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Mechanisms and Treatment of Intradialytic Hypertension

Peter Noel Van Buren, MD, MSCS¹ and Julia K Inrig, MD, MHS²

¹University of Texas Southwestern Medical Center, Department of Internal Medicine and Division of Nephrology

²Duke University Medical Center, Department of Internal Medicine, Division of Nephrology; Therapeutic Science and Strategy Unit, Quintiles Clinical Research Organization

Abstract

Background—Intradialytic hypertension is an increase in blood pressure from pre to post hemodialysis that has recently been identified as an independent mortality risk in hypertensive hemodialysis patients. The mechanisms and management of intradialytic hypertension have been explored in numerous research studies over the past few years.

Summary—Patients with intradialytic hypertension have been found to be more chronically volume overloaded compared to other hemodialysis patients, although no causal role has been established. Patients with intradialytic hypertension have intradialytic vascular resistance surges that likely explain the blood pressure increase during dialysis. Acute intradialytic changes in endothelial cell function have been proposed as etiologies for the increase in vascular resistance, although it is unclear if endothelin-1 or some other vasoconstrictive peptide is responsible. There is an association between dialysate to serum sodium gradients and blood pressure increase during dialysis in patients with intradialytic hypertension, although it is unclear if this is related to endothelial cell activity or acute osmolar changes.

In addition to probing the dry weight of patients with intradialytic hypertension, other management strategies include lowering dialysate sodium and changing antihypertensives to include carvedilol or other poorly dialyzed antihypertensives.

Key Messages—Hemodialysis patients with intradialytic hypertension have an increased mortality risk compared to patients with modest decreases in blood pressure during dialysis. Intradialytic hypertension is associated with extracellular volume overload in addition to acute increases in vascular resistance during dialysis. Management strategies should include reevaluation of dry weight and modification of both the dialysate prescription and medication prescription.

Keywords

Hemodialysis; Hypertension; Intradialytic Hypertension; Endothelial Cell Dysfunction; Extracellular Volume Overload

INTRODUCTION

Hemodialysis is a life-sustaining procedure for end-stage kidney disease (ESKD) patients, but an accepted consequence of hemodialysis is the tendency for blood pressure (BP) to change frequently both during and between hemodialysis treatments. Large variability in BP measurements during hemodialysis is a risk factor for increased mortality in ESKD patients¹. The adverse outcomes associated with large decreases in BP during hemodialysis are well known², but nephrologists should be aware of the clinical significance of *increases* in BP during hemodialysis, as well. Intradialytic hypertension is an increase in BP from pre to post-hemodialysis that has been shown to be associated with poor outcomes^{3–5}. In recent years, numerous studies have explored the possible mechanisms responsible for intradialytic hypertension yielding potential interventions to consider to improve outcomes in this high-risk group of patients. The purpose of this review is to provide the most recent update in the epidemiology and pathogenesis of intradialytic hypertension and to consider the implications for management in the clinical setting.

EPIDEMIOLOGY

Cohort data demonstrates the expected response to a hemodialysis treatment is a reduction in systolic BP of about 10–15 mmHg with BP decreasing steeply during the first hour and then decreasing more slowly for the remainder of the treatment⁶. However, there is a spectrum of BP responses, with a notable subgroup even demonstrating increases in BP during the treatment. While BP variability occurs frequently both during and between hemodialysis treatments in most hemodialysis patients, observing BP patterns over prolonged periods of time will reveal which patients experience intradialytic hypertension most frequently. One recent cohort study defined intradialytic hypertension as an increase in systolic BP 10 mmHg from pre to post dialysis to identify the prevalence of this phenomenon over long term follow up. Over the course of 6 months, intradialytic hypertension occurred in over 90% of the patients at least once⁷. The median percentage of treatments where intradialytic hypertension occurred during that 6 month period in this cohort was 18% such that half the subjects experienced intradialytic hypertension in 18% of the treatments or less, and half experienced intradialytic hypertension in 18% or more of their treatments. Intradialytic hypertension occurred in at least 31% of the treatments in the quartile of subjects that experienced it most often. Finally, there were 9% of the subjects whose *mean* change in systolic BP from pre to post-hemodialysis, when assessed over 6 months, was an increase of at least 10 mmHg. In an even larger cohort study (n>100,000) extending out to 5 years, 10% were also noted to have a similar mean increase in systolic BP of at least 10 mmHg³. In summary, the overall frequency of which this phenomenon occurs will vary from cohort to cohort through different periods of time. However, there is clearly a subset of patients who experience this routinely.

OUTCOMES

The clinical significance of intradialytic hypertension lies in the fact that, among hypertensive hemodialysis patients, those with intradialytic hypertension appear to have some of the worst outcomes. Prior observational analysis showed that compared to patients

whose systolic BP decreased by at least 10 mmHg from pre to post-hemodialysis, those with increases in BP of that magnitude had a higher odds ratio for hospitalization or mortality after 6 months⁴. Intradialytic hypertension identified patients with decreased survival out to 2 years in comparison to patients whose BP decreased⁵. Since these studies, new epidemiologic data has placed intradialytic hypertension in direct comparison with the known morbidity associated with intradialytic *hypotension*. In a cohort of more than 100,000 hemodialysis patients followed for more than 5 years, a mean systolic BP decrease of 14 mmHg represented the group with the best survival³. The highest mortality occurred in patients with either a 30 mmHg decrease or *any* increase in systolic BP. This important study highlights the risk associated intradialytic hypertension and the need to identify the underlying mechanism responsible for it.

PATHOPHYSIOLOGY

Extracellular Volume Overload

Some of the earliest literature reporting on intradialytic hypertension suggested that the etiology of this “paradoxical” increase in BP was related to dynamic changes in the cardiac output during dialysis^{8,9}. These studies, which were limited by small sample size and lack of control groups for comparison, concluded that reduction in end diastolic volume that occurred through the process of ultrafiltration enabled BP to increase along with cardiac output. Agarwal et al¹⁰ demonstrated in retrospective analysis of the Dry-weight reduction in hypertensive hemodialysis patients (DRIP) study that dry weight probing over the course of several weeks lowered not only ambulatory BP, but also modified the intradialytic BP slope. The subjects in the dry weight reduction arm whose post-hemodialysis weight decreased the most over the course of the trial were noted to change from flat intradialytic BP slopes at baseline to steep declines in intradialytic BP at the end of the study. Although cardiac output was not measured in this study, the conclusion was that the phenotype of intradialytic hypertension could be modified through adjustment in dry weight over time. As more elaborate methods to directly measure fluid volumes in vivo are being utilized in research and clinical settings, the relationship between extracellular volume overload and intradialytic hypertension continues to be explored. Most recently, a cross sectional study used multifrequency bioimpedance spectroscopy to compare body composition among hemodialysis patients with differing intradialytic BP patterns during a single hemodialysis treatment¹¹. While the measurements were only reflective of a single treatment, a major strength of the study was the large sample size that was included. The findings demonstrated that those patients with BP increases during dialysis had higher ratios of extracellular water to total body water. These studies collectively support the practice that the initial approach to patients with intradialytic hypertension should be reassessment of dry weight.

Vasoconstriction

While it is appropriate to consider dry weight reduction in any hemodialysis patient with evidence of poorly controlled hypertension, there should be additional considerations to the etiology of a BP surge in patients with intradialytic hypertension. One case control study by Chou et al provided opposing evidence to the theories in the uncontrolled studies by Gunal and Cirit that changes in cardiac output are responsible for intradialytic hypertension¹².

Using cardiac output estimated from echocardiograms and BP measurements before and after hemodialysis, the authors found there was a significant increase in vascular resistance from pre to post-hemodialysis in the intradialytic hypertension patients compared to patients whose BP did not increase during hemodialysis. There were no significant differences in the change in cardiac output (or in the individual pre-dialysis or post-dialysis measurements) between the two groups. These authors and others have sought to identify specific mediators of the increase in BP that may be related to endogenous vasoconstrictive pressors. In the Chou study¹², there was no evidence that surges in sympathetic nervous system activity (assessed using plasma catecholamines) or in renin-angiotensin-aldosterone system activity (assessed using plasma renin activity) could explain the increase in vascular resistance. They did find, however, imbalances in endothelial cell derived mediators after dialysis in the intradialytic hypertension patients. Specifically, there were higher levels of the vasoconstrictor endothelin-1 (ET-1) and smaller ratios of the vasodilator nitric oxide to ET-1. Other studies have investigated changes in ET-1 during dialysis in intradialytic hypertension and found suggestive, but not confirmatory evidence to support this hypothesis. Raj et al¹³ found *decreases* in ET-1 in the hypotension prone patients with a trend towards increases in ET-1 in the intradialytic hypertension group. El-Shafey et al¹⁴ found increases in ET-1 in intradialytic hypertension patients, although there was no statistically significant between-group comparison with controls demonstrated.

Based on the evidence that vasoconstriction may be a predominant mechanism for intradialytic hypertension and that ET-1 has been implicated as a causative mediator for this, further investigation has been directed towards the overall role of endothelial cell dysfunction in intradialytic hypertension. One case control study found that patients with intradialytic hypertension (defined as an increase in systolic blood pressure of at least 10 mmHg from pre to post-hemodialysis in 4/6 screening treatments) had lower flow mediated vasodilation compared to hypertensive hemodialysis controls with BP decreases of that magnitude during screening¹⁵. This assessment of endothelial cell function occurred on a non-hemodialysis day such that it was not influenced by the hemodialysis procedure. The intradialytic hypertension patients also had lower levels of circulating endothelial progenitor cells than the controls. Endothelial progenitor cells have been validated as a marker of cardiovascular risk (lower levels indicating higher risk)¹⁶. These were measured prior to dialysis so that they also were not influenced by the hemodialysis procedure. It is of note that in the Inrig study¹⁵ the change in ET-1 from pre to post-hemodialysis was not different between the intradialytic hypertension patients and hemodialysis controls. So while endothelial cell dysfunction appears to be greater in intradialytic hypertension patients, the most recent research has focused on specific mechanisms by which the endothelial cells influence intradialytic blood pressure.

Dialysate Sodium

Dialysate composition has long been recognized as a factor in intradialytic hemodynamics¹⁷. As the serum sodium contributes largely to an individual's serum osmolarity, the role of dialysate sodium should be considered as a potential source of intradialytic hypertension. Previous studies have demonstrated how fluctuations in dialysate sodium and osmolarity can induce changes in fluid movement from various intracorporeal compartments. Using

dilutional techniques to estimate extracellular volume, it was shown that low dialysate sodium concentration (7% lower than serum) causes intradialytic *hypotension* with extracellular volume removal that exceeds the total fluid removed during hemodialysis¹⁸. Movement of fluid from the extracellular space to the intracellular space accounted for the additional extracellular fluid loss which likely resulted in the increased likelihood for symptomatic hypotension. High dialysate sodium achieved removal of fluid from both the intracellular and extracellular spaces such that the overall extracellular fluid reduction was much lower than with hypotonic or isotonic dialysate. A correlation between the dialysate to serum sodium gradient and the reduction in intracellular volume has been confirmed in a subsequent study utilizing whole body bioimpedance spectroscopy¹⁹. Based on the above studies, it would seem that stabilization of the BP during dialysis could be achieved by maintaining sufficient plasma volume at all times throughout the dialysis procedure to prevent clinically significant reductions in cardiac output. What has not been studied extensively is whether high sodium dialysate causes vasoconstriction via vasopressin increases driven by changes in serum osmolality. While the clinical application of dialysate sodium modification is typically directed to increasing dialysate sodium to prevent severe intradialytic *hypotension*, the question remains whether the use of prescribed dialysate sodium which is higher than serum sodium is contributing to intradialytic hypertension.

One randomized crossover study investigated how dialysate sodium modification affected BP in patients with a history of recurrent intradialytic hypertension²⁰. In this study, both serum sodium and blood pressure measured throughout the dialysis treatment were much higher when subjects were exposed to high dialysate sodium (pre dialysis serum sodium + 5) compared to low dialysate sodium (pre dialysis serum sodium – 5). The study also sought to explore how endothelial cell function was influenced by the different sodium gradients. Based on experimental evidence in endothelial cell culture that high extracellular sodium concentration impairs nitric oxide release²¹, it was hypothesized that the nitric oxide release would be lower and ET-1 would be higher while patients were exposed to high dialysate sodium. In the randomized study, there was no significant difference in nitric oxide or ET-1 level based on the dialysate sodium, but it was found that the subjects that were exposed to high dialysate as the first intervention had higher ET-1 and lower nitric oxide throughout the study suggesting that there may be some long-lasting carry over effect from high dialysate sodium.

MANAGEMENT AND TREATMENT

The management of intradialytic hypertension warrants consideration of both the short term and long term consequences of intradialytic hypertension. One case control study showed that ambulatory BP was higher in intradialytic hypertension patients compared to other hypertensive HD patients whose BP decreased during dialysis²². One can consider approaches to target the higher interdialytic blood pressure or targeting the intradialytic increase in BP itself. Table 1 includes a hypothetical case of a patient with intradialytic hypertension and addresses many of management options we will discuss next.

As stated above, we recommend that dry weight reduction be considered as an initial approach in any hypertensive hemodialysis patient. Agarwal demonstrated in a randomized

clinical trial that slow probing of dry weight results in decreased BP both in the HD unit and during the interdialytic time period²³. The efficacy of this approach has yet to be confirmed specifically in patients with intradialytic hypertension, but the evidence that intradialytic hypertension patients tend to be chronically volume overloaded supports consideration of this as the first intervention.

As dry weight reduction may not be sustainable or tolerable, pharmacologic antihypertensive therapy will be required in most hypertensive hemodialysis patients. Awareness of the dialyzability of commonly used pharmacologic antihypertensive agents is important when evaluating intradialytic hypertension patients²⁴. Changing a highly dialyzable drug to a less dialyzable drug should be a consideration in this patient population, but there should also be consideration of selecting drugs that may have pleiotropic properties that specifically benefit intradialytic hypertension patients beyond overall lowering of BP. There is data from an uncontrolled pilot study that the initiation of carvedilol in intradialytic hypertension patients decreases the ambulatory BP, improves FMD, and reduces the overall percentage of treatments in which intradialytic hypertension occurs²⁵. It is not possible to establish the mechanisms responsible for these findings due to the design of the study, but there is some in vitro data that carvedilol can inhibit the release of ET-1 in endothelial cell culture²⁶. In the case control portion of the formerly mentioned pilot study, there were no differences between intradialytic hypertension patients and controls regarding the change in ET-1 from pre to post-hemodialysis. Therefore it is difficult to interpret the significant reduction in *change in* ET-1 from pre to post-hemodialysis that occurred with carvedilol. Direct ET-1 antagonists are not routinely prescribed for hypertension alone and are not well studied in the HD population, so it is not currently recommended to consider one of these agents for the specific management of intradialytic hypertension. As more research is done in identifying the exact mechanisms responsible for intradialytic hypertension, the decision of which antihypertensive agent to use may become clearer.

The benefits of lowering the dialysate sodium were shown in the above randomized study²⁰, where all subjects were prone to intradialytic hypertension at baseline. However, there remains much debate over the individualization of dialysate sodium to manage hypertension in the general hypertensive hemodialysis population.^{27,28} In intradialytic hypertension prone patients, the BP reduction with lower dialysate sodium may in fact be beneficial as long as BP does not decrease excessively. In general, it is advised to regularly check serum sodium and to closely monitor for intradialytic *hypotension* if dialysate sodium is lowered. The long term effects of this intervention remain to be explored.

Conclusion

Intradialytic hypertension is now recognized as a recurrent and persistent phenomenon in a subset of hemodialysis patients. There continues to be epidemiologic evidence that intradialytic hypertension is associated with an increased risk for adverse outcomes that is comparable to patients that have excessive and dramatic decreases in BP during HD. Patients with intradialytic hypertension may be more chronically volume overloaded than hypertensive hemodialysis patients whose blood pressure decreases during hemodialysis, but they also appear to have a large, unexpected increase in vascular resistance that accounts for

the blood pressure increase during dialysis. Endothelial cell dysfunction is prevalent in intradialytic hypertension patients, although it remains undetermined whether ET-1 is the specific mediator of the intradialytic BP surge. Management of intradialytic hypertension patients should include an initial reassessment of dry weight. Patients with persistent intradialytic hypertension should be managed with less dialyzable drugs, and there is some evidence that Carvedilol may provide a specific benefit. Modification of the dialysate sodium can be considered, although labs and hemodynamics should be carefully monitored.

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References

1. Flythe J, Inrig J, Shafi T, et al. Intradialytic Blood Pressure Variability is Associated With Increased All-Cause and Cardiovascular Mortality in Patients Treated With Long-term Hemodialysis. *American Journal of Kidney Diseases*. 2013; 61:966–974. [PubMed: 23474007]
2. Flythe J, Xue H, Lynch K, Curhan G, Brunelli S. Association of mortality risk with various definitions of intradialytic hypotension. *Journal of the American Society of Nephrology*. 2015; 2015:724–734. [PubMed: 25270068]
3. Park J, Rhee C, Sim J, et al. A comparative effectiveness research study of the change in blood pressure during hemodialysis treatment and survival. *Kidney International*. 2013; 84:795–802. [PubMed: 23783241]
4. Inrig J, Oddone EHV, Gillespie B, et al. Association of intradialytic blood pressure changes with hospitalization and mortality rates in prevalent ESRD patients. *Kidney International*. 2007; 71:454–461. [PubMed: 17213873]
5. Inrig J, Patel U, Toto R, Szczech L. Association of blood pressure increases during hemodialysis with 2-year mortality in incident hemodialysis patients: a secondary analysis of the Dialysis Morbidity and Mortality Wave 2 Study. *American Journal of Kidney Diseases*. 2009; 54:881–890. [PubMed: 19643520]
6. Dinesh K, Kunaparaju S, Cape K, Flythe J, Feldman H, Brunelli S. A model of systolic blood pressure during the course of dialysis and clinical factors associated with various blood pressure behaviors. *American Journal of Kidney Diseases*. 2011; 58:794–803. [PubMed: 21803464]
7. Van Buren P, Kim C, Toto R, Inrig J. The prevalence of persistent intradialytic hypertension in a hemodialysis population with extended follow-up. *International Journal of Artificial Organs*. 2012; 35:1031–1038. [PubMed: 23065874]
8. Gunal A, Karaca I, Celiker H, Ilkay E, Duman S. Paradoxical rise in blood pressure during ultrafiltration is caused by increased cardiac output. *Journal of Nephrology*. 2002; 15:42–47. [PubMed: 11936425]
9. Cirit M, Akcicek F, Terzioglu E, et al. “Paradoxical” rise in blood pressure during ultrafiltration in dialysis patients. *Nephrology Dialysis Transplantation*. 1995; 10:1417–1420.
10. Agarwal R, Light R. Intradialytic hypertension is a marker of volume excess. *Nephrology Dialysis Transplantation*. 2010; 25:3355–3361.
11. 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 International*. 2015; 87:452–457. [PubMed: 25075771]
12. Chou K, Lee P, Chen C, et al. Physiologic changes during hemodialysis in patients with intradialysis hypertension. *Kidney International*. 2006; 69:1833–1838. [PubMed: 16691262]
13. Raj D, Vincent B, Simpson K, et al. Hemodynamic changes during hemodialysis: Role of nitric oxide and endothelin. *Kidney International*. 2002; 61:697–704. [PubMed: 11849413]

14. El-Shafey E, El-Nagar G, Selim M, El-Sorogy H, Sabry A. Is there a role for endothelin-1 in the hemodynamic changes during hemodialysis? *Clinical and Experimental Nephrology*. 2008; 2008:370–375. [PubMed: 18568290]
15. Inrig J, Van Buren P, Kim C, Vongpatanasin W, Povsic T, Toto R. Intradialytic Hypertension and its Association with Endothelial Cell Dysfunction. *Clinical Journal of the American Society of Nephrology*. 2011; 6:2016–2024. [PubMed: 21757643]
16. Werner N, Kosiol S, Schiegl T, et al. Circulating Endothelial Progenitor Cells and Cardiovascular Outcomes. *New England Journal of Medicine*. 2005; 353:999–1007. [PubMed: 16148285]
17. Palmer, B. *Dialysate Composition in Hemodialysis and Peritoneal Dialysis*. 4. Philadelphia: Lippincott Williams and Wilkins; 2009.
18. Van Stone J, Bauer J, Carey J. The effect of dialysate sodium concentration on body fluid distribution during hemodialysis. *Trans American Society of Artificial Internal Organs*. 1980; 26:383–386.
19. Sarkar S, Wystrychowski G, Usvyat L, Kotanko P, Levin N. Fluid dynamics during hemodialysis in relationship to sodium gradient between dialysate and plasma. *ASAIO*. 2007; 53:339–342.
20. Inrig J, Molina C, D'Silva K, et al. Effect of low versus high dialysate sodium concentration on blood pressure and endothelial-derived vasoregulators during hemodialysis: a randomized crossover study. *American Journal of Kidney Diseases*. 2015; 65:464–473. [PubMed: 25530107]
21. Oberleithner H, Riethmuller C, Schillers H, MacGregor G, de Wardener H, Hausberg M. Plasma sodium stiffens vascular endothelium and reduces nitric oxide release. *Proceedings of the National Academy of Sciences*. 2007; 104:16281–16286.
22. Van Buren P, Kim C, Toto R, Inrig J. Intradialytic Hypertension and the association with interdialytic ambulatory blood pressure. *Clinical Journal of the American Society of Nephrology*. 2011; 6:1684–1691. [PubMed: 21734087]
23. Agarwal R, Alborzi P, Satyan S, Light R. Dry-Weight Reduction in Hypertensive Hemodialysis Patients (DRIP): A Randomized, Controlled Trial. *Hypertension*. 2009; 53:500–507. [PubMed: 19153263]
24. Inrig J. Antihypertensive agents in hemodialysis: a current perspective. *Seminars in Dialysis*. 2010; 23:290–297. [PubMed: 20374548]
25. Inrig J, Van Buren P, Kim C, Vongpatanasin W, Povsic T, Toto R. Probing the Mechanisms of Intradialytic Hypertension: A Pilot Study Targeting Endothelial Cell Dysfunction. *Clinical Journal of the American Society of Nephrology*. 2012; 7:1300–1309. [PubMed: 22700888]
26. Saijonmaa O, Metsarinne K, Fyhrquist F. Carvedilol and its metabolites suppress endothelin-1 production in endothelial cell culture. *Blood Pressure*. 1997; 6:24–28. [PubMed: 9116922]
27. Sang G, Kovithavongs C, Ulan R, Kjellstrand C. Sodium ramping in hemodialysis: a study of beneficial and adverse effects. *American Journal of Kidney Diseases*. 1997; 29:669–677. [PubMed: 9159299]
28. Hecking M, Karaboyas A, Saran R, et al. Predialysis serum sodium level, dialysate sodium, and mortality in maintenance hemodialysis patients: the Dialysis Outcomes and Practice Patterns Study (DOPPS). *American Journal of Kidney Diseases*. 2012; 59:238–248. [PubMed: 21944663]

TABLE 1

Table 1 includes a hypothetical case scenario of a patient with intradialytic hypertension. In the dialysis flowsheet, there is information on pre and post dialysis systolic blood pressure and weight, in addition to dialysate/serum sodium and medications. This case illustrates some of the key information to monitor in managing a patient with intradialytic hypertension.

Pre HD SBP (mmHg)	Post HD SBP (mmHg)	Low SBP (mmHg)	Pre HD Weight (kg)	Post HD Weight (kg)	Target Weight	Weight Gain (kg)	Dialysate Na (mg/dL)	Serum Na (mg/dL)
157	183	139	74.6	70.5	70.5	5.0	140	135
169	140	125	75.3	70.6	70.5	4.8	140	
145	163	140	75.7	70.5	70.5	5.1	140	
167	176	139	74.5	70.3	70.5	4	140	136
148	169	128	75	70.4	70.5	4.7	140	
145	155	130	75.8	70.6	70.5	5.4	140	
<div><div>1</div><div>2</div><div>3</div></div> <div>MEDICATIONS Lisinopril 40 mg po qday Amlodipine 5 mg po qday Sevelamer 1600 mg po three times daily with meals Lipitor 20 mg po qhs</div> <div>4</div>								

1. The patient is hypertensive according to recommendations of the National Kidney Foundation to target a systolic blood pressure less than 140 mmHg pre dialysis or less than 130 mmHg post dialysis. Most of his treatments involve a significant increase in blood pressure from pre to post dialysis.
2. While intradialytic hypotension is not limiting the fluid removal during dialysis, the overall high blood pressure and large weight gains in between dialysis treatments may indicate that the patient could be chronically volume overloaded. An assessment of extracellular volume status should be the first step in management of intradialytic hypertension. This includes not only limiting the interdialytic sodium intake, but also assessing whether the dry weight needs to be challenged.
3. The patient has a dialysate-serum sodium gradient that favors transfer of sodium into the patient during dialysis. This likely contributes to increased fluid intake and weight gain. Furthermore, the acute changes in sodium during hemodialysis may acutely influence blood pressure through changes in osmolarity or endothelial cell function.
4. Review of the antihypertensive medications indicates that the primary drug, Lisinopril, is easily dialyzable and may contribute to increases in blood pressure during dialysis. There is observational evidence that the medication carvedilol blunts the increase in blood pressure from pre to post dialysis in patients with intradialytic hypertension, although the proposed mechanism of inhibiting release of endothelin-1 during dialysis needs to be confirmed in controlled studies. Consideration should be placed to changing this patient to a less dialyzable inhibitor of the renin angiotensin aldosterone system (losartan) or starting the patient on a medication like carvedilol.