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Editorial Review

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What Did We Learn about VADs in 2022?

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Abstract

This is our 9th annual literature review on mechanical circulatory support devices. Our previous reports were well received by the readers.[1-8]

In this paper, we summarized the most interesting and important, from our standpoint, publications from 2022. There may be some slight overlap with the end of 2021 because some papers were published online first, and the year of the publication changed when they became available in print.

For the sixth time, we wrote a section on extracorporeal membrane oxygenation (ECMO) which primarily addresses new developments in veno-arterial ECMO.

Readers who wish to supplement this review, argue with the author's statements or express their opinions are encouraged to do so by sending letters to the editor at mguglin@iu.edu.

Outcomes

The Interagency Registry for Mechanically Assisted Circulatory Support (INTERMACS) 2021 Annual Report (12th) reviewed the outcomes of 26,688 patients undergoing primary continuous flow left ventricular assist device (LVAD) implantation between 2010-2020.[9] The study population represents an approximately equal mix of centrifugal and axial devices. Despite the vast majority of LVADs being implanted in older, sicker, and transplant-ineligible patients, there were continued improvements in patient outcomes across categories; the median time on pump support was approximately five years. The 1-year, 5-year, and 10-year survivals were 81.9%, 44.2%, and 16.8%, respectively (the latter is a Kaplan-Meier estimate). There was also a gradual improvement in complication rates.[9]

The report focused on the role of hospital readmission in patients with LVADs. Patients without readmission in the first six months had the largest survival advantage in each successive year. The estimated 5-year survival for patients on LVAD therapy without 6-month readmission was 51% compared to 30% in those readmitted 3 or more times during the first 6 months.[9]

The report recognized the impact of the 2018 change on the allocation policy. Table 1 details the status changes that were implemented. In the previous allocation system, stable LVAD patients were afforded a status of 1B, which accounted for 28.3% of all heart transplants. The current six-tiered heart allocation system assigns patients with LVADs to status 4. Currently, status 4 accounts for 18.3% of all transplants.

Table 1. Impact of 2018 transplant allocation policy update. Table is adapted from a previous publication.[5] *Abbreviations: Biventricular Assist Device (BiVAD), Left Ventricular Assist Device (LVAD)*

	Status Prior to 2018	**Status After 2018**
Extracorporeal Membrane Oxygenation	1A	1
Intra-aortic Balloon Pump	1A	2
Inpatient total artificial heart	1A	2
Non-dischargeable BiVAD or Right Ventricular Assist Device	1A	1
Acute Percutaneous Endovascular Circulatory Support Device	1A	2
Mechanical Circulatory Support With Device Malfunction/Mechanical Failure	1A	2
Dischargeable BiVAD or Right Ventricular Assist Device	1B	2
Uncomplicated LVAD or BiVAD for 30 days with arbitrary timing	1A	3
Mechanical circulatory support with significant device-related complications (thromboembolism, device infection, pump thrombosis, bleeding)	1A	3
Mechanical Circulatory Support with life-threatening ventricular arrhythmias	1A	1
Uncomplicated LVAD or BiVAD, outside of 30 days	1B	4

Also, a growing proportion of patients are listed for transplantation at status 1-3, reducing transplant access for patients with LVADs. These changes in the allocation priority resulted in fewer patients receiving LVADs with the bridge to transplant (BTT) strategy in mind. The BTT indication, which previously accounted for 51% of all device implants in 2017, now accounts for only 22% of all device implants. In 2020, 78% of LVADs were implanted as destination therapy (up from 49.5% in 2017).[9]

The Society of Thoracic Surgeons Intermacs 2022 Annual Report (13th) was also published this year[10] and highlighted outcomes for 27,314 patients receiving continuous flow durable LVADs over the last decade (2012-2021). In 2021, 2,464 primary LVADs were implanted, representing a 23.5% reduction in the annual volume compared to peak implantation in 2019 and an ongoing trend from the previous year. This decline likely reflects the untoward effects of the COVID-19 pandemic and the 2018 change in the US heart transplant allocation system. The proportion of eligible patients receiving a transplant has declined from 56.5% to 46.0% at 3 years, while the proportion remaining alive with ongoing support has improved from 24.1% to 38.1% at 3 years.[10]

HeartMate 3

HeartMate3 (HM3) (Abbott) had a monopoly among newly implanted pumps in 2022. As we learn more, it is evident that the monopoly is largely due to the advantages of the HM3, making it the best clinically performing LVAD to date.

The most remarkable publication of 2022 was the analysis of the 5-year outcomes of the Multicenter Study of MagLev Technology in Patients Undergoing Mechanical Circulatory Support Therapy With HeartMate 3 (MOMENTUM 3) cohort.[11] The study compares patients with HM3 and HeartMate II (HMII, Abbott) devices. The 5-year Kaplan-Meier estimate of survival to transplant, recovery, or LVAD support free of debilitating stroke or reoperation to replace the pump was 54.0% and 29.7% (HM3 versus HMII, hazards ratio [HR] 0.55; 95% confidence interval [CI]: 0.45-0.67; P < .001). Overall the Kaplan-Meier survival was 58.4% in the centrifugal-flow group versus 43.7% in the axial-flow group (HR 0.72; 95% CI: 0.58-0.89; P = .003). Serious adverse events were less frequent in the centrifugal-flow pump compared to the axial-flow group; the events/patient-years for thrombosis, stroke, and bleeding were 0.010 versus 0.108, 0.050 versus 0.136, and 0.430 versus 0.765, respectively. Infection, cardiac arrhythmias, and right ventricular failure were similar between the groups.[11]

The rates of rehospitalization were lower with the HM3 than with the HMII in the pivotal trial (225.7 versus 246.4 events per 100 patient-years, respectively; P < .05). In HM3 recipients, the most frequent causes of rehospitalization included infection, heart failure (HF)-related events, and bleeding.[12]

Many other developments were derived from the MOMENTUM 3 trial data and the post-approval study. Specifically, the incidence of de-novo moderate or severe aortic regurgitation appeared to be lower in HM3 than in HMII recipients. At 2 years of LVAD support, freedom from significant aortic regurgitation was greater in the HM3 (92%) than HMII (82%) groups (HR 0.45, 95% CI: 0.27-0.75, P < .01). Increasing age and female sex were significant predictors of aortic regurgitation.[13]

While a sternotomy remains the most common surgical technique for the implantation of an HM3, a growing body of literature shows that there may be clinical benefits to a thoracotomy approach.

In a single-center study, a thoracotomy was associated with improved cardiac index and decreased need for vasopressors in the early postoperative period.[14] In the thoracotomy group, there was a three-fold lower incidence of in-hospital severe right ventricular failure (8.7% versus 28.6%, P < .001) and need for right ventricular assist device support (5.0% versus 17.1%, P = .003). There was also a 68% reduction in the risk of 6-month mortality after LVAD implantation (HR 0.32; CI: 0.13-0.78; P = .012).[14] Preservation of pericardial restraint and right ventricular geometry may be the most important factors limiting right ventricular failure after LVAD implantation via a thoracotomy.[15]

In another cohort, a propensity score match compared patients who underwent a lateral thoracotomy with those who underwent a median sternotomy. There were no differences in an intensive care unit (ICU) or hospital stay duration, time to extubation, vasopressor requirement, in-hospital mortality, or one-year survival.[16] A lateral thoracotomy was associated with significantly less early right ventricular failure (24.4% versus 53.7%, P = .004); however, this group experienced more follow-up tricuspid regurgitation (17.6% versus 0%, P = .030) and volume overload readmissions (P = .0005) compared to the median sternotomy group.[16]

A meta-analysis of this topic included 25 observational studies and 3,072 patients and reported no mortality difference.[17] A thoracotomy approach compared to a sternotomy was associated with a decreased need for blood product transfusions (mean difference [MD]: -4.7; 95% CI: -7.2 to -2.3 units; P < .001), reoperation for bleeding (RR: 0.34; 95% CI: 0.22-0.54; P < .001), postoperative right ventricular assist device (RVAD) implantation (RR: 0.53; 95% CI: 0.36-0.77; P < .001), days requiring inotropes (MD: -1.1; 95% CI: -2.1 to -0.03 inotrope days; P = .04), ICU days (MD: -3.3; 95% CI: -6.0 to -0.7 ICU days; P = .01), and hospital length of stay (MD: -5.1; 95% CI: -10.1 to -0.1 hospital days; P = .04) in matched/adjusted studies.[17]

Mechanical device malfunction was compared in HMII versus HM3 devices in the Rotterdam study.[18] The authors defined major malfunction as those issues that either directly caused or could potentially induce a state of inadequate circulatory support (low cardiac output) or death. These events required urgent surgery or transplantation. Potential major device malfunction was defined as a technical problem that resulted in a pump stoppage or near total blood flow loss for a short time but did not require urgent surgery. During the median support time of 24.6 months (interquartile range [IQR]: 32.4) and 21.1 months (IQR: 27.2) for the HMII and HM3, respectively, mechanical device malfunction consisting of both major and potential major malfunction occurred significantly less in the HM3 patients with an HR of 0.37 (95% CI: 0.15-0.87, P = .022). A major malfunction occurred significantly less in HM3 patients with an HR of 0.18 (95% CI: 0.05-0.66; P = .009). HM3 patients had a significantly decreased hazard for a pump or outflow graft exchange (HR: 0.13; 95% CI: 0.08-0.81; P = .008). In HM3 recipients, significantly fewer system controller defects occurred (P = .007), but battery-clips defects occurred at a significantly higher incidence (P = .039).[18]

Candidate Selection

Substance Abuse

The analysis of the INTERMACS database (2006-2017) showed similar mortality rates for LVAD-supported patients who engaged in alcohol abuse, illicit drug use, and control groups (25%, 21%, and 29%, respectively).[19] However, after adjusting for other covariates, a history of alcohol abuse

or illicit drug use was associated with increased device malfunction/pump thrombosis, device-related infection, or all-cause hospitalization (all P < .05). Furthermore, after LVAD implantation, patients with a history of substance abuse had a lower quality of life than those who did not, as assessed by the Kansas City Cardiomyopathy Questionnaire.[19]

Amyloidosis

As more LVADs are implanted as a form of destination therapy, there is an ongoing exploration of borderline candidates. Patients with restrictive cardiomyopathy, especially cardiac amyloidosis, represent one of the pools of potential LVAD candidates. Two significant factors restrict their eligibility. First, amyloid deposition in the heart results in a small left ventricular (LV) cavity, which can result in device suction events. Second, indiscriminate amyloid deposition leads to similar exposure of the right ventricle, increasing the odds of right ventricular failure post-implantation. Finally, the availability of new amyloidosis stabilizers, such as tafamidis, creates an unknown as to whether the treatment prevents the closure of the LV cavity at a rate that allows for normal functioning of the LVAD.

With all this in mind, a review summarized the up-to-date experiences with LVADs in patients with cardiac amyloidosis.[20] The survival rates reported in the case series are vastly different, ranging from < 50% at 1 month to 100% at 1 year. We need more experience and granularity of the data stratified by the type of cardiac amyloidosis, type of VAD, and the presence of concomitant disease-specific therapy.[20] To avoid the implantation into a small LV, some clinicians advocate for a modified left atrium to aorta implantation technique.[21]

Muscular dystrophy

Young adults with muscular dystrophy are another category of recipients where LVAD support is being actively explored. The first results from the Advanced Cardiac Therapies Improving Outcomes Network registry analysis indicated good survival outcomes.[22]

Management of Patients on LVAD Support

Although LVAD support provides years of life, the compromises on the quality of life are significant. For the first time, there was an attempt to quantify the time patients with LVADs spent in contact with healthcare workers. In a single-center study, the median number of days patients were alive with an LVAD was 390 (IQR: 158-840 days). Patients had a median number of 88 days (IQR: 45-161) with ≥ 1 healthcare encounter, accounting for 23.2% of their time.[23]

Anticoagulation

The idea of using direct thrombin inhibitors instead of warfarin as anticoagulation for patients on LVAD support is attractive. However, initial adverse reports discouraged many clinicians from experimenting with direct oral anticoagulation (DOAC). The randomized controlled trial comparing dabigatran to phenprocoumon in patients with a Heartware ventricular assist device (HVAD, Medtronic) device was prematurely stopped after 50% of patients on dabigatran experienced

thromboembolic events; only 13% of patients on phenprocoumon experienced this complication (P = .28).[24] Despite these results, case reports and case series with successful substitution of DOACs for warfarin continued to be published.[25-27] Because the hemocompatibility of the HM3 is better than other pumps, there is hope that DOACs are a viable alternative to warfarin.

In 2022, Whitehouse et al.[28] prospectively administered either apixaban or warfarin. This study was not randomized, as patients chose their treatment after being educated on the drugs. The median duration of treatment with apixaban was 148 days. Thrombotic complications and death were not different between the groups at six months. The two deaths in the apixaban group were from right heart failure. The apixaban group had clinically lower rates of bleeding complications (5% versus 30%). The adverse events of bleeding, stroke, and death were similar in HM3 patients receiving warfarin or apixaban.[28]

Medical Management

Once again, the role of the phosphodiesterase-5 inhibitor, sildenafil, was tested using the INTERMACS data in patients who received either an HM3 or HVAD.[29] In the propensity-matched analysis, the primary endpoint (a composite of all-cause mortality, ischemic stroke, and pump thrombosis) was lower in the treatment (sildenafil) group (adjusted HR: 0.77; 95% CI: 0.69-0.86; P < .0001). The benefits were robust for both HM3 and HVAD recipients. A separately analyzed all-cause mortality was also lower with sildenafil (adjusted HR: 0.75; 95% CI: 0.65-0.86; P < .0001) for both devices. There were also fewer ischemic strokes, while the rate of pump thrombosis was unaffected.[29]

Interestingly, another group of authors querying the same database came to a diametrically opposite conclusion. Grandin et al.[30] found no effect of sildenafil on late right ventricular failure, mortality, or gastrointestinal bleeding.

To resolve this controversy, Starling et al. suggested a randomized controlled trial to determine the efficacy and safety of sildenafil after LVAD implantation. Assuming an annual event rate (death and or ischemic stroke) of approximately 10% (7%-8% mortality and 2%-3% ischemic stroke) in the control group and projecting 80% power with an HR of 0.75, 6,379 events would be anticipated for a sample size of 1,224. With a projected 90% power and HR of 0.80, 844 events would be expected for a sample size of 2,648. Thus, a sample size of approximately 1,200-2,600 patients is required, representing one of the largest clinical trials on patients with LVADs. The trial design is event-driven, such that the study will be finalized when the number of events is reached.[31]

Arrhythmia and Electrophysiology

Patients with chronic amiodarone therapy at the time of LVAD implantation were compared to those not receiving amiodarone therapy. Treatment was associated with higher all-cause mortality during long-term follow-up (32.9% versus 29.0%, P = .008). The difference persisted after propensity matching.[32] Moreover, Larson et al.[33] showed no difference in ventricular tachycardia events after LVAD implantation in patients discharged on amiodarone versus those not receiving the medication. The authors speculate that ablation may be a better strategy for patients whose quality of life is compromised by frequent defibrillator shocks due to ventricular tachycardia.

Cardiac Resynchronization Therapy

According to previous reports, cardiac resynchronization therapy (CRT) does not improve hemodynamics or outcomes in patients with LVADs, including mortality, hospitalization, LV dimensions, and atrial or ventricular arrhythmias.[34,35] By default, all patients who received CRT but proceeded to LVAD implantation are non-responders to CRT. On the other hand, higher energy consumption by biventricular pacemakers mandates more frequent generator changes—a low-risk but invasive procedure with potential complications.

Surprisingly, the prospective study by Chung et al.[36] demonstrated the clinical benefits of turning the LV lead pacing off after LVAD implantation. Compared with biventricular pacing, RV-only pacing improved LV geometry, patient functional capacity, and quality of life. Patients with RV-only pacing also had a reduced number of ventricular arrhythmias. We covered this study in more detail in our 2021 review.[8]

Another study highlighted the benefits of discontinuing LV pacing after LVAD implantation. In their retrospective analysis, Chou et al.[37] found that in patients with CRT devices and ongoing LV pacing, ventricular arrhythmias were more frequent in the LV-paced group; this was true in the first month after LVAD implantation (21% versus 4%, P = .0001) and the long-term follow-up (43% versus 27%, P = .01). Post-operative ventricular tachycardia storm was also experienced more frequently in the group with active LV pacing (17% versus 9% P = .04).[37]

Valvular Issues: Mitral Regurgitation

The severity of mitral regurgitation subsides after LVAD implantation, but it rarely completely resolves. In an analysis of the INTERMACS database, Jain et al.[38] found that if mitral regurgitation remains in the moderate and severe range after LVAD implantation, patients have worse LV remodeling, higher right heart pressures, and a higher rate of right ventricular dysfunction (HR: 1.88; P = .014) and renal failure (HR: 1.36; P = .040). Younger age, female gender, non-ischemic heart failure, increased left and right intracardiac pressures, and right ventricular dysfunction before the implantation were predictors of residual mitral regurgitation. However, residual mitral regurgitation did not significantly compromise survival.[38]

Complications of LVADs

Right Ventricular Failure

According to the STS-INTERMACS findings, early right ventricular failure (within 1 month of device implantation) occurred in 24% of LVAD recipients.[39] By the end of the first year, it only persisted in 5.3% of patients. If right ventricular failure developed later (>1, but <3 months of support), the prevalence was 4.8%; in 17% of cases, it persisted for 1 year. Right ventricular failure occurred more commonly in centrifugal than in axial devices (OR: 1.17-2.20; P = .004). Other risk factors included higher preimplant blood urea nitrogen (OR: 1.03-1.09 per 5 mg/dL increase; P < .0001), previous tricuspid valve repair/replacement (OR: 2.01-10.09; P < .001), and severely depressed right ventricular systolic function. Patients with persistent right heart failure (RHF) at 3 months had the lowest 2-year survival (57%), while patients with de novo RHF or RHF which resolved by 3 months had more favorable survival outcomes (75% and 78% at 2 years, respectively; P < .001).[39]

Outflow Graft Obstruction

We summarized the LVAD outflow graft obstruction information in a previous review.[5] Outflow graft obstruction typically results from the gradual build-up of thrombus and other biological substances in the bend relief around the outflow graft. We summarized the entity as follows:

- Surgical technique: gore-tex around the outflow tract
- Timing: months to years after the implantation
- Symptoms: HF, low flows (with or without low flow alarm), and decreased pulsatility index
- No hemolysis
- Diagnosis: computer tomography angiography
- Treatment: stenting
- Prevention: discontinuation of wrapping the outflow tract with the Gore-tex®[5]

This year, the condition was specifically studied in HM3 recipients. The patients were considered to have an obstruction if >25% of the cross-sectional area in imaging (percutaneous angiography, computed tomography, or intravascular ultrasound) was compressed. The prevalence of external outflow graft obstruction was 3.0%, and the incidence at 1, 2, 3, 4, and 5 years of support was 0.6%, 2.8%, 4.0%, 5.2%, and 9.1%, respectively. Of 62 patients with this condition, 9 were observed, 27 underwent surgical revision, 15 underwent percutaneous stent implantation, 8 received a heart transplant, and 2 died before intervention. One patient underwent surgical revision and later stent implantation. The mortality with a therapeutic intervention was 17.0%.[40]

Different VADs and Devices

Comparison between different pumps remained of interest in 2022. Such comparisons were performed on large databases, where the clinical details are limited. When mechanical circulatory support was used for nonemergent, high-risk percutaneous coronary interventions (PCIs), the Impella (Abiomed) device was associated with improved survival (OR: 1.55; 95% CI: 1.02 to 2.36) and fewer acute myocardial infarctions (OR: 0.29; 95% CI: 0.18 to 0.46) and cardiogenic shock cases (OR: 0.54; 95% CI: 0.39 to 0.74) compared to the intra-aortic balloon pump (IABP); complication rates were similar.[41]

These results are inconsistent with the previously published results from the PROTECT II (A Prospective, Multi-Center, Randomized Controlled Trial of the Impella Recover LP 2.5 System Versus Intra Aortic Balloon Pump in Patients Undergoing Non Emergent High Risk percutaneous coronary intervention) trial.[42] PROTECT II was a randomized, multicenter clinical study that compared outcomes between the Impella 2.5 and the IABP during high-risk PCI. The Impella 2.5 provided superior hemodynamic support compared to the IABP; the baseline maximal decrease in cardiac power output was -0.04 ± 0.24 W and -0.14 ± 0.27 W for the Impella and IABP, respectively (P = .001). The primary endpoint (30-day major adverse events) was not statistically different between groups: 35.1% for the Impella 2.5 versus 40.1% for the IABP. There were no significant differences in the occurrence of in-hospital death, stroke, myocardial infarction, or the composite of the above between the Impella 2.5 and IABP.[42]

The results differed in another propensity-matched analysis of a large administrative database that compared the same devices but in the setting of PCI for acute myocardial infarction complicated by cardiogenic shock.[43] The Impella compared to the IABP was associated with significantly higher in-hospital (36.2% versus 25.8%, respectively; OR: 1.63; 95% CI: 1.32-2.02), 30-day mortality (40.1% versus 28.3%, respectively; OR: 1.71; 95% CI: 1.37-2.13), and 1-year mortality (58.9% versus 45.0%, respectively; HR: 1.44; 95% CI: 1.21-1.71). At 30 days, intravascular LVAD use was associated with significantly higher bleeding when compared to IABP (19.1% versus 14.5%; OR: 1.35; 95% CI: 1.04-1.76), renal replacement therapy (12.2% versus 7.0%; OR: 1.88; 95% CI: 1.30-2.73), and mean cost (+$51,680; 95% CI: $31,488-$75,178).[43] Utilizing the data from a different database, Kim et al.[44] received principally similar findings in favor of the IABP. Similar results in the same clinical setting were also reported by Dhruva et al.[45]

Interestingly, randomized controlled trials did not provide similar results. The ISAR-SHOCK (Left Ventricular Assist Device [Impella LP 2.5] vs Intraaortic Balloon Counterpulsation [IABP] in Patients With Cardiogenic Shock and Acute Coronary Syndromes) trial was a multicenter, randomized control trial that compared IABP with the percutaneous micro axial flow pump device in 25 patients with cardiogenic shock secondary to acute myocardial infarction.[46] The change in cardiac index at 30 minutes was greater in patients with the Impella (0.49 ± 0.46 L/min/m^2) compared to the IABP (0.11 ± 0.31 L/min/m^2). There were no differences in mortality between the two groups.[46]

In the IMPRESS (Impella Versus IABP Reduces Mortality in STEMI Patients Treated With Primary PCI in Severe Cardiogenic Shock) trial, the IABP was compared with the Impella CP in cardiogenic shock after acute myocardial infarction.[47] There was no mortality difference at 30 days.[47] Additional research has shown no clinical benefit with the Impella 5.0 compared to the IABP.[48]

The different sample sizes may explain the different results that were obtained. There were 25 patients in the ISAR-SHOCK trial[46] and 48 patients in the IMPRESS trial[47] compared to 1,634, 11,500, and 3,360 patients enrolled in the studies by Miller et al., Kim et al., and Dhruva et al., respectively.[43-45]

A growing body of evidence supports that the Impella 5.5 is a reliable pump, providing robust support. Several cases were reported where it was used as a bridge to transplant with a mean support duration of 70 days (maximum 83 days).[49] Another group reported an average time on support of 27 days with a range of 15–80 days and good recovery of end-organ function.[50]

Intra-aortic Devices

One concept in the pathophysiology of HF states that decreased cardiac output and systemic blood pressure result in decreased renal perfusion and urine production and diuretic resistance. Several experimental devices are currently undergoing investigations. Because all of them are designed to increase renal perfusion pressure by increasing the flow in renal arteries, Rosenblum et al.[51] called them "pushers" in their review of innovations in the field of acute HF. The novel intravascular rotary flow pumps are positioned in the descending aorta. They include the Reitan (Cardiobridge GmbH) catheter pump (a collapsible device with a rotational propeller), the Aortix axial pump (Procyrion), and the Second Heart Assist pump (Leonhardt's Launchpads Utah, Inc.) with a propeller (an aortic stent-based pump).

These devices are deployed in the suprarenal descending thoracic aorta and displace blood, create a negative pressure head above the pump, and reduce LV afterload, thereby potentially increasing cardiac output. In an animal study, the Aortix device significantly increased cardiac output and reduced arterial elastance and systemic vascular resistance at low speeds.[52] Compared with baseline values, abdominal activation also increased transpulmonary pressure gradients at medium and high speeds, which was driven by trends toward higher mean pulmonary artery pressure and lower pulmonary capillary wedge pressure.[52]

In a small prospective clinical study, using the Aortix pump resulted in a 10-fold increase in urine output (range 2.5-25.0x) after the mean support time of 70 minutes (range 47-95).[53] The estimated glomerular filtration rate improved at discharge compared with baseline (mean increase 6.95 ± 8.09 mL/min). The insertion of the device took 4-9 minutes.[53] In the first-in-man experience with the Reitan pump, there was an increase in diastolic and mean femoral arterial pressures, and urine output progressively increased over time.[54] In a prospective observational study with the Reitan pump where patients were supported for a mean of 18.3 (±6.3) hours, their cardiac index increased from 1.84 L/min/m^2 (± 0.27) to 2.41 L/min/m^2 (± 0.45, P = .04). Urine output increased from 71 mL/h (± 65) to 227 mL/h (± 179) (P = .006) with a concomitant reduction in serum creatinine (188 µmol/L (± 87) to 161 µmol/L (± 78) (P = .0007). There were no clinically significant hemolysis, vascular injury, or thrombo-embolic complications.[55]

What is New in the V-A ECMO World?

The evidence in the field of cardiogenic shock and short-term MCS usually comes from retrospective studies, meta-analyses, or querying of large databases. This year, a randomized controlled trial on veno-arterial extracorporeal membrane oxygenation (V-A ECMO) in cardiogenic shock was published.[56] In the Extracorporeal Membrane Oxygenation in the Therapy of Cardiogenic Shock (ECMO-CS) trial, immediate implementation of V-A ECMO was compared with an initially conservative therapy (allowing downstream use of V-A ECMO) in patients with rapidly deteriorating or severe cardiogenic shock.[56] The primary endpoint was a composite of death from any cause, resuscitated circulatory arrest, or implementation of another mechanical circulatory support device at 30 days. A total of 63.8% of patients who were provided with immediate V-A ECMO support reached the primary endpoint as compared to 71.2% in the no early V-A ECMO group (HR 0.72; 95% CI 0.46 to 1.12; P = .21). Only 39% of patients in the no early ECMO group required downstream ECMO support. Investigators concluded that the initial conservative strategy was reasonable.[56]

The principles and benefits of LV unloading during V-A ECMO support remain a hot topic. Last year, the propensity-matched analysis of the data from multiple institutions revealed that venting with Impella was associated with a lower 30-day mortality despite the higher complication rate.[57] This year, similar conclusions were achieved based on the query of the Extracorporeal Life Support Organization (ELSO) registry for adults receiving peripheral V-A ECMO support from 2010 to 2019.[58] Patient groups were stratified by the use of an IABP or Impella device. The rate of venting was 26.7%. The mortality in patients with venting was lower than those with no venting (56.6% versus 59.3%, P = .006); no difference was noted between the IABP or Impella usage. Not surprisingly, there were fewer complications with the IABP.[58] In a nationwide Japanese study of cardiogenic shock due to acute myocardial infarction, patients managed with V-A ECMO plus IABP demonstrated a significantly lower in-hospital, 7-day, and 30-day mortality rate when

compared to those managed with V-A ECMO alone (adjusted OR [95% CI] of 0.47 [95% CI, 0.38-0.59], 0.41 [95% CI, 0.33-0.51], and 0.30 [95% CI, 0.25-0.37], respectively).[59]

Interesting studies were done in the area of blood pressure and V-A ECMO support. Using the ELSO Registry data, Rali et al.[60] found that every 10-mm Hg increase in baseline systolic blood pressure (HR: 0.92; 95% CI: 0.89-0.95; P < .001), and baseline pulse pressure (HR: 0.88; 95% CI: 0.84-0.91; P < .001) at 24 hours was associated with a statistically significant reduction in mortality. Specifically, at baseline, median systolic (88 mm Hg versus 85 mm Hg) and diastolic blood pressure (55 mm Hg versus 52 mm Hg) were significantly higher among survivors compared with non-survivors (P < .001). Whereas the pulse pressure was ~31 mmHg in both survivors and nonsurvivors. After 24 hours of V-A ECMO support, the median systolic (98 mm Hg versus 92 mm Hg), diastolic (65 mm Hg versus 63 mm Hg), and pulse pressures (32 mm Hg versus 27 mm Hg; P < .001) were all higher among the survivors versus non-survivors.[60] Another study showed that low pulse pressure (< 20 mm Hg) in patients supported with V-A ECMO was independently associated with acute brain injury (adjusted OR 2.57; 95% CI = 1.05-6.24). Every 10-mm Hg decrease in pulse pressure was associated with a 31% increased odds of acute brain injury (95% CI = 1.01-1.68).[61]

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