The arteriovenous fistula (AVF) was adopted in the clinical practice of dialysis in the 1960s without prospective randomized trials, simply on the basis of utility. It was widely hailed as a major improvement from the Scribner shunt, and is rightfully credited with allowing chronic hemodialysis in the 1960s without prospective randomized controlled trials. We must rely on our understanding of physiology, longitudinal observations of patients (i.e. natural history), logic, reason and common sense. The latter three should dictate caution. Patent ductus arteriosus (PDA) and traumatic AVFs lead to cardiac decompensation and are always closed surgically when detected. Why should dialysis patients be immune to these effects? Indeed, due to a high prevalence of underlying cardiac disease, HD patients are more susceptible to the negative consequences of AVF.

The physiology and hemodynamics of the AVF have been well described [3]. The cardiac output (CO), and thus work of the heart, increases in proportion to the size of the shunt. This usually requires an increase in the blood volume, as total peripheral resistance decreases, and this is reflected in high levels of atrial natriuretic peptide and brain natriuretic peptide. Sympathetic tone increases on a more or less permanent basis, which raises the heart rate, contractility and blood pressure over time. Cardiac remodeling results in chamber dilatation and hypertrophy [4]. Pulmonary flow increases, and in dialysis patients, this frequently leads to pulmonary hypertension [5]. The AVF decreases the subendocardial viability index by increasing the work of the heart while decreasing coronary perfusion. This sets the stage for subendocardial ischemia, myocardial stunning.

Con: On cardiovascular outcomes and the arteriovenous fistula: lesser of evils

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The arteriovenous fistula (AVF) was adopted in the clinical practice of dialysis in the 1960s without prospective randomized trials, simply on the basis of utility. It was widely hailed as a major improvement from the Scribner shunt, and is rightfully credited with allowing chronic hemodialysis (HD) to flourish as a modality [1]. But this history does not negate the fact that the AVF is a harmful, non-physiological anomaly, with considerable downsides. These are either not mentioned or downplayed in programs to increase fistula use [2]. Anointing any particular remedy in medicine is hazardous, in that it deters research into better alternatives.

I am not advocating that the AVF be banned, nor am I a catheter proponent. I wish to encourage a thoughtful, common sense, individualized approach to HD access; an approach that matches the access to the needs of each patient, and that takes into account the negative effects of the AVF. I am suggesting that emotion be removed from this process and replaced by cool, clinical judgment.

We must recognize the complete absence of any prospective, randomized outcome trial of HD access, and the extreme unlikelihood of any ever being performed. Let us also accept that observational or retrospective outcome data are inconclusive. This is not a unique situation; the majority of clinical decisions fall outside of territory covered by randomized controlled trials. We must rely on our understanding of physiology, longitudinal observations of patients (i.e. natural history), logic, reason and common sense. The latter three should dictate caution. Patent ductus arteriosus (PDA) and traumatic AVFs lead to cardiac decompensation and are always closed surgically when detected. Why should dialysis patients be immune to these effects? Indeed, due to a high prevalence of underlying cardiac disease, HD patients are more susceptible to the negative consequences of an AVF.

The physiology and hemodynamics of the AVF have been well described [3]. The cardiac output (CO), and thus work of the heart, increases in proportion to the size of the shunt. This usually requires an increase in the blood volume, as total peripheral resistance decreases, and this is reflected in high levels of atrial natriuretic peptide and brain natriuretic peptide. Sympathetic tone increases on a more or less permanent basis, which raises the heart rate, contractility and blood pressure over time. Cardiac remodeling results in chamber dilatation and hypertrophy [4]. Pulmonary flow increases, and in dialysis patients, this frequently leads to pulmonary hypertension [5]. The AVF decreases the subendocardial viability index by increasing the work of the heart while decreasing coronary perfusion. This sets the stage for subendocardial ischemia, myocardial stunning.
and eventual cardiomyopathy, which is the end stage of AVF-related cardiac toxicity [6]. Along the way, the initially elevated CO normalizes, which I refer to as pseudo-normalization. These patients already have a decreased ‘effective’ CO, which is the total output minus the AVF flow. Thus, measurements of CO in dialysis patients which do not take into account the AVF flow are meaningless.

The AVF is solely responsible for cardiopulmonary re-circulation (CPR), which limits HD efficiency and may cause under-dialysis. CPR increases with larger AVF that drain a greater percentage of CO [7]. And the AVF tends to dilate, with increased flow, over time.

The natural history of the AVF is readily discerned from observations of patients with traumatic AVF or PDA, and from longitudinal studies of dialysis patients. Several studies have documented cardiac chamber enlargement with AVF creation and shrinkage after AVF closure [8]. In our own experience, we have seen AVF patients who develop cardiac decompensation and/or pulmonary hypertension and improve, often dramatically, after AVF reduction or closure [9, 10]. The creation of a high-flow AVF (>1 L/min) will eventually lead to heart failure, even in a healthy individual. This is one reason why few HD patients survive >20 years, unless they are transplanted. I also believe, although cannot prove, that the use of smaller AVFs in Japan and Europe at least partially explains their superior survival rates [11].

Central venous catheters (CVCs) have their share of problems, including infection and central vein stenosis. A complete discussion of the CVC in HD is beyond the scope of this essay. Suffice it to say that we can do a better job in preventing catheter-related infections through the use of aseptic techniques, and antimicrobial locking solutions [12]. This would be more productive than the constant denunciation of CVCs, which is particularly unfortunate for those HD patients in whom they represent a lifeline.

A rational approach to HD access should take into account individual patient factors and should strive to limit exposure to the toxic effects of both CVCs and AVFs. As patients approach end-stage renal disease, every effort should be made towards pre-emptive transplantation, as this provides the best survival and quality of life [13]. Peritoneal dialysis should be offered as a first modality in most patients. For patients choosing HD, cardiac screening should be done. Those with compromised function (ejection fraction <35%, severe coronary artery disease) will likely do badly with an AVF and should be considered for a CVC. Very elderly patients, those with limited life expectancy, anticipated recovery of renal function (post-acute tubular necrosis, etc.), or those with anticipated live donor transplantation, should be dialyzed via CVCs. The optimal timing for placement of an AVF pre-HD has not been determined, and should be individualized. Three months before anticipated start of HD should be adequate. It is unreasonable to place an AVF in a patient with Stage IV chronic kidney disease, as it might be unused for years. Surgeons should attempt to create AVFs with flow rates of 400–800 mL/min. Anything above this is unnecessary and harmful. Grafts may be helpful in this regard, as they are less likely to dilate [14]. Fistula flow rates should be monitored on a regular basis. Those with high flow (>1.5 L/min) should have routine echocardiography. Flow reduction should be performed for any sign of decompensation or significant pulmonary hypertension. This procedure carries a risk of shunt failure. AVFs should be ligated 1 year after successful transplantation, unless there is a high likelihood of graft failure. Finally, HD patients with severe cardiomyopathy should be considered for AVF reduction or ligation [15].

The AVF is a useful device, but is not benign and certainly is not beneficial (other than as HD access). Its only indication is chronic HD where it is at best the lesser of evils. There is no optimal rate of AVF use, and some patients require CVCs. Thus, central planning efforts to increase AVF use via ‘payment for performance,’ or other coercive measures, should be discontinued. We should not abandon the search for an improved blood access system, as this remains the Achilles’ heel of HD.

References


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