Better Hemodialysis Patient Care

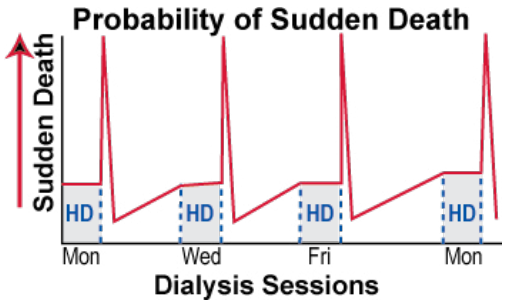
THROUGH CARDIAC FUNCTION ASSESSMENT

transonic
THE MEASURE OF BETTER RESULTS.

www.transonic.com

Cardiovascular Disease — An ESRD Epidemic

There is an epidemic among hemodialysis patients. It is the leading cause of death and morbidity in patients with end-stage renal disease (ESRD)[2-3]. It accounts for half of dialysis patient deaths and a third of patient hospitalizations.[4] And it is not being monitored very well.



"35% of deaths occurred in the first 12-hour interval ... 27% of these deaths occurred during dialysis and 33% occurred in the first hour after the dialysis treatment."[6]

Cardiovascular disease (CVD) is wreaking havoc on ESRD patients—both during their hemodialysis treatments and during their time away from clinics.

"In addition, cardiovascular collapse is a major cause of complications during hemodialysis treatments."[5] Congestive heart failure (CHF) in ESRD patients results from cardiac overload, anemia, severe hypertension and cardiac dysfunction. CVD mortality rates are approximately 30 times that of the general population,[4] and in adolescents, CVD mortality rates are over 1,000 times that of their age-related peers.[28]

Patients who do not feel well at the end of a dialysis session are subject to an unidentified decrease in Cardiac Index (CI) to critical ICU levels of <2 L/min/m².

As an AV fistula steals flow from an already limited systemic circulation, low CI can become a major contributor to decreased myocardial perfusion leading to sudden death.

Hemodialysis — A Stress Test for Cardiac Function

> *Hemodynamic stability is threatened and often severely compromised by hemodialysis largely because of the obligate fluid removal during a short time span.*[7]

Thomas Depner, MD, underscores the importance of testing cardiac function during hemodialysis.[7,8] He notes that the rapid removal of large volumes of fluid during hemodialysis severely tests the limits of a patient's cardiac function. Just as a treadmill stress test gauges a heart's response to exercise, cardiac output measurements during hemodialysis monitor a heart's response to fluid removal during the dialysis treatment. Because cardiovascular parameters can change dramatically during dialysis, multiple cardiac measurements are advised during a dialysis session in order to assess a patient's clinical condition.[8]

Cardiac Output and Access Flow

Although extensively documented in the literature, the AV access is also often overlooked as a source of cardiac dysfunction. By bypassing the customary arteriole/capillary beds and establishing a direct high flow connection between the arterial and venous systems, an AV access creates a drop in peripheral arterial resistance which significantly affects blood flow. In order to maintain blood pressure and improve cardiac output, the body compensates for this precipitous drop in resistance by increasing heart rate and stroke volume.[3,9,10] This phenomena was first observed in World War II soldiers with trauma-induced arteriovenous fistulas.[3] Iwashima et al reported a 15% increase in cardiac output by the seventh day after arteriovenous fistula creation.[10] This increased cardiac workload can lead to an increase in size of the left ventricle (left ventricular hypertrophy).[9,10]

"An Easily Overlooked Diagnosis"

In 1995, Engelberts and Tordoir et al (Maastricht University, the Netherlands) reported a case where excessive shunting in a hemodialysis access fistula led to high-output cardiac failure. They termed it "an easily overlooked diagnosis." Following surgical closure of the fistula, the patient's condition improved, and signs of congestive heart failure subsided."[7] In 1998, PR Young Jr. et al (Bowman Gray School of Medicine, Wake Forest University) reported two renal transplant patients who developed high-output cardiac failure from brachiocephalic fistulas. Successful transplantation, coupled with fistula ligation, resolved the cardiac complications.[8] Additional reports[30-32] cemented the relationship between high-volume AV access flows and cardiac complications.

Access Flow - Cardiac Output (AF/CO) Ratio

MacRae et al (University of Calgary, Canada) reported the high output cardiac failure associated with high flow AVFs (> 1.5 L/min), particularly in men with upper arm fistulas and previous access surgeries.[2,4,5] In her 2006 comprehensive review, "The Cardiovascular Effects of Arteriovenous Fistulas in Chronic Kidney Disease: A Cause for Concern?", MacRae documents the evidence, to date, on the subject.[2] She emphasizes that the ratio between access flow and cardiac output is an important clinical indicator and notes that the average flow in an upper arm fistula is 1.13 to 1.72 L/min. In the same study, 15% of patients were found to have flows of over 2 L/min. Access flow that exceeds 25% of cardiac output indicates a potential cardiac problem. In most cases, high output cardiac failure was associated with a access flow to cardiac output ratio of more than 40 percent.[10] MacRae recommends that hemodialysis patents be screened for potential high output cardiac failure using a Qa/CO ratio and patients with a Qa/CO ratio of more than 30 percent undergo further testing.[10]

Italian Study Sets 2L/min AVF Flow Cut-off Value

In 2008, Basile et al (Miulli General Hospital, Acquaviva delle Fonti, Italy) published a study of 96 patients with AV fistulas and cardiac failure.[24] The study showed that upper arm AVFs are associated with an increased risk of high output cardiac failure. It was the first published study with a high predictive power for AV fistula flows greater or equal to 2.0 L/min to result in high-output cardiac failure. In this landmark study, both AV access flow and cardiac output were measured using the Transonic Hemodialysis Monitor.

"A high flow AV access can produce life-threatening cardiac complications. The volume flow level that will induce high-output failure or extremity ischemia will vary with each patient, based on co-morbidities, especially the degree of cardiac disease and peripheral arterial disease. For patients at risk based on such pre-existing conditions, which can be a majority of patients in a given hemodialysis population, the widespread consensus (evidence-based) is that patients with access flows of 2L/min or higher should be tested and followed for these complications--and have a flow-reduction procedure performed at the earliest signs of cardiac complications or extremity ischemia.

Unfortunately, with the high prevalence of cardiac disease in the HD population, an insidious and silent access flow as a major cause or contributor to a potentially deadly cardiac complication, is often overlooked. Therefore, it is critically important for the practitioner to be aware of the relationship between access flow and cardiac failure, since many of these high-flow patients will have morbidity and mortality that otherwise could have been avoided."[39]

Lawrence Spergel MD, FACS, founding father and clinical director of the Fistula First Breakthrough Initiative

Studies/Reviews Highlight High AVF - CO Link

In the 2013 October issue of Clinical Transplant, Schier et al (Innsbruck University, Austria) reported the results of a 2005-2010 retrospective study of kidney-transplant recipients. Twenty-five percent of the recipients (29 of 113) needed an AV fistula closure, mostly due to cardiac failure symptoms.[25] Stern et al from UNC Kidney Center's Division of Nephrology and Hypertension, in Chapel Hill, NC describes how an increase in preload can lead to increased cardiac output when a large proportion of arterial blood is shunted from the left-sided circulation to the right-sided circulation via the fistula. Patients may present with the usual signs of high-output heart failure including tachycardia, elevated pulse pressure, hyperkinetic precordium, and jugular venous distension. The nephrologist is then faced with the dilemma of preventing progression of heart failure at the expense of losing a vascular access. The authors conclude that treatment should be directed at correcting the underlying problem by surgical banding or ligation of the fistula.[26]

In her 2012 Seminars in Nephrology article, "High-output Heart Failure: How to Define It, When to Treat It, and How to Treat It," Wasse et al (Emory University) succinctly outlines the problem.[27] Dr. Wasse describes the mechanisms by which a dialysis AV access may promote the development of high-output cardiac failure, the risk factors for and diagnosis of high-output heart failure, and recommends management strategies for patients with high-output heart failure. The literature addressing the various types of cardiac complications (congestive heart failure, left ventricular hypertrophy, coronary artery disease, right ventricular dysfunction, valvular heart disease, aortic stenosis) of AV fistulas in patients with end-stage renal disease has been most recently reviewed by Dr. Alkhouli and colleagues in their 2015 publication in Nefrologia.[28]

EXCERPT: CARDIAC COMPLICATIONS OF ARTERIOVENOUS FISTULAS IN PATIENTS WITH END-STAGE RENAL DISEASE

Alkhouli M et al,, Nefrologia. 2015 May-Jun;35(3):234-45[28]

"Despite their association with a lower mortality, AVFs have significant effects on cardiac functions predominantly related to the increase in preload and cardiac output (CO). Patients with end stage renal disease (ESRD) requiring dialysis almost invariably have volume overload due to water and salt retention. They also have pressure load due to arterial sclerosis and hypertension, and increased CO secondary to chronic anemia. In addition, many hemodialysis patients have significant pre-existing myocardial, valvular or coronary heart disease. It is, therefore, often difficult to tease out the exact contribution of an AVF to cardiac dysfunction in hemodialysis patients. Nevertheless, worsening in cardiac functions soon after AVF creation has been observed favoring a causative effect of the AVF on certain cardiac functions. Current literature suggests that the creation of an AVF can cause or exacerbate the following conditions: congestive heart failure, left ventricular hypertrophy, pulmonary hypertension, right ventricular dysfunction, coronary artery disease, and valvular dysfunction."

Proactive Cardiac Function Assessment During Hemodialysis

It is therefore incumbent upon the nephrologist to order periodic cardiac function tests, and track the results along with its associated vascular access flow rates. While access flow remains fairly constant during a hemodialysis treatment, cardiac output decreases an average of 20% during the treatment causing less blood flow to be available to sustain the body's vital functions. A healthy body will respond to this by increasing peripheral resistance to sustain the blood supply to the heart and brain. Other considerations include:

- The site of a vascular access affects average flow values. Upper arm sites typically have higher flows than lower arm sites.
- Patients with initial high flow fistulas are at greater risk for cardiovascular problems. A fistula may "over-mature" and present a flow over 2 L/min.
- Autologous fistulas tend to remain sufficiently patent to sustain dialysis at lower flows than do prosthetic grafts.
- A straight upper arm prosthetic graft may initially exhibit an overly high flow. Graft flow tends to decrease over time, so banding a prosthetic graft is not advised. Access flow and cardiac function of these patients should be monitored monthly to ensure that access flow drops before cardiac complications arise.

"The ability to monitor cardiac output is one of the important cornerstones of hemodynamic assessment …in particular in patients with pre-existing cardiovascular comorbidities." Tucker T et al[11]

The Cardiovascular Effects of Arteriovenous Fistulas in Chronic Kidney Disease: A Cause for Concern

Immediate hemodynamic effects of AVF creation	• Increase in cardiac output (10-20%). • Increase in sympathetic nervous system activity (increasing contractility). • Increase in stroke volume and heart rate. • Decrease in peripheral resistance.
Hemodynamic changes within one week of AVF creation	• Increase in circulating blood volume resulting in increased left atrial, inferior vena cava, and left ventricle end-diastolic volume (LVEDV). • Increase in neuro-hormones: vasodilator atrial natriuretic peptide (ANP) and brain natriuretic peptide (BNP) implying atrial and ventricular filling pressure are increased. • Decrease in plasma renin and aldosterone levels. • Decrease in systemic vascular resistance and systolic/diastolic blood pressure.
Long-term consequences of AVF creation	**Left Ventricular Hypertrophy (LVH)** • An adaptive response to increased cardiac workload caused by volume or pressure overload.
	High-Output Cardiac Failure • High-flow AVF patients have a greater risk of developing CHF and greater increase in LVEDV. • AVFs in HD patients may contribute to the development of heart failure. • Left ventricle enlargement at the start of HD is very common and progressive left ventricle dilation with hypertrophy continues over time. Most of the left ventricle growth occurs during the first year of dialysis.
	Exacerbation of Coronary Ischemia • AVF placement is associated with increased myocardial O2 demand that may not be met, especially in patients with established coronary artery disease (CAD) or left ventricle hypertrophy (LVH). • Increased O2 consumption may have clinical manifestations in dialysis patients who have had CABG. A decrease in coronary perfusion that occurred with the onset of HD was demonstrated by the reduction in graft flow and reversible hypokinesis of the anterior left ventricle wall. • High-flow AVFs with associated high cardiac output may increase O2 demand.
	Central Vein Stenosis • The endothelium plays an active role in vascular remodeling by secreting vasoactive substances and growth factors in response to alterations in flow and shear stress. • Increased blood flow due to AVF creation alters the shear stress on the endothelium and promotes production of substances like transforming growth factor (TGF-ß) and NO which dilate the vessel lumen. • A majority of central vein stenosis occurs at the junction of the cephalic and subclavian veins. There was a high correlation between the location of a central vein stenosis and ipsilateral AVF. It suggests that altered flow hemodynamics due to a fistula may result in endothelial damage and vascular remodeling, leading to stenosis.

MacRae JM et al, Seminar in Dialysis 2006; 19:349-352.

Conclusions

- AVFs are superior to catheters and grafts due to fewer thrombogenic and infectious complications.
- A thorough cardiac assessment should be performed in patients with CAD prior to placing an AVF.
- Regular careful evaluations are necessary in patients with cardiac disease and AVFs.
- Patients with high flow fistulas (flow greater than 2L/min) and increasing LVEDV are recommended to have a flow reduction procedure on their AVF.
- Patients with preexisting severe ischemic heart disease should avoid AVF placement if the underlying ischemia cannot be treated.

The Quality of Cardiovascular Disease Care for Adolescents with Kidney Disease: *A Midwest Pediatric Nephrology Consortium Study.*

Background	Cardiovascular disease (CVD) is the leading cause of increased mortality for adolescents with advanced kidney disease. Many patients have CVD mortality rates 1,000 times that of their age-matched peers and will die prematurely in early adulthood. Guidelines call for screening for cardiovascular risk factors in this population of patients.
Objective	To ascertain if the quality of preventive cardiovascular care may impact long-term outcomes for these patients.
Methods	• Records of 196 consecutive adolescents from seven American centers and one Canadian pediatric center with pre-dialysis chronic kidney disease, on dialysis or with a kidney transplant, who transferred to adult-focused providers were reviewed. • Cardiovascular risk assessment and therapy within and across centers were compared. • Predictors of care were assessed using multilevel models.
Results	• Overall, 58% of five recommended cardiovascular risk assessments (family history of CVD, smoking status, lipid profile, physical activity, echocardiography for patients with a history of hypertension) were documented. • Documented most frequently was smoking status (74%); an echocardiogram in patients with a history of hypertension (70%); family CVD history (53%); fasting lipid profiles and physical activity (47%) respectively. • Only 20 of the 196 total patients (10%) received 100% of all indicated cardiovascular risk factor assessments. • Recommended therapy for six modifiable cardiovascular risk factors was documented 57% of the time. • Transfer after 2006 and kidney transplant status were also associated with increased cardiovascular risk assessment.
Conclusions	• Adolescents with kidney disease receive suboptimal preventive cardiovascular care, that may contribute to their high risk of future cardiovascular mortality. • A opportunity exists to improve outcomes for children with kidney disease by improving the reliability of preventive care that may include formal transition programs.

Hooper DK et al, Pediatr Nephrol 2013; 28(6): 939-49.29

Cardiac Function Assessment

Methodology

Cardiac output is the volume of blood being pumped by the heart in one minute. An average resting cardiac output is 5.6 L/min for a human male and 4.9 L/min for a female.[1]

> *It is astonishing that no one has arrived at the following obvious method by which the amount of blood ejected by the ventricle of the heart with each systole may be determined directly... Adolf Fick, 1870.*

Adolf Fick introduced a method to measure an animal's cardiac output (CO) from arterial and venous blood oxygen measurements. His principle later formed the foundation of Stewart's indicator-dilution technology. In 1928, Stewart's equation was modified by Hamilton who described the bell-shape of a classic dilution curve (Fig. 1).

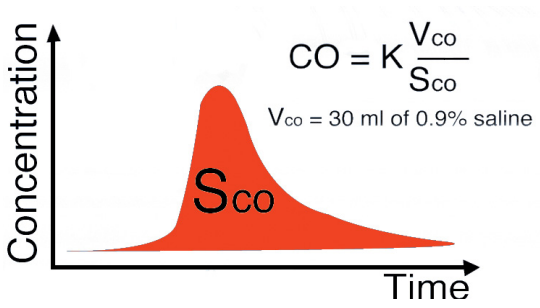


Fig. 1: Time concentration curve showing saline indicator dilution curve. CO is inversely related to the average dilution indicator concentration and the total time of indicator passage or

$$CO = \frac{\text{the amount of indicator injected}}{\text{area of the dilution curve}}$$

A variety of indicators have been used with this time-tested technology. All require that three criteria be met. They are:

1. **Injection Phase:** a known indicator is introduced into the circulatory system.
2. **Mixing/dilution Phase:** the indicator mixes with the blood.
3. **Detection Phase:** The indicator concentration is measured downstream from its introduction.

Ultrasound dilution methodology, pioneered by Nikolai Krivistki PhD, DSc, uses body temperature saline, an innocuous indicator, that is injected into a patient's peripheral vascular access during the dialysis treatment. Injected into the venous blood line, the indicator travels through the heart and lungs and returns via the arterial system where a Flow/dilution Sensor records the diluted blood concentration (Fig. 2). Classic Stewart-Hamilton equations are used to calculate cardiac function and central hemodynamic parameters including Cardiac Output (CO), Cardiac Index (CI), Peripheral Resistance (PR) and Central Blood Volume (CBV).

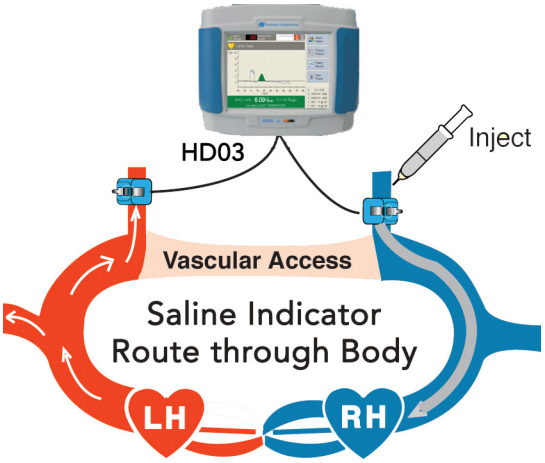


Fig. 2: Saline Indicator Route: Body temperature saline is injected into the venous line, travels through the heart and lungs and returns via the arterial system where a flow/dilution sensor records the diluted concentration.

Flow-QC® Cardiac Function Assessment

Transonic Flow-QC® Cardiac Function Assessment with ultrasound indicator dilution technology provides a way to integrate cardiac function studies into a hemodialysis clinic's treatment protocol in order to forestall the devastating consequences of CVD.

Transonic Flow-QC cardiac function measurements help diagnose cardiac overload in ESRD patients.[1,11] When access flows measured during the dialysis session are unusually high (>2 L/min), cardiac overload can be suspected. A follow-up Flow-QC cardiac output measurement will verify whether the heart is stressed.

Cardiac output measurements during hemodialysis combined with access flow identify:

- **A.** Prolonged high access flow to cardiac output ratio that stresses the heart and can result in cardiomegaly and heart failure.
- **B.** Dangerously low cardiac index that places patients at high risk for cardiovascular complications and failure.
- **C.** Dramatic decreases of cardiac index during hemodialysis due to inaccurate dry weight estimation and/or inadequate medication.
- **D.** Dangerous decrease in central blood volume during hemodialysis that may portend hypotensive episodes.

Flow-QC® Cardiac Function Parameters

Cardiac Output and calculated parameters are related to age and gender, and depend on a patient's clinical status such as the presence of diabetes or cardiac diseases and may change dramatically during a hemodialysis session.

CARDIAC OUTPUT (CO)

Normal Range[1]: 5 - 8 L/min;

The volume of blood (in liters) ejected by the heart in one minute, is a fundamental measure of human hemodynamic performance. Typical values for hemodialysis patients range from 4 to 8 L/min with the determination of "normal CO."

CARDIAC INDEX (CI)

Normal Range[1]: 2.2 - 4.5 L/min/m²

Cardiac output divided by estimated body surface area (BSA). A primary criterion of cardiac adequacy, CI is useful in comparing patients of different sizes. Cardiac indices from 6 - 8 L/min/m² may indicate high access flow. A low CI (< 2 L/min/m²) at the beginning of a hemodialysis session indicates significant deterioration of cardiac function. A decrease in CI during the hemodialysis session indicates potential cardiac problems, inadequate dry weight estimation, and/or inadequate medication prescription.

PERIPHERAL RESISTANCE (PR)

Normal Range[1]: 9.6 - 18.8 mmHg x min/L (770 - 1500 dyne x sec/cm[5])

The average resistance to systemic blood flow is approximated as mean arterial pressure divided by cardiac output. Patients diagnosed with diabetes may have substantially higher PR. Since CO generally decreases during hemodialysis and pressure is maintained, PR will increase during hemodialysis for most patients. Dr. Depner suggests that patients whose PR does not increase may have fluid overload. A Depner study correlated a higher initial PR, lower initial CO, and failure of PR to increase during hemodialysis with an increased 1-year mortality risk.[8]

CENTRAL BLOOD VOLUME (CBV)

Normal values range from 0.8 - 1.6 L

Central blood volume is the volume of blood in the heart, lungs, and great vessels. Central blood volume index (CBVI) is CBV divided by the patient's weight (typical range, 11 - 17 mL/kg). CBV maintenance may be a factor in blood pressure regulation. CBV decreases during hemodialysis are similar to CO, and probably precedes a decrease in CO. When CBV is depleted, hypotensive episodes may occur. Monitoring CBV during ultrafiltration may indicate how fast a patient can be dialyzed without hypovolemic collapse.

Flow-QC® Cardiac Function Parameters

Parameter	Typical Range	Abnormal Range	Clinical Relevance	Suggested Interpretation & Recommendations
ACCESS FLOW (AF)	500 - 1600 mL/min	< 500 mL/min > 1600 mL/min for naive fistula	Heart compensates AF > 30% of CO CI < 2.2	Consider reducing AF by banding or other surgical procedure to avoid prolonged heart overload. Body tissues are not adequately perfused due to A-V fistulae stealing. Repair or consider closure of fistula.
CARDIAC INDEX (CI)	2.5 - 4.2 L/min/m^2	CI > 5 L/min/m^2 CI < 2.0 L/min/m^2	Usually indicates heart overload due to high access flow (see above) Significant volume of accumulated liquid between dialysis sessions. May indicate low hematocrit Observed at the beginning of the HD session: indicates significant deterioration of CO function. Observed as a drop in CI during HD session: indicates potential cardiac conditions, inadequate dry weight estimation and/or medication prescription.	The reason for the increased CI should be identified and proper treatment implemented including: • A-V access intervention; • Change in dialysis prescription; • Change of erythropoietin prescription. Refer to cardiologist for full study. The dry weight and medications should be examined and/or changed and central hemodynamic profiling (CHP) measurements repeated.
CENTRAL BLOOD VOLUME INDEX (CBVI)	11 - 17 ml/kg	< 10 ml/kg > 20 ml/kg	Usually observed in obese patients where heart-lung system is relatively small compared to body weight. High CBVI usually (especially if maintained during CHP) indicates extra fluid in lung circulation or left ventricular dilation	Observation of CBVI decrease during or at the end of CHP may indicate patient is at risk for hypovolemic collapse. Dialysis prescription may be reconsidered Perform follow-up studies.

** Parameters are given for research purposes. Some do not have well-established normal values.*

1Darovic G.O.: Hemodynamic Monitoring Invasive and Noninvasive Clinical Application. WB Saunders Company, 1987.

Measuring Cardiac Function

Cardiac function measurements with a Transonic® HD03 Flow-QC® Hemodialysis Monitor require:

- Cardiac Output DTM inserted into the top rear of the HD03 Hemodialysis Monitor
- Flow-QC Clear Advantage Tubing Set with a dedicated injection port for saline indicator injections into the venous blood line
- 30-mL syringes filled with saline warmed to body temperature

DISPOSABLE FLOW-QC® CLEAR ADVANTAGE TUBING SET

A Flow-QC Clear Advantage Tubing Set provides a safe injection port for a rapid 4 -7 second injection of a cardiac output saline bolus. The tubing set provides a consistent measurement environment. The ultrasonic and mechanical properties of these tubing sets are controlled to guarantee measurement accuracy, eliminate measurement variability from blood line brands, and reduce the need for periodic sensor calibration.

The Flow-QC Clear Advantage Tubing Set is placed in the hemodialysis circuit between the bloodline tubing and the venous and arterial needle tubing with the Flow/dilution Sensors positioned on the Flow-QC Clear Advantage Tubing. A bolus injection at another site, such as the bubble trap, would take too long to pass through the sensor and the software program may not be able to separate the timing of the first pass of the saline bolus from subsequent passes.

NORMAL CARDIAC FUNCTION VALUES

(Hemodialysis Population)

Cardiac function depends on age, gender, and medical history (diabetes or cardiac disease). Cardiac parameters may fluctuate dramatically during a hemodialysis treatment. Flow-QC Surveillance measures:

CO	**Cardiac Output**	*5 to 8 L/min (wgt & hgt dependent)*
CI	**Cardiac Index**	*2.2 to 4.5 L/min/m²*
CBV	**Central Blood Volume**	*0.8 to 1.6 L (weight dependent)*
CBVI	**Central Blood Volume Index**	*11 - 17 ml/kg*
PR	**Peripheral Resistance**	*9.6 - 18.8 mmHgxmin/L*

To measure cardiac output and related parameters, fill a 30 mL syringe with 30 mL of saline warmed to body temperature. Insert Flow-QC® Clear Advantage® tubing segment into the hemodialysis circuit as shown (Fig. 3) and then prime tubing.

Attach the arterial & venous Flow-QC Clear Advantage tubing to the needle tubing (c) in normal line position with the Flow/dilution Sensors positioned in the middle of the Flow-QC Clear Advantage tubing lines with the arrows on the sensors each pointed in the direction of flow.

With a Cardiac Output Data Transfer Module (DTM-CO) inserted in the HD03 Monitor, press the [Measure Patient] icon. Select the Flow-QC Tubing icon on the [Select Tubing] screen. Then press the [Cardiac Output] button to initiate the cardiac output measurement sequence. Enter parameters in the required fields and follow on-screen directions for the 6-7 second injection of 30 mL warmed saline. Measurement results including a CO dilution curve, calculated CO, CI and CBV values will display on the monitor. Notes:

- If two measurements are within 15% of each other, a third measurement is not needed. If a Repeat Measurement message displays, repeat injection.
- CO can be measured in patients with access flow and no access recirculation. CO cannot be measured in patients with a central venous catheter.

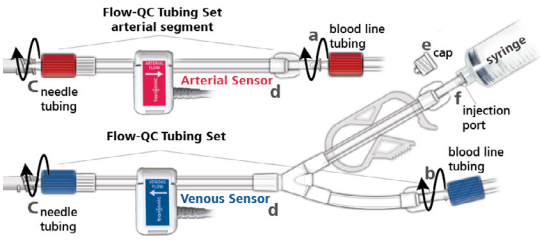


Fig. 3: The red-banded end of the arterial segment is connected to the male end of the arterial bloodline and the blue-banded branch of the Y end is connected to the male luer-lock connector on the venous bloodline.

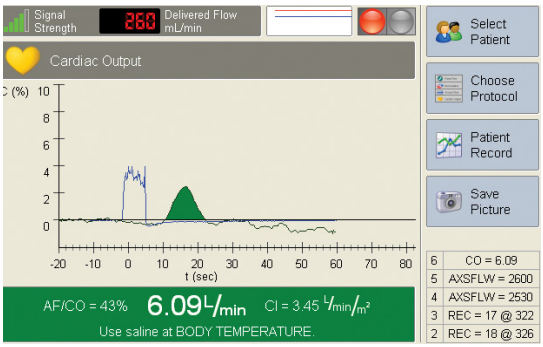


Fig. 4: Monitor display of measurement results.

Central Hemodynamic Profiling (CHP)

Central Hemodynamic Profiling identifies low CI and offers the physician the opportunity to improve CI by adjusting dry weight medication and length of dialysis.[1,11]

Effective cardiac function management depends on a routine screening program such as Central Hemodynamic Profiling (Fig. 5) that identifies patients who leave hemodialysis sessions with dangerously low cardiac indices (CI ≤ 2.0), thereby increasing their risk for death, stroke or myocardial infarction. CHP is the periodic assessment of cardiac function during hemodialysis in order to track the heart's response to the stress of a dialysis treatment.

A CHP study (Flow Chart, page 18) consists of hourly cardiac output measurements throughout the hemodialysis treatment. Transonic® Flow-QC Cardiac Output software automatically calculates cardiac index. If cardiac index drops below 2 L/min/m² during treatment, the hemodialysis prescription should be reviewed and adjusted immediately.

After adjustments are made, another CHP study should be performed during the next dialysis session. If this profile is stable and in the appropriate range, the patient's cardiac status can then be monitored as usual.

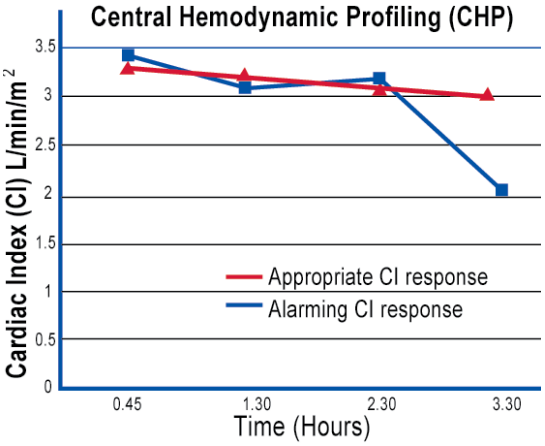


Fig. 5: Central Hemodynamic Profiling (CHP): four measurements taken during a single hemodialysis session shows Cardiac Index responses to the hemodialysis treatment. Acceptable CI results range between 2.5 - 4.2 L/min/m².[37,38]

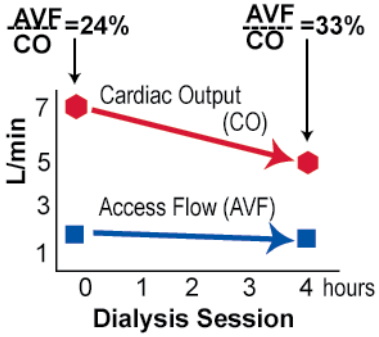


Fig. 6: One-third of CO is redirected from the systemic circulation to the AV fistula placing patients at cardiac risk.

Cardiac Function Study Protocol

CHP identifies:

- Prolonged high levels of access flow (>1,600-2,000 mL/min) that can lead to cardiomegaly and high output cardiac failure identified by an access flow to cardiac output ratio (AVF/CO) exceeding 25-30% (Fig. 6).
- Cardiac Index of <2 L/min/m².
- Dramatic 20-30% drop in cardiac output during dialysis due to inaccurate dry weight estimation and/or medication that places patients at high risk for cardiovascular complications and sudden death following the session (Figs. 6,7).

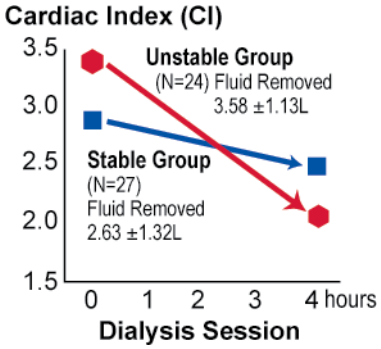


Fig. 7: Inadequate dry weight estimation increases the risk of cardiac failure.[1,11]

FLOW-QC CARDIAC FUNCTION STUDY PROGRAM

INITIAL CARDIAC STABILITY ASSESSMENT

For new patients, patients who have had interventions, and patients with suspected cardiac complications. Transonic Flow-QC Protocol begins with a Tucker Central Hemodynamic Profiling (CHP) study consisting of hourly cardiac output measurements during the hemodialysis session. If a patient is stable (CI > 2.5), the measurements serve as the first data point for the patient's cardiac function baseline.

THREE-PART BASELINE CARDIAC FUNCTION STUDY

The Baseline Cardiac Function Study established reliable average cardiac function parameters for the patient and consists of:

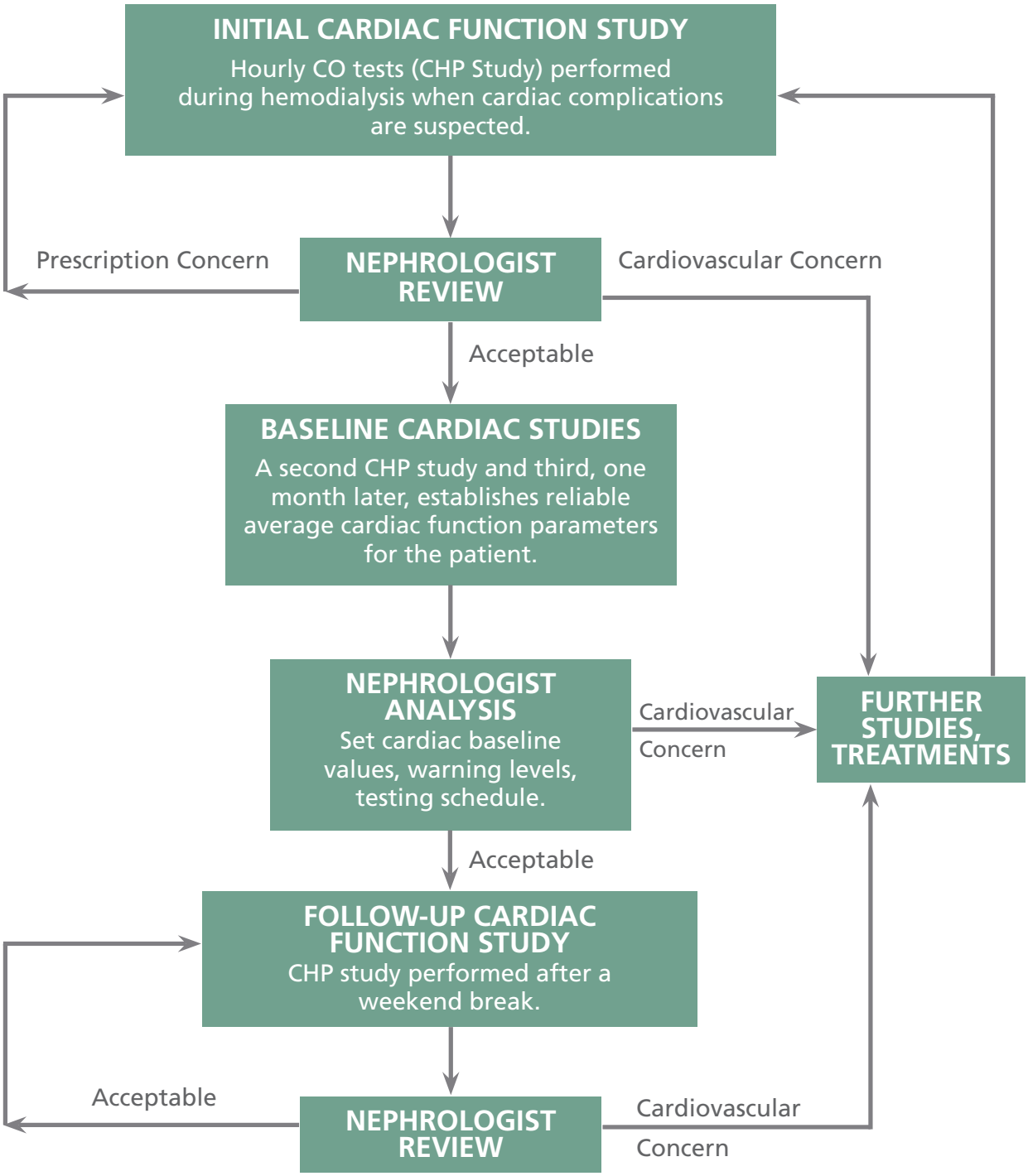
- **The first baseline CHP study performed on a stable patient (see above).**
- **A second CHP study performed shortly after the first. (One baseline study should follow a two-day dialysis break, another, after a three-day break.)**
- **A third CHP study one month later, after a weekend dialysis break, to confirm a patient's stability and serve as the third data point for the patient's cardiac function baseline.**

The nephrologist reviews the baseline study results, assesses the patient's status and prescribes a follow-up monitoring program.

FOLLOW-UP CARDIAC STUDIES

Follow-up studies serve to monitor any progression of cardiovascular disease. A follow-up study consists of periodic CHP, preferably after a weekend break. The Flow-QC Protocol recommends quarterly testing for ESRD patients whose cardiovascular condition is stable and more frequent testing for patients with cardiovascular complications.

Cardiac Function Study Protocol cont.



Cardiac Function Case Studies

High Access Flow & Potential Cardiac Overload

A patient complaining of chest pains had 3630 mL/min AV fistula flow (Fig. 8), which prompted a CO measurement. CO was 10.8 L/min (Fig. 9). The vascular access was briefly occluded with a finger, and the patient's pulse rate dropped from 112 to 88 beats per min. An X-ray identified cardiomegaly. The vascular access was banded. Following banding, access flow measured 1700 mL/min and CO dropped to 7-8 L/min. The patient exhibited fewer post-dialysis hypotensive episodes, his dry weight decreased, his chest X-ray cleared and he reported significant improvement in his previous symptoms.

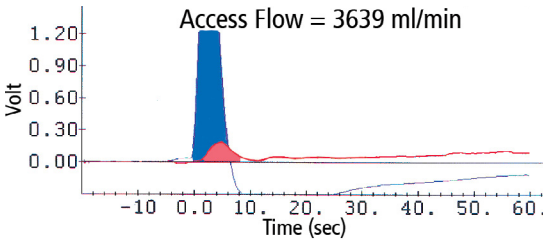


Fig. 8: Access flow measured over 3.6 L/min which prompted a CO measurement.

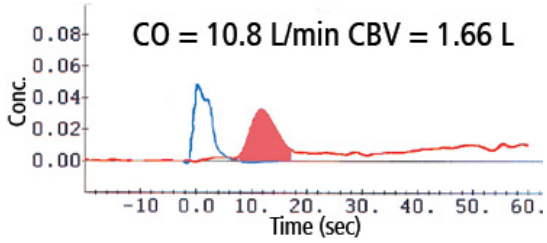


Fig. 9: Access flow measured over 3.6 L/min which prompted a CO measurement.

Deterioration of Cardiac Output & Cardiac Index during Hemodialysis

Flow-QC® Cardiac Function screening commenced 40 minutes into the hemodialysis session for a patient with ischemic heart disease. The first CO measurement was 4.3 L/min with a CI of 2.5 (Fig. 10). When the test was repeated two hours later, the patient's CO had dropped to 2.7 L/min and his CI was 1.6. The nephrologist was alerted, the patient's hemodialysis prescription was adjusted, and his cardiac condition was closely monitored.

Case studies courtesy of Dr. T.A. Depner, University of CA at Davis

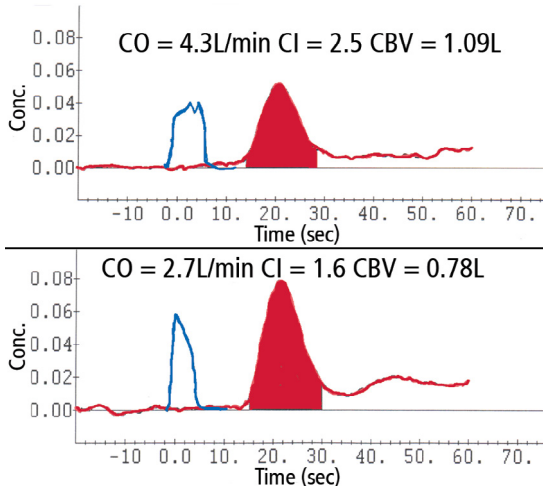


Fig. 10: Flow-QC® software screens showing deterioration of cardiac function during the course of the hemodialysis session.

Conclusion

Your hemodialysis patients do not have to be at risk for CVD or other cardiac episodes once they leave their dialysis sessions. To provide the best patient care, cardiac function screening should be added to hemodialysis treatments. The awareness of cardiac function coupled with the right technology can make providing exceptional hemodialysis patient care a reality.

START PROVIDING BETTER PATIENT CARE TODAY.
Request a demo of the technology that makes it possible.

REQUEST A DEMO

References

1. Tucker T et al, "Unrecognized Deterioration of Cardiac Function during Hemodialysis," J Am Soc of Nephrol Abstracts 2002; 13: 213A. (Transonic Reference # HD267A).

2. Cardiovascular Disease — An ESRD Epidemic. Am J Kid Dis 1998; 32(5): Suppl 3.

3. MacRae JM et al, "The Cardiovascular Effects of Arteriovenous Fistulas in Chronic Kidney Disease: A Cause for Concern?" Sem in Dialysis 2006; 19(15): 349-352. (Transonic Reference # HD7337A)

4. Locatelli F et al, "Cardiovascular Disease in Chronic Renal Failure; the Challenge Continues," Nephrol Dial Transplant 2000; 15(Suppl 5): 69-80. (Transonic Reference # HD9643R)

5. Krivitski NM, Depner, TA, "Cardiac Output and Central Blood Volume during, Hemodialysis: Methodology," Adv Ren Replace Ther 1999; 6(3): 225-232. (Transonic Reference # HD8T)

6. Bleyer AJ et al, "The Timing and Characteristics of Sudden Death in Hemodialysis Patients" J Am Soc Nephrol 2002; 13: SU-PO737.

7. Depner TA, Krivitski NM, "Central Blood Volume: A New Criterion for Predicting Morbid Events during Hemodialysis," J Am Soc of Nephrol Abstr 1996; 7(9) 1511. (Transonic Reference # HD15A)

8. Depner TA, "Cardiac Output, Peripheral Resistance, and Central Blood Volume in Hemodialyzed Patients: Correlations with Clinical Status," Satellite Presentation ASN 1998. (Transonic Reference # VP-17)

9. MacRae JM, "Vascular Access and Cardiac Disease: Is There a Relationship? Curr Opin Nephrol Hypertens 2006; 15(6): 577-82. (Transonic Reference # HD7382A)

10. MacRae JM et al, "Arteriovenous Fistula-associated High-output Cardiac Failure: A Review of Mechanisms, "Am J Kidney Dis 2004; 43(5): 17-22. (Transonic Reference # HD408A)

11. Tucker T et al, "Central Hemodynamic Profiling (CHP) during Outpatient Hemodialysis (HD)," J Am Soc of Nephrol Abstr 2002; 13: 209A. (Transonic Reference # HD268A)

12. Krivitski NM, "Novel Method to Measure Access Flow during Hemodialysis by Ultrasound Dilution Technique," ASAIO J 1995; 41: M741-M745. (Transonic Reference # HD4T)

13. Nikiforov UV et al, "Validation of a New Method to Measure Cardiac Output during Extracorporeal Detoxification," ASAIO J 1996; 42: M903-M905. (Transonic Reference # HD15V)

14. Kislouchine VV, Dean DA, "Validation of a Novel Ultrasound Dilution Method to Measure Cardiac Output during Hemodialysis," ASAIO J 1996; 42: M906-M907. (Transonic Reference # HD16V)

15. Sands JJ et al, "Access Flow Measured during Hemodialysis," ASAIO J 1996; 42: M530-M532. (Transonic Reference # HD6A)

16. Pandeya S, Lindsay RM, "The Relationship between Cardiac Output and Access Flow during Hemodialysis," ASAIO J 1999; 135-138. (Transonic Reference # HD89A)

17. Leypoldt JK, Lindsay RM, "Hemodynamic Monitoring during Hemodialysis," Adv in Ren Replacement Ther 1999; 6: 233-242. (Transonic Reference # HD239A)

18. Hoeben H et al, "Hemodynamics in Patients with Intradialytic Hypotension Treated with Cool Dialysate or Midodrine," Am J Kid Dis 2002; 39(1): 102-107. (Transonic Reference # HD249A)

19. Pandeya S, Lindsay RM, "Cardiac Output and Access Flow during Hemodialysis [HD]: Are They Related?" J Am Soc of Nephrol Abstr 1998; 9: 179A. (Transonic Reference # HD74A)

References

20. Krivitski NM, "Cardiac Output Measurement in Extracorporeal Systems by Ultrasound Velocity Dilution," ASAIO Abstracts 1994; 82. (Transonic Reference # HD3T)

21. Depner TA, Krivitski NM, "Influence of Access Blood Flow (AF) on Systemic Blood Flow in Hemodialysis Patients," J Am Soc of Nephrol Abstr 1997; 8: 155A. (Transonic Reference # HD23A)

22. Depner T et al, "Peripheral Resistance and Systemic Blood Flow in Hemodialysis Patients," J Am Soc of Nephrol Abstr 1998; 9: 170A. (Transonic Reference # HD58A)

23. Krivitski NM et al, "Measurement of Cardiac Output and Central Blood Volume during Hemodialysis: Sources of Error," 25th International Congress of Nephrology Abstract 1999; 333. (Transonic Reference HD95A)

24. Basile C, Lomonte C, Vernaglione L et al, "The relationship between flow of arteriovenous fistula and cardiac output in haemodialysis patients," Nephrol Dial Transplant 2008; 23: 282–287 (Transonic Reference HD7542A)

25. Schier T et al, "Incidence of Arteriovenous Fistula Closure Due to High-output Cardiac Failure in Kidney-transplanted Patients," Clin Transplant. 2013; 27(6): 858-65 (Transonic Reference # HD9869AHR)

26. Stern AB, Klemmer PJ, "High-output Heart Failure Secondary to Arteriovenous Fistula," Hemodial Int. 2011 Jan 12.

27. Wasse H, Singapuri MS, "High-output heart failure: how to define it, when to treat it, and how to treat it,"Semin Nephrol. 2012; 32(6): 551-7.

28. Hooper DK et al, "The quality of cardiovascular disease care for adolescents with kidney disease: a Midwest Pediatric Nephrology Consortium study," Pediatr Nephrol. 2013 Jun;28(6):939-49. Nefrologia. 2015 May-Jun;35(3):234-45. (Transonic Reference HD10619AHR)

29. Alkhouli M et al, "Cardiac complications of arteriovenous fistulas in patients with end-stage renal disease," Nefrologia 2015; 35(3): 234-45. (Transonic Reference HD10618AHR)

30. Huu, TC et al, "Access Flow (Qac) and Cardiac Output (CO) Measurements by Non-Invasive Methods in Hemodialysed Patients." Angioaccess for Hemodialysis, 2nd International Multi-disciplinary Symposium. May 31-June 2, 1999, 170. (Transonic Reference # HD108A)

31. Huu TC et al, "Detection of High Access Flow (Qac) and Cardiac Output (CO) in Hemodialysed Patients," J Am Soc of Nephrol Abstr 1999; 10: 202A. (Transonic Reference # HD129A)

32. Huu, TC et al, "Non-Invasive Measurement of Access Flow (Qac) and Cardiac Output (CO) in Hemodialysis Patients," Nephrol Hemodialy Transplant 1999; 14(9): A175. (Transonic Reference # HD34V)

33. Barril, G et al, "Vascular Access (VA) Assessment by Dilutional Methodology (Transonic QC)," J Am Soc Nephrol, 1999; 10: 201A. (Transonic Reference # HD42V)

34. Schneditz D et al, "Systematic Underestimation of Cardiac Output by Thoracic Bioimpedance in Hemodialysis Patients: Relation to Access Blood Flow," J Am Soc of Nephrol Abstr 1999; 10: 217A. (Transonic Reference # HD131A)

35. Depner TA et al,"Contraction of Central Blood Volume and Reduced Cardiac Index in Hemodialyzed Diabetic Patients," J Am Soc of Nephrol Abstr 2000; 11(3): 263A. (Transonic Reference # HD154A)

36. Dobson A et al, "Cardiac End Diastolic Blood Volume from Cardiac Output during Hemodialysis," J Am Soc of Nephrol Abstr 2000; 11(3): 264. (Transonic Reference # HD155A)

37. Kiaii M et al, "What "Blood Volume" Do We Dialyze?" J Am Soc of Nephrol Abstr 2000; 11(3): 277. (Transonic Reference # HD156A)

38. Lindsay RM et al, "Extracellular Fluid (ECF) and Blood Volume Changes during Hemodialysis [HD]," J Am Soc of Nephrol Abstr 2001. (Transonic Reference # HD213A)

39. http://www.fistulafirst.org/Professionals/FrequentlyAskedQuestions.aspx#Q5

40. Cheung AK et al, "Cardiac diseases in maintenance hemodialysis patients: results of the HEMO Study," Kidney Int. 2004; 65(6): 2380-9. (Transonic Reference # HD10620AHR)

41. Korsheed S et al, "Effects of arteriovenous fistula formation on arterial stiffness and cardiovascular performance and function. Nephrol Dial Transplant 2011; 26(10): 3296-302. (Transonic Reference # HD10622AHR)

42. Raza F, et al, "Case series of 5 patients with end-stage renal disease with reversible dyspnea, heart failure, and pulmonary hypertension related to arteriovenous dialysis access,"Pulm Circ 2015; 5(2): 398-406.

43. Basile C, Lomonte C, Konner K. "The arteriovenous fistula: lesser evil or God's blessing?" Blood Purif 2011; 32: 253.

44. Wasse H1, Speckman RA, McClellan WM, "Arteriovenous fistula use is associated with lower cardiovascular mortality compared with catheter use among ESRD patients," Semin Dial 2008; 21(5): 483-9. (Transonic Reference # HD10636AHR)

transonic
THE MEASURE OF BETTER RESULTS.

www.transonic.com

Transonic Systems Inc. is a global manufacturer of innovative biomedical measurement equipment. Founded in 1983, Transonic sells “gold standard” transit-time ultrasound Flowmeters and Monitors for surgical, hemodialysis, pediatric critical care, perfusion, interventional radiology and research applications. Transonic® also provides pressure and pressure volume systems, laser Doppler Flowmeters and telemetry systems.

Americas

Transonic Systems Inc.
34 Dutch Mill Rd
Ithaca, NY 14850
U.S.A.
Tel: +1 607-257-5300
Fax: +1 607-257-7256
support@transonic.com

Europe

Transonic Europe B.V.
Business Park Stein 205
6181 MB Elsloo
The Netherlands
Tel: +31 43-407-7200
Fax: +31 43-407-7201
europe@transonic.com

Asia/Pacific

Transonic Asia Inc.
6F-3 No 5 Hangsiang Rd
Dayuan, Taoyuan County
33747 Taiwan, R.O.C.
Tel: +886 3399-5806
Fax: +886 3399-5805
support@transonicasia.com

Japan

Transonic Japan Inc.
KS Bldg 201, 735-4 Kita-Akitsu
Tokorozawa Saitama
359-0038 Japan
Tel: +81 4-2946-8541
Fax: +81 4-2946-8542
japan@transonic.com