Clinical and Haemodynamic Effects of Arteriovenous Shunts in Patients with Heart Failure with Preserved Ejection Fraction

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Abstract

The arteriovenous shunt (AVS) is the most commonly used vascular access in patients receiving regular haemodialysis. The AVS may have a significant haemodynamic impact on patients with heart failure. Many studies have sought to understand the effect of AVS creation or closure on heart structure and functions, most of which use non-invasive methods, such as echocardiography or cardiac MRI. Data are mainly focused on heart failure with reduced ejection fraction and there are limited data on heart failure with preserved ejection fraction. The presence of an AVS has a significant haemodynamic impact on the cardiovascular system and it is a common cause of high-output cardiac failure. Given that most studies to date use non-invasive methods, invasive assessment of the haemodynamic effects of the AVS using a right heart catheter may provide additional valuable information.

Keywords
Arteriovenous shunt, echocardiography, heart failure, heart failure with preserved ejection fraction

Chronic kidney disease (CKD) is a worldwide public health problem. The overall prevalence of CKD in the US adult population was 14.8%, using an estimated glomerular filtration rate of <60 ml/min/1.73 m² as a definition for CKD. The prevalence of end-stage renal disease (ESRD) continues to increase. According to the US Renal Data System, the incidence rate is 357 per million per year. Of these ESRD patients, 63% were receiving haemodialysis, 7% peritoneal dialysis and 29.6% had a functioning kidney transplant.

Patients with ESRD need long-term vascular access for haemodialysis. The most commonly used vascular access is the arteriovenous shunt (AVS). The AVS is a connection between the arterial and venous systems created either using an anastomosis between a limb artery and superficial native vein (arteriovenous fistula, AVF) or insertion of graft (arteriovenous graft) as dialysis access, creating a left-to-right shunt.

The presence of an AVS has a significant haemodynamic impact on the cardiovascular system – both short- and long-term. It is a common cause of high-output cardiac failure. The mechanism underlying this haemodynamic effect is based on shunting blood from a high-pressure artery via the AVF to a low-pressure vein, thus bypassing capillary beds and decreasing systemic vascular resistance (SVR). These haemodynamic changes stimulate a compensatory increase in heart rate, stroke volume and total plasma volume. The elevation in cardiac output (CO) associated with AVS depends upon the size of the shunt and the magnitude of the resultant reduction in SVR. Because blood flowing through the shunt bypasses the capillary circulation, the total CO increases by the quantity of blood flowing through the shunt to maintain capillary perfusion.

In high-output heart failure (HF), low SVR results in borderline preserved or depressed systemic arterial blood pressure and elevated cardiac filling pressures. Ineffective blood volume and pressure lead to activation of the sympathetic nervous system and the renin–angiotensin–aldosterone axis along with increased serum vasopressin (antidiuretic hormone) concentrations. This neurohormonal activation results in increased renovascular resistance and reduced renal blood flow and glomerular filtration rate, with retention of salt and water. Chronic volume overload may gradually cause ventricular enlargement, remodelling and HF.

Definition of Heart Failure with Preserved Ejection Fraction

HF with preserved ejection fraction (HFpEF) is a clinical syndrome in patients with current or prior symptoms of HF with a left ventricular ejection fraction (LVEF) ≥50% and evidence of cardiac dysfunction as a cause of symptoms (e.g. abnormal LV filling and elevated filling pressures). Patients with HFpEF represent half of all HF patients worldwide. The remaining half have an LVEF <50%, which includes HF with reduced ejection fraction (HFrEF; LVEF ≤40%) and HF with mid-range ejection fraction (LVEF 41–49%).

History of the Arteriovenous Fistula and Relation to Heart Failure

The AVF was first described and used as a reliable form of haemodialysis...
Conversely, acute compression of AVS increases the SVR and the higher risk 22
of precipitating HF was much higher in patients who had a brachiocephalic AVF
compared with those with a radial-cephalic AVF (40% versus 8%). Similar
rates of HF have been observed among patients with AVFs compared with
those with arteriovenous grafts.9 There are no data to assess whether the
technique used to create the AVF (direct versus through translocation or
transposition) has any relation with the development of HF.

Haemodynamic Changes After Arteriovenous Shunt Creation

The creation of AVS results in acute, sub-acute and chronic cardiovascular
changes.

Acute Changes

Acute effects of AVS creation include an immediate decrease in SVR and
consequent increases in forward stroke volume, heart rate and CO. The
decline in total SVR is the result of both changes in the vessels
associated with the arteriovenous access (called access resistance) and
changes in other systemic vessels. In response to increases in blood flow
and shear stress, the vascular endothelium releases nitric oxide and other
endothelium-dependent relaxing factors that dilate the artery, reducing
shear stress towards normal.5,18

The decrease in SVR causes an acute fall in both central and peripheral
blood pressure. In response, there is an increase in sympathetic nervous
system activity (which increases contractility and heart rate). It is this
combination of decreased cardiac afterload and increased sympathetic
activation that causes acute increases in CO.5 The CO increases
immediately upon creation of the AVS and continues to increase over time.9,20 This increase in CO leads to an increase in venous return to the
right side of the heart, leading to right ventricular dilatation in some
patients.9 Conversely, acute compression of AVS increases the SVR and
blood pressure and decreases CO. The increase in blood pressure leads to
baroreceptor-reflex-mediated reduction in heart rate (Nicoladoni-
Branham’s sign; Figure 1).

Subacute and Chronic Changes

Subacute changes occur within days after creation of the AVS. Within 2
weeks of AVS creation, blood volume increases, leading to greater
venous return and increased right atrial, pulmonary artery and LV end-
diastolic pressures. Both plasma atrial natriuretic peptide and brain
natriuretic peptide concentrations increase after AVS creation, peaking 10
days postoperatively.11,15 CO continues to increase over days and weeks
after creating the AVS.9,20

Many studies have sought to understand the effect of AVF creation or
closure on heart structure and function. Most use non-invasive methods
— mainly echocardiographic parameters — while others use Doppler
ultrasound to assess AVF flow and its effect on LV parameters. One study
used cardiac MRI as an accurate non-invasive tool for the assessment of
cardiac functions and dimensions.4 In 2018, Saleh et al. published a study
investigating patients with AVFs and HF.17 The study showed that higher
AVF flow was associated with an increased risk of high-outflow HF (HOHF).
Furthermore, the study demonstrated a strong relationship between the
vascular access by Brescia et al. in 1966.11 Improvements in dialysis
technology and the expansion of dialysis eligibility (for example the
inclusion of patients with diabetes) resulted in rapid growth of the ESRD
population. Many of these patients benefited from the development of
prosthetic grafts when autogenous AVFs were not feasible.

In the mid-1980s, the use of permanent catheters (central venous
catheters; CVCs) in the internal jugular vein dramatically increased. The
cumulative effect was a decrease in AVF use and an increase in graft and
CVC use in the 1990s.12 This was associated with increased patient care
costs, for example up to 73% of patients were hospitalised to initiate
dialysis and almost invariably had a temporary CVC inserted.12,13 This led
the Centers for Medicare and Medicaid Services and National Kidney
Foundation in the US implementing in 2003 the Fistula First Initiative to
increase AVF placement and use to 65% along with lowering costs.14 AVF
use is still the best choice for dialysis access in terms of patient outcomes/
 survival and reducing health care cost, but the approach can be associated
with complications.

Incidence of Heart Failure Post-arteriovenous Shunt

Studies have shown that an estimated 17–26% of patients with a
functioning AVS develop symptoms of HF.14,15 Factors associated with AVS
precipitating HF include development of right ventricular dilatation, left
atrial dilatation, development of AF, male sex, prior vascular access surgery
and high haemodialysis arteriovenous access flow rate.15 The risk of
worsening HF is directly proportional to the flow of the haemodialysis
arteriovenous access and is greater with pre-existing poor cardiac
function.17 There is no threshold access flow rate that defines risk. Even
what is considered to be a normal flow may worsen or precipitate HF in
patients with pre-existing HF or heart disease.15

The risk of precipitating HF appears to be higher among patients who
have an upper-arm AVF compared with forearm AVF.14,15 The higher risk
associated with upper-arm AVFs appears to be related to higher blood
flow. In an observational study of 562 pre-dialysis patients, the incidence
of HF was much higher in patients who had a brachiocephalic AVF
compared with those with a radial-cephalic AVF (40% versus 8%).16 Similar
rates of HF have been observed among patients with AVFs compared with
those with arteriovenous grafts.9 There are no data to assess whether the
technique used to create the AVF (direct versus through translocation or
transposition) has any relation with the development of HF.
### Table 1: Summary of Key Studies Evaluating Effects of Arteriovenous Shunt on the Heart

| Authors                  | n   | Aims                                                                 | Methods                                                                 | Assessment Method                                                                 | Results                                                                 | Conclusions                                                                                                                                 |
|--------------------------|-----|----------------------------------------------------------------------|-------------------------------------------------------------------------|-----------------------------------------------------------------------------------|--------------------------------------------------------------------------|-------------------------------------------------------------------------------------------------------------------------------------------|
| Saleh et al.²²           | 100 | Effect of high flow AVF on HF patients                               | Two groups of patients:                                                 | Echo at baseline and after closure of AVF                                          | HFA group showed significant increase in LV and LA volumes compared to non-HFA group | HFA was associated with dilated LV dimensions, impaired LV systolic function                                                          |
|                          |     |                                                                      | • HFA group, Qa >2,000 m/min                                            | US Doppler for Quantification of AVF flow (Qa)                                   | Significant association between high Qa/QO ratio (≥20%) and HOHF           | High Qa/QO ratio (≥20%) was an independent predictor of HOHF                                                                       |
|                          |     |                                                                      | • Non-HFA group, Qa ≤2,000 m/min                                         |                                                                                  |                                                                          |                                                                                                                                            |
| Glowiński et al.²³      | 18  | Effect of AVF closure on heart functions in patients after kidney transplantation | Nine patients after closure of AVF compared to nine patients with patent AVF | Echo baseline and 3 months after AVF closure                                        | Echo did not reveal any significant differences compared to baseline examination | AVF closure does not seem to have a beneficial effect on cardiac function during short-term follow-up                                       |
|                          |     |                                                                      | Patients did not have HF                                                 |                                                                                  |                                                                          |                                                                                                                                            |
| Movili et al.²⁰         | 61  | Evaluate the effect of AVF closure on heart function and structure by Echo | 25 patients underwent AVF closure-matched with 36 patients with well-functioning AVF | Echo at baseline and 6 months after AVF closure                                   | In the AVF-closure group, LVM decreased                                    | AVF closure resulted in significant decrease in LV internal diastolic diameter, IVS and PW thickness with significant improvement in LVEF and significant decrease in LVM |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
| Iwashima et al.²⁴       | 16  | Serial changes in cardiac functions and hormonal levels after the AVF creation | Echo before and 3, 7, and 14 days after AVF creation                      |                                                                                  |                                                                          | Ave closure has significant effects on cardiac systolic and diastolic performance, and ANP release, induced by volume loading. ANP release is stimulated by LV diastolic dysfunction |
|                          |     |                                                                      | ANP and BNP concentrations were measured before and 1, 3, 6, 10, and 14 days after the operation |                                                                                  |                                                                          |                                                                                                                                            |
| Rao et al.²⁵            | 54  | Effect of AVF closure in patients 12 months post-KT                   | 27 patients underwent AVF closure and 27 are the control group.          | Cardiac MRI, Echo and NT pro-BNP before and 6 months after AVF closure            | AVF closure group showed a decrease in LVM compared to a small increase in the control group. Significant decreases in LV EDV and ESV, CI and NT-pro BNP | Electro ligation of patent AVF in adults with stable KT resulted in clinically significant reduction of LV myocardial mass                      |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
|                          |     |                                                                      | Randomised controlled trial                                              |                                                                                  |                                                                          |                                                                                                                                            |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
| Unger et al.²⁶          | 16  | Effects of AVF closure on ABPM and on LV geometry                    | AVF closure in patients with stable KT, studied before and 1 month after AVF closure by Echo, ABPM, Qa | Echo, ABPM, Qa at baseline and 1 month after AVF closure                          | Increase in the mean DBP without significant change in SBP. The increase in DBP correlated with a reduction in LVM | AVF closure induces an increase in DBP correlated with the reduction in LVM                                                        |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
| Cridlig et al.²⁷        | 76  | Effect of persistent AVF in patient post-KT and without previous cardiovascular disease | 38 patients with a functioning AVF and a matched group with no AV         | 76 Patients underwent Echo for assessment of LVMI, LVH                             | Patients with AVF have significantly higher LVM and higher LVH LVM is higher with higher Qa. Also, LV EDD, ESD are larger in those patients | Persistent functioning AVF resulted in significant increase in cardiac dimensions, LVH and LVMI                                      |
|                          |     |                                                                      |                                                                           |                                                                                  |                                                                          |                                                                                                                                            |
| Gumus et al.²⁸          | 81  | Effect of AVF creation on right ventricle functions. Identify new parameters can contribute to the prediction of RVF after AVF creation | 81 patients underwent AVF creation divided into two groups: patients with RVF (18.5%) and without RVF (72.5%) | Echo assessment of right ventricle functions including RVLS, TAPSE, RV FAC, TRJV | Increase risk of development of RVF After AVF creation                          | Independent predictors of developing RVF following AVF creation are RVLS free wall ≥14.2% and TRJV ≥2.61 m/s                                  |

**Abbreviations:**
- ABPM = ambulatory 24 hours blood pressure monitoring
- ANP = plasma atrial natriuretic peptide
- AVF = arteriovenous fistula
- BNP = brain natriuretic peptide
- CI = cardiac index
- CO = cardiac output
- DBP = diastolic blood pressure
- Echo = echocardiogram
- HF = heart failure
- HFA = high-flow access
- HOHF = high-output heart failure
- IVS = left ventricle interventricular septum
- KT = kidney transplantation
- LA = Left atrium
- LV = left ventricle
- LVDD = left ventricle diastolic diameter
- LVEDD = left ventricle end diastolic volume
- LVESV = left ventricle end systolic volume
- LV EDD = left ventricle end diastolic volume
- LVEDV = left ventricle end diastolic volume
- LVM = left ventricular mass
- LVMi = left ventricular mass index
- NT-proBNP = N-terminal pro-brain natriuretic peptide
- PA = pulmonary artery
- PW = left ventricle posterior wall
- Qa = amount of blood flow across the AVF measured by ultrasound Doppler
- RA = right atrium
- RV = right ventricular failure
- RVFAC = right ventricle fractional area change
- RVLS = right ventricle longitudinal strain
- SBP = systolic blood pressure
- TAPSE = tricuspid annular plane systolic excursion
- TRJV = tricuspid regurgitation jet velocity
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Cardiac Follow-up After Creation of an Arteriovenous Shunt

Evaluation of all patients following an AVS includes an evaluation for HF. All patients who undergo access placement have markedly reduced kidney function and are at risk for HF. Patients who are at particular risk to develop HF related to the arteriovenous access include those with a large distended AVS, especially in the upper-arm position.

Table 2: Summary of a Suggested Non-invasive Approach to Follow Up Patients After Arteriovenous Shunt Creation and Closure

| Investigation | Baseline | Follow-up After 3–6 Months |
|---------------|----------|---------------------------|
| NT-proBNP     | Baseline before procedure | Follow-up after creation |
| Ultrasound Doppler | Quantification of AVS flow (Qa) | Follow-up AVS flow (Qa) |
| Echocardiography | With following measurements: LVEDV, LVESV, LVEF, LAVI, TAPSE, RV FAC, RVLS, TRJV, RVEF, RAVI, IVC, PASP | Suggested predictors of worsening heart functions: |

- High Qa/CO ratio (≥20%) predicts development of HOHF
- Independent predictors of developing RVF following AVS creation are RVLS free wall ≤14.2% and TRJV >2.61 m/s²

AVS = arteriovenous shunt; HOHF = high-output heart failure; IVC = inferior vena cava diameter; LAVI = left atrial volume index; LVEDV = left ventricle end diastolic volume; LVEF = left ventricular ejection fraction; LVESV = left ventricle end systolic volume; NT-proBNP = N-terminal pro-brain natriuretic peptide; PASP = pulmonary artery systolic pressure; Qa = amount of blood flow through AVS by ultrasound Doppler; Qa/CO = ratio of blood flow through AVS by ultrasound Doppler and cardiac output estimated by echo; RAVI = right atrium volume index; RV FAC = right ventricle fractional area change; RVEF = right ventricular ejection fraction; RVF = right ventricular failure; RVLS = right ventricle longitudinal strain; TAPSE = tricuspid annular plane systolic excursion; TRJV = tricuspid regurgitation jet velocity

Monitoring Strategies

Patients should be followed for signs and symptoms of HF as a routine part of every visit to determine whether HF is present. An echocardiogram should be obtained when any new signs or symptoms suggestive of cardiac dysfunction develop, and follow-up echocardiography 3–6 months after creation of the AVS is also recommended. Echocardiographic findings suggesting the development of HF include dilatation of the inferior vena cava, new right ventricular dilation or dysfunction and increasing estimated pulmonary artery pressures.

Some studies have suggested assessing the cardio-pulmonary recirculation value, which is the ratio of arteriovenous access flow (Qa) to the CO in patients with arteriovenous access flow >2 l/min. A Qa/CO ratio >0.3 indicates a significant risk of developing high-output cardiac failure. However, a Qa/CO ratio ≤0.3 or a Qa ≤2 l/min does not exclude access-related HF.

Examination and Transient Occlusion of the Arteriovenous Shunt

The presence of a large, distended AVS with very strong pulse augmentation suggests high volume flow and should prompt an evaluation to determine effect of the access on systemic haemodynamics. When the AVS is transiently occluded, the degree of the arterial pulse increase (augmentation) distal to the AVS anastomosis is proportional to the AVS flow.

Right Heart Catheterisation in Patients with Arteriovenous Shunt

Among patients with AVS, the contribution of the AVS to pulmonary artery hypertension can be initially assessed by manually compressing the AVS under heparinisation and a tourniquet set to at least 30 mmHg above systolic blood pressure for 1 minute, while measuring pulmonary haemodynamics by right heart catheterisation at rest and with transient fistula occlusion can be helpful. This approach allows the definitive assessment of volume status, direct determination of CO and pulmonary artery pressures and examination of the haemodynamic response to transient fistula occlusion.

For patients with an AVS who have new or worsening HF with supportive findings on echocardiography, invasive evaluation of cardiac haemodynamics by right heart catheterisation at rest and with transient fistula occlusion can be helpful. This approach allows the definitive assessment of volume status, direct determination of CO and pulmonary artery pressures and examination of the haemodynamic response to transient fistula occlusion.

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Transient maximal occlusion (sphygmomanometer inflated to 50 mmHg above systolic pressure for 30 seconds) of a haemodynamically significant arteriovenous access usually decreases heart rate, raises arterial pressure, and lowers venous pressure; this has been termed the Nicoladoni-Branham sign. The Nicoladoni-Branham sign has been shown to be related to arterial baroreceptor activation and increased arterial baroreflex sensitivity. In addition to a decrease in heart rate, there is also an increase in arterial blood pressure and increase in SVR, lead to a decrease in CO. Presence of a Nicoladoni-Branham sign was found to be predictive of reduction in LV hypertrophy after AVF ligation.

References:

1. Nicoladoni-Branham sign. The Nicoladoni-Branham sign has been shown to be related to arterial baroreceptor activation and increased arterial baroreflex sensitivity. In addition to a decrease in heart rate, there is also an increase in arterial blood pressure and increase in SVR, lead to a decrease in CO. Presence of a Nicoladoni-Branham sign was found to be predictive of reduction in LV hypertrophy after AVF ligation.

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haemodynamics on right heart catheterisation. If a significant component of the patient’s pulmonary artery hypertension is related to the AVS, the mean pulmonary artery pressure, right atrial pressure, and possibly the pulmonary capillary wedge pressure and LV end diastolic pressure will significantly decrease (and even normalise) by at least 20% when the arteriovenous access is compressed. However, the definition of what constitutes a significant decrease is not established and is highly subjective. There may be some concern that such compression will lead to thrombosis of the access, particularly if the access is an arteriovenous graft. However, in practice, it is much harder to thrombose an arteriovenous access with manual compression than one would expect.

Management of HFpEF in Patients with Arteriovenous Shunt

In patients with AVS-related HF, management begins with control of volume status with diuresis and diuretics, correction of anaemia, treatment of hypertension and pharmacological management of HF. If HF remains uncontrolled despite medical therapy, the following approach is suggested:

1. Close any unused AVS. If the patient has more than one arteriovenous access, one should be closed immediately if it is thought to be contributing, with preservation of the shunt with the best blood flow. The patient’s clinical status should then be reassessed.

2. If refractory HF persists with absence of an unused AVS, reduce blood flow of the AVS as close as possible to minimum volume flow necessary for adequate dialysis (600 ml/min). Several different surgical techniques have been used to reduce AVF flow. The goal of surgery is to reduce fistula blood flow while maintaining sufficient flow for adequate dialysis. These techniques have included access banding and plication or distalisation of the anastomosis to a smaller artery. In one study of 12 patients with a high-flow AVF and clinical signs of high-output HF, a precision banding procedure was effectively used for access flow reduction. Adequacy of access flow restriction was evaluated intraoperatively using ultrasound flow measurements, adjusting the banding diameter in 0.5 mm increments to achieve the targeted AVF flow. Mean access flow was reduced to a mean of 598 ml/min (481 to 876) after banding. The clinical signs of HF disappeared, and AVFs remained patent in all patients. Two patients had renal transplant failure and later successfully used the AVF. Follow-up post banding was 1–18 months (mean = 12).

3. If refractory HF persists, occlude the AVS. If the approach defined above is ineffective, the AV should be occluded and replaced with a tunneled catheter or small graft since the resistance is higher in grafts than greatly diluted fistulas. Peritoneal dialysis may also be an option among some patients.

Conclusion

The presence of AVS in ESRD patients carries a significant impact on cardiac functions, especially in patients with reduced cardiac reserve (HFpEF or HFpEF). It can precipitate HF decompensation in the short term or long term. The available data on the effect of AVS creation on worsening of HF are limited, with most focused on HFpEF and conducted using non-invasive imaging techniques such as echocardiography or cardiac MRI. Using right heart catheterisation – the gold standard for assessment of haemodynamics and intracardiac pressures – to evaluate the haemodynamic effects of AVS creation or closure may provide more valuable information.
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