhang H, Hu SL, et al. Association of mineralocorticoid receptor antagonists with the mortality and cardiovascular effects in Dialysis patients: A Meta-analysis. Front Pharmacol. 2022;13:823530.
144. Liu J, Jia W, Yu C. Safety and efficacy of spironolactone in Dialysis-Dependent patients: Meta-Analysis of randomized controlled trials. Front Med (Lausanne). 2022;9:828189.
145. Quach K, Lvtvyn L, Baigent C, Bueti J, Garg AX, Hawley C, et al. The safety and efficacy of mineralocorticoid receptor antagonists in patients who require dialysis: A systematic review and Meta-analysis. Am J Kidney Dis. 2016;68(4):591–8.
146. Weir MA, Dixon SN, Fleet JL, Roberts MA, Hackam DG, Oliver MJ, et al. β-Blocker dialyzability and mortality in older patients receiving Hemodialysis. J Am Soc Nephrol. 2015;26(4):987–96.
147. Kuo PY, Saran R, Argentina M, Heung M, Bragg-Gresham J, Krein S, et al. Cramping, crashing, cannulating, and clotting: a qualitative study of patients' definitions of a bad run on Hemodialysis. BMC Nephrol. 2020;21(1):67.
148. Li H, Xue J, Dai W, Liao X, Zhu P, Zhou Q, et al. Blood pressure variability and outcomes in End-Stage renal disease patients on dialysis: A systematic review and Meta-Analysis. Kidney Blood Press Res. 2020;45(5):631–44.
149. Mackenzie IS, Rogers A, Poulter NR, Williams B, Brown MJ, Webb DJ, et al. Cardiovascular outcomes in adults with hypertension with evening versus morning dosing of usual antihypertensives in the UK (TIME study): a prospective, randomised, open-label, blinded-endpoint clinical trial. Lancet. 2022;400(10361):1417–25.
150. Prakash S, Garg AX, Heidenheim AP, House AA. Midodrine appears to be safe and effective for dialysis-induced hypotension: a systematic review. Nephrol Dial Transpl. 2004;19(10):2553–8.
151. Brunelli SM, Cohen DE, Marlowe G, Van Wyck D. The impact of midodrine on outcomes in patients with intradialytic hypotension. Am J Nephrol. 2018;48(5):381–8.
152. Chewcharat A, Chewcharat P, Liu W, Cellini J, Phipps EA, Melendez Young JA, et al. The effect of levocarnitine supplementation on dialysis-related hypotension: A systematic review, meta-analysis, and trial sequential analysis. PLoS ONE. 2022;17(7):e0271307.
153. Nishioka N, Luo Y, Taniguchi T, Ohnishi T, Kimachi M, Ng RC, et al. Carnitine supplements for people with chronic kidney disease requiring Dialysis. Cochrane Database Syst Rev. 2022;12(12):Cd013601.
154. Chavers BM, Solid CA, Daniels FX, Chen SC, Collins AJ, Frankenfield DL, et al. Hypertension in pediatric long-term Hemodialysis patients in the united States. Clin J Am Soc Nephrol. 2009;4(8):1363–9.
155. Kramer AM, van Stralen KJ, Jager KJ, Schaefer F, Verrina E, Seeman T, et al. Demographics of blood pressure and hypertension in children on renal replacement therapy in Europe. Kidney Int. 2011;80(10):1092–8.
156. Halbach SM, Martz K, Mattoo T, Flynn J. Predictors of blood pressure and its control in pediatric patients receiving dialysis. J Pediatr. 2012;160(4):621-5.e1.
157. Flynn JT, Kaelber DC, Baker-Smith CM, Blowey D, Carroll AE, Daniels SR et al. Clinical practice guideline for screening and management of high blood pressure in children and adolescents. Pediatrics. 2017;140(3).
158. Lurbe E, Agabiti-Rosei E, Cruickshank JK, Dominiczak A, Erdine S, Hirth A, et al. 2016 European society of hypertension guidelines for the management of high blood pressure in children and adolescents. J Hypertens. 2016;34(10):1887–920.
159. Bakkaloglu SA, Borzych D, Soo Ha I, Serdaroglu E, Büscher R, Salas P, et al. Cardiac geometry in children receiving chronic peritoneal dialysis: findings from the international pediatric peritoneal Dialysis network (IPPN) registry. Clin J Am Soc Nephrol. 2011;6(8):1926–33.
160. Ulinski T, Genty J, Viau C, Tillous-Borde I, Deschênes G. Reduction of left ventricular hypertrophy in children undergoing Hemodialysis. Pediatr Nephrol. 2006;21(8):1171–8.
161. Litwin M, Wühl E, Jourdan C, Trelewicz J, Niemirska A, Fahr K, et al. Altered morphologic properties of large arteries in children with chronic renal failure and after renal transplantation. J Am Soc Nephrol. 2005;16(5):1494–500.
162. KDIGO. 2021 clinical practice guideline for the management of blood pressure in chronic kidney disease. Kidney Int. 2021;99(3s):S1–87.
163. Sinha MD, Gu H, Douiri A, Cansick J, Finlay E, Gilbert R, et al. Intensive compared with less intensive blood pressure control to prevent adverse cardiac remodelling in children with chronic kidney disease (HOT-KID): a parallel-group, open-label, multicentre, randomised, controlled trial. Lancet Child Adolesc Health. 2023;7(1):26–36.
164. Raina R, Lam S, Raheja H, Krishnappa V, Hothi D, Davenport A, et al. Pediatric intradialytic hypotension: recommendations from the pediatric continuous renal replacement therapy (PCRRT) workgroup. Pediatr Nephrol. 2019;34(5):925–41.
165. Hothi DK, Rees L, Marek J, Burton J, McIntyre CW. Pediatric myocardial stunning underscores the cardiac toxicity of conventional Hemodialysis treatments. Clin J Am Soc Nephrol. 2009;4(4):790–7.
166. Parati G, Ochoa JE, Bilo G, Agarwal R, Covic A, Dekker FW, et al. Hypertension in chronic kidney disease part 1: Out-of-Office blood pressure monitoring: methods, thresholds, and patterns. Hypertension. 2016;67(6):1093–101.
167. Chaudhuri A, Sutherland SM, Begin B, Salsbery K, McCabe L, Potter D, et al. Role of twenty-four-hour ambulatory blood pressure monitoring in children on Dialysis. Clin J Am Soc Nephrol. 2011;6(4):870–6.
168. Ozçakar ZB, Yalçinkaya F, Tutar E, Cakar N, Uçar T, Elhan A, et al. Hypertension and left ventricular hypertrophy in pediatric peritoneal Dialysis patients:

ambulatory blood pressure monitoring and echocardiographic evaluation. Nephron Clin Pract. 2006;104(2):c101–6.
169. Roszkowska-Blaim M, Skrzypczyk P, Jander A, Tkaczyk M, Bałasz-Chmielewska I, Żurowska A, et al. Effect of hypertension and antihypertensive medications on residual renal function in children treated with chronic peritoneal Dialysis. Adv Med Sci. 2015;60(1):18–24.
170. Mitsnefes M, Stablein D. Hypertension in pediatric patients on long-term dialysis: a report of the North American pediatric renal transplant cooperative study (NAPRTCS). Am J Kidney Dis. 2005;45(2):309–15.
171. Ateya AM, El Hakim I, Shahin SM, El Borolossy R, Kreutz R, Sabri NA. Effects of Ramipril on biomarkers of endothelial dysfunction and inflammation in hypertensive children on maintenance hemodialysis: the SEARCH randomized Placebo-Controlled trial. Hypertension. 2022;79(8):1856–65.
172. Srisuwan K, Hongsawong N, Lumpaopong A, Thirakhupt P, Chulamokha Y. Blood volume monitoring to assess dry weight in pediatric chronic Hemodialysis patients. J Med Assoc Thai. 2015;98(11):1089–96.
173. Fadel FI, Makar SH, Eskander AE, Aon AH. Decreasing intra-dialytic morbid events and assessment of dry weight in children on chronic Hemodialysis using non-invasive changes in hematocrit. Saudi J Kidney Dis Transpl. 2014;25(5):1030–7.
174. Patel HP, Goldstein SL, Mahan JD, Smith B, Fried CB, Currier H, et al. A standard, noninvasive monitoring of hematocrit algorithm improves blood pressure control in pediatric Hemodialysis patients. Clin J Am Soc Nephrol. 2007;2(2):252–7.
175. Paglialonga F, Ardissino G, Galli MA, Scarfia RV, Testa S, Edefonti A. Bioimpedance analysis and cardiovascular status in pediatric patients on chronic Hemodialysis. Hemodial Int. 2012;16(Suppl 1):S20–5.
176. Paglialonga F, Consolo S, Galli MA, Testa S, Edefonti A. Interdialytic weight gain in oligoanuric children and adolescents on chronic Hemodialysis. Pediatr Nephrol. 2015;30(6):999–1005.
177. Karava V, Benzouid C, Kwon T, Macher MA, Deschênes G, Hogan J. Interdialytic weight gain and vasculopathy in children on hemodialysis: a single center study. Pediatr Nephrol. 2018;33(12):2329–36.
178. Shroff R, Smith C, Ranchin B, Bayazit AK, Stefanidis CJ, Askiti V, et al. Effects of hemodiafiltration versus conventional Hemodialysis in children with ESKD: the HDF, heart and height study. J Am Soc Nephrol. 2019;30(4):678–91.
179. De Zan F, Smith C, Duzova A, Bayazit A, Stefanidis CJ, Askiti V, et al. Hemodiafiltration maintains a sustained improvement in blood pressure compared to conventional Hemodialysis in children-the HDF, heart and height (3H) study. Pediatr Nephrol. 2021;36(8):2393–403.
180. KDOQI Clinical Practice Guideline for Nutrition in Children with CKD. 2008 update. Executive summary. Am J Kidney Dis. 2009;53(3 Suppl 2):S11–104.
181. Shaldon S. Dietary salt restriction and drug-free treatment of hypertension in ESRD patients: a largely abandoned therapy. Nephrol Dialysis Transplantation. 2002;17(7):1163–5.
182. Maduell F, Navarro V. Dietary salt intake and blood pressure control in haemodialysis patients. Nephrol Dial Transpl. 2000;15(12):2063.
183. He FJ, MacGregor GA. Importance of salt in determining blood pressure in children: meta-analysis of controlled trials. Hypertension. 2006;48(5):861–9.
184. Paglialonga F, Shroff R, Zagozdzon I, Bakkaloglu SA, Zaloszyc A, Jankauskiene A, et al. Sodium intake and urinary losses in children on dialysis: a European multicenter prospective study. Pediatr Nephrol. 2023;38(10):3389–99.
185. Parekh RS, Flynn JT, Smoyer WE, Milne JL, Kershaw DB, Bunchman TE, et al. Improved growth in young children with severe chronic renal insufficiency who use specified nutritional therapy. J Am Soc Nephrol. 2001;12(11):2418–26.
186. Paglialonga F, Schmitt CP. Sodium handling in pediatric patients on maintenance Dialysis. Pediatr Nephrol. 2023;38(12):3909–21.
187. Kupferman JC, Aronson Friedman L, Cox C, Flynn J, Furth S, Warady B, et al. BP control and left ventricular hypertrophy regression in children with CKD. J Am Soc Nephrol. 2014;25(1):167–74.
188. Lin JJ, Mitsnefes MM, Smoyer WE, Valentini RP. Antihypertensive prescription in pediatric dialysis: a practitioner survey by the Midwest pediatric nephrology consortium study. Hemodial Int. 2009;13(3):307–15.
189. Han S-W, Won Y-W, Yi J-H, Kim H-J. No impact of hyperkalaemia with renin–angiotensin system Blockades in maintenance haemodialysis patients. Nephrol Dialysis Transplantation. 2007;22(4):1150–5.
190. Mactier R, Hoenich N, Breen C. Renal association clinical practice guideline on haemodialysis. Nephron Clin Pract. 2011;118(Suppl 1):c241–86.

Publisher's note