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REVIEW

Generation of Pulsatile Flow using Clinical Continuous Flow Pumps

Joav Birjiniuk,[a,*] Deane E. Smith,[b] Eugene A. Grossi,[b] Aubrey C. Galloway,[b] and Nader Moazami,[b]

[a]*Department of Surgery, New York University Langone Medical Center, New York, New York;*
[b]*Department of Cardiothoracic Surgery, New York University Langone Medical Center, New York, New York.*

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BACKGROUND: With the increasing use of mechanical circulatory support for long-term augmentation of cardiopulmonary function, the need for safer devices is apparent. Pump thrombosis and failure, inadequate ventricular unloading, progressive right-sided dysfunction, and end-organ hypoperfusion are seen with long-term mechanical circulatory support devices. Generation of pulsatile flow has been proposed to mitigate some of these risks by providing physiologic flow and pressure profiles to the vascular system and end-organs. Modification of continuous flow devices to provide pulsatility may prove a cheap and effective way to achieve physiologic flow; however, effective use of such a technique has yet to be demonstrated. This work aims to describe these efforts, as well as mechanical arguments regarding the challenges to be overcome in achieving this goal.
METHODS: Prior literature and textbooks were used to develop the theoretical basis for the paper.
RESULTS: Attempts at generating pulsatile flow with continuous flow devices have been marred by difficulty in mitigating viscous effects on oscillating mechanical systems. Currently available devices and research setups have been unable to generate truly physiologic pulsatile flow systems. New devices are needed that utilize various forms of positive displacement in order to generate true pulsatile flow that mimic native waveforms generated by the heart.
CONCLUSIONS: The mechanical challenges in generating pulsatile flow with continuous flow devices have precluded their widespread adoption in clinical practice. New pulsatile pumps are needed to achieve adequate physiologic pulsatility with improved side-effect profile.
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Motivation

Need for mechanical circulatory support

As the number of patients requiring heart transplant increases, and the donor pool remains relatively stable, there remains an inadequate supply of donor organs to meet demand. This deficit is predicted to persist for years to come.[1] Mechanical circulatory support (MCS) devices have thus become a critical component in the management of end-stage cardiac failure. These devices typically consist of an inlet fitting, chamber with pumping mechanism, outflow conduit, and pump driveline. With increasing durability and reliability, and decreasing size, these devices have become

*Corresponding author: Joav Birjiniuk, NYU School of Medicine, Department of Surgery, 550 First Avenue, New York, NY 10016.
E-mail address: joav.birjiniuk@nyulangone.org.

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attractive options for improving both survival and quality of life as patients await transplant, or as an alternative to transplantation altogether.[2]

Failure modes of long-term therapy

Despite their increasing use, MCS devices are plagued by several problems, which are compounded by longer duration of therapy.[3] These may be broadly categorized as being mechanical or nonmechanical in nature. The former includes failure of the driveline or other external components, and overt pump failure secondary to mechanical or electrical causes.[4] Unlike previous devices used clinically, the newer generation continuous flow centrifugal pump, the HeartMate 3, has been associated with fewer reports of mechanical failure.[5] On the other hand, nonmechanical complications, including strokes, gastrointestinal bleeding, right heart failure, prolonged renal insufficiency, and aortic valve insufficiency, persist.[3,6] Devices that can generate pulsatile flow in relation to the native heart flow may be the next important step in mitigating some of the aforementioned nonmechanical complications. Pulsatile flow may reduce the rates of embolic and hemorrhagic strokes, arterio-venous malformation, aortic valve insufficiency, and right ventricular dysfunction.

If there is hemodynamic and clinical benefit to pulsatile flow, an ideal device would be able to incorporate current continuous-flow devices with new modes of generating pulsatile flow. Initial attempts at long-term support devices, such as the Liotta-Cooley and Jarvik artificial hearts, were pulsatile.[7] At the time, this technology allowed for intracorporeal implantation with extracorporeal control of the circuit via pneumatic pumping of bladders, obviating the need for reservoirs and extracorporeal circulation. Unfortunately, these devices required cardiectomy and were not durable enough for long-term use. With improvements in biomedical manufacturing, small continuous flow pumps were able to be produced that could remain in intracorporeal position with continued native function. As the elements for developing pulsatile flow remained bulky, and their benefit unclear, these devices were largely abandoned.

In this review, we attempt to reconcile the available data as to the question of pulsatile vs continuous flow and investigate the difficulty in achieving pulsatile flow with commercially available devices.[8] We hypothesize that currently available continuous-flow devices are unable to overcome the challenges necessary to generate such pulsatile flow, and that novel devices are necessary to achieve this flow mode.

Clinical differences between continuous and pulsatile flow modes

Maintenance of end-organ perfusion, as well as off-loading of the cardiac chambers and maintaining physiologic flow architecture, are chief among functions of adequate mechanical support. Given the pulsatile nature of physiologic flow, much effort has been placed in the understanding of the role of such conditions in tissue perfusion, vascular remodeling, ventricular loading, and systemic organ function.[9,10] Despite numerous studies investigating these effects in vitro, in silico, and in vivo, there have been limited attempts at large-scale studies in clinical settings. Furthermore, despite understanding of differences between pulsatile and continuous flow, clinical studies have not been uniform in their use of truly pulsatile, physiologic flow profiles.[11,12] In light of these shortcomings, significant retrospective studies have elucidated some clinical differences between pulsatility modes.

Renal and end-organ perfusion and dysfunction

Maintenance of adequate end-organ perfusion pressure

As described, the ultimate goal of any mechanical support device is to assist the failing heart in delivering flow to end organs to maintain tissue perfusion. Since the advent of John Gibbon's heart-lung machine, there has been significant interest in the interaction between human and machine physiology, as well as the effects of long-term extracorporeal circulation. Clinical evidence of secondary hepatic and renal dysfunction during extended pump runs prompted a burgeoning field of study of continuous flow physiology. Early work in the 1960s demonstrated some level of deficient renal blood flow in continuous flow circulation, even at high flow rates.[13] Work in depulsation of native aortic flow demonstrated inadequate renal blood flow, urine output, and sodium clearance when both kidneys were subjected to abnormal flows. These disturbances, however, were found to be restored with return of pulsatile flow to at least one of the kidneys.[14,15] Other work demonstrated increased rates of acute tubular necrosis on histology of explanted kidneys from dogs left on extended continuous-flow pump runs.[16] Again, as many different types of systems were used to generate pulsatile flow, it is difficult to compare adequately the true pulsatility of each system. However, these studies do point to an inherent need for physiologic flow waveforms in long-term circulatory support.

Ensuing clinical studies of pulsatile flow for cardiopulmonary bypass recapitulated the improved urine output with pulsatile flow.[17,18] Furthermore, pulsatile flow was found to attenuate part of the vasopressin response to surgical and bypass stress. Other works demonstrated lower systemic vascular resistance, acidosis, and volume or transfusion requirements.[9,19] Due to the difficulty in enrolling patients in large trials to study pulsatile and continuous flow head-to-head, there has been a dearth of work to directly assess these differences. With improved surgical techniques and operative times, as well as newer pumping technologies, there has been waning interest in applying pulsatile flows to intraoperative cardiopulmonary bypass.

As circulatory support modalities are used increasingly for patients inside and out of the hospital, there is renewed interest in the role of pulsatility in relation to renal dysfunction. Current evidence suggests overall improvement in glomerular filtration rate and serum creatinine values after implantation of left ventricular assist device (LVAD) with continuous flow technology.[20] However, head-to-head

studies have also demonstrated persistent and increased elevations in renin and aldosterone following implantation of continuous flow devices as compared to pulsatile devices.[21] Some of these effects may be explained by residual right ventricular dysfunction following LVAD implantation, but there remains concern that this phenomenon may be explained by the lack of pulsatile flow. The effects of pulsatility on end-organ perfusion and function may be somewhat explained by the role of the vascular system in conducting energy.

Energy-equivalent pressure and surplus hemodynamic energy

The elasticity of human arteries imbues them with the capacity to store energy, allowing for energy delivery, via pressure work, throughout the cardiac cycle.[22] This is particularly important in conditioning pressure waveforms through the vascular tree and ensuring sustained distending pressure. Thus, the pulsatile nature of native blood flow can be seen as a critical component in preventing microvascular collapse. Furthermore, this persistent pressure and energy gradient between major vessels and end-organ vascular systems promote perfusion of peripheral tissues. Energy equivalent pressure (EEP) is a metric used to describe the pressure work done through a surface over a given cardiac cycle relative to the blood volume ejected through said surface.[10] This quantity may be used as an estimate of the convective energy, and thus driving force, promoting blood flow to end tissues, for a given cardiac cycle (Equation 1, Supplement). Surplus hemodynamic energy (SHE) is the difference between this quantity and the mean arterial pressure as seen in Equation 2 (Supplement). It is then evident that as a system tends to completely continuous flow, the SHE trends to 0 (Equation 3, Supplement). As the complex impedance of various vascular beds may be modeled as a Windkessel-type circuit, elimination of the periodic component of the pressure waveform renders a completely resistive circuit.[23] The resulting pressure and energy transport are therefore distributed solely by relative resistances of vascular beds, regardless of their distensibility. This may promote preferential perfusion to less clinically relevant end-organs.

Clinical investigation has borne out these effects of pulsatile flow beyond their purely hemodynamic and physiologic basis. As described in the original studies delineating EEP, greater flow rates and pressures are necessary in continuous flow modes to meet the kinetic and potential energies delivered with pulsatile flow.[10] Several studies have since demonstrated decreased microvascular density and flow, as well as adverse microvascular overcirculation in patients undergoing cardiopulmonary bypass with continuous flow as compared to pulsatile flow.[24,25] These differences were found to persist in the immediate postoperative period, though long-term follow-up was not performed. Animal studies in models of cardiac failure and cardiogenic shock have demonstrated worse base deficit and lactic acidosis with nonpulsatile cardiac support, with improved cerebral oxygen saturation and recovery of regional liver, stomach, and renal cortical perfusion when pulsatile regimes are utilized.[26,27] Unfortunately, there are few studies in humans bearing out these effects, and in fact, some touting no difference in end-organ function between continuous and pulsatile flow modes.[28,29] Further study in human microcirculation differences between continuous and pulsatile flow modes is warranted.

Left ventricular augmentation

Principle among functions of any support device is the augmentation of ventricular output with concomitant off-loading of the supported chamber. In this regard, there are clear differences between continuous and pulsatile flow modes. Continuous flow can be considered a "DC" (direct current) signal that elevates the overall flow rate throughout the cardiac cycle (Figure 1). Alternatively, pulsatile flow by nature interacts with the periodicity of native cardiac function. Differing modes of pulsatility can have varying effects on both stroke volume and stroke work as a result of their timing relative to the native cardiac cycle. In copulsation modes, pulsatile devices are triggered by native function and thus tuned to the natural frequency of the patient's heart. This amplifies stroke volume and thus cardiac output, as the native left ventricle (LV) contractility is augmented by the pulsatile flow device (Figure 2).[30] Alternatively, counter-pulsation modes provide sustained cardiac output by delivering flow into the aorta during cardiac diastole, while effectively off-loading the LV during cardiac systole (Figure 3). Asynchronous modes capitalize on the advantages of both of these variations, with variable phase relative to the native cardiac period, thus at times maximizing LV augmentation while at others increasing LV unloading. Additionally, such modes allow for intermittent opening of the aortic valve, reducing risk of developing aortic root thrombosis and valvular insufficiency. These varying methods of synchronizing human and machine also have a clear effect on stroke work, as can be calculated by the EEP and SHE. Furthermore, the significant left ventricular off-loading performed by counter-pulsation with pulsatile devices may help promote augmentation of native cardiac function.

With increasing flow rate using continuous flow LVAD, the pulsatile component of the resultant flow decreases relative to the continuous component, as calculated via the pulsatility index (PI).[31] It is apparent that as continuous flow LVAD support increases, raising the mean flow, the pulsatility index by definition will decrease without a

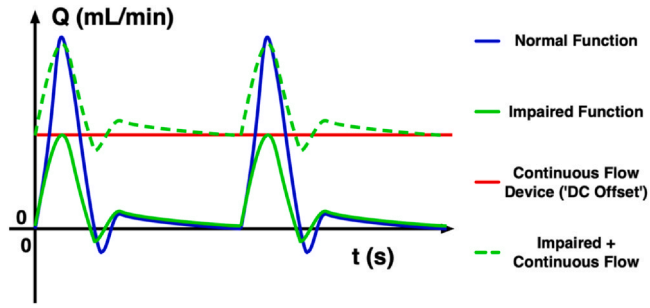

Figure 1 "Direct current" offset produced by continuous flow mechanical support in addition to native cardiac function.

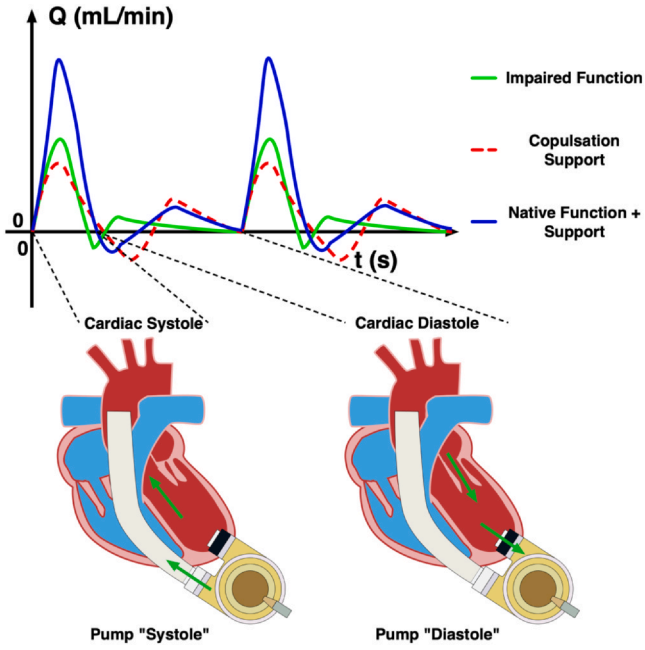

Figure 2 Pulsatile flow support system operating in copulsation mode. Pump "systole" is synchronized to native cardiac systole to maximize flow during systole.

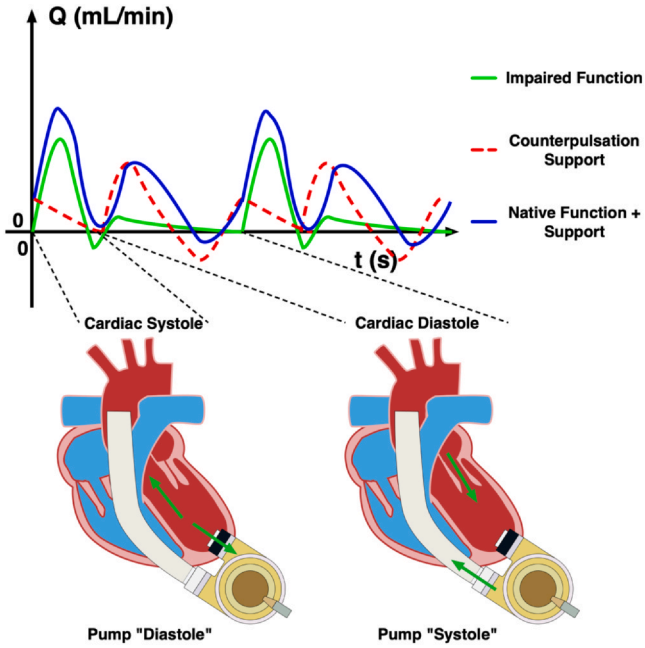

Figure 3 Pulsatile flow support system operating in counterpulsation mode. Pump "systole" is synchronized to native cardiac diastole to maximize left ventricular unloading.

concomitant increase in native pulsatility (Equation 4, Supplement). While increased flow as a result of LVAD support may increase the overall hydrostatic pressure, the hydrodynamic pressure resulting from pulsatile flow, critical in maintaining vascular integrity and SHE, and in washing out areas of stagnation, is diminished. Thus, despite further augmentation of cardiac output as device support increases, effects on pulsatility may adversely affect the vasculature and elimination of stagnant, prothrombotic zones.

Right ventricular loading

While continuous flow LVADs are able to augment LV function, and perhaps aid in offloading the LV as well, there is concern that this does not adequately offload the right ventricle. In fact, there remains concern that while aiding left ventricular mechanics, continuous flow devices may worsen RV mechanics. This is hypothesized to result from both the increased preload delivered to the failing RV as a result of augmented cardiac output, but more importantly, the alteration of interventricular septum mechanics. As the VAD inlet at the apex sucks fluid in, a relative Venturi effect is created, which in conjunction with the decreased LV volume is thought to lead to bowing of the septum inwards toward the LV. As such, the contribution of the septum to RV output may be diminished with this phenomenon. Prior work has demonstrated that upward of 60% of flow through the PA may be attributed to contributions of the LV to RV function, via the interventricular septum.[32,33] Furthermore, this action may further chronically lead to RV volume overload. Additionally, with chronic use of continuous flow devices, the microvascular dysfunction described previously may lead to visceral and peripheral arteriovenous shunting and eventual RV volume overload and pulmonary circulatory alterations. Unfortunately, there is little study of these effects on the pulmonary vasculature, interventricular septum, and RV function after VAD implantation. Further characterization of their effects may aid in future device design.

Bleeding

Early long-term outcome studies of continuous LVAD systems demonstrated a marked propensity for development of gastrointestinal (GI) bleeding in comparison to earlier pulsatile models.[34-36] This has since been recapitulated in a number of studies, with further demonstration of formation of gastrointestinal arterio-venous malformation in these patients as the etiology of their bleeding.[37-40] To compound these sources of potential GI bleeding, many patients with continuous-flow support appear to develop an acquired von Willebrand disease, as seen with Heyde syndrome.[41-43] High shear generated along continuous flow devices is believed to elongate high-molecular-weight von Willebrand Factor (vWF), exposing cleavage sites for the endogenous enzyme ADAMTS13. This appears to be exacerbated by RV dysfunction and subsequent venous back-pressure.[44,45] The exact mechanism causing such an effect remains unclear. Some of these effects seem to have been mitigated by newer generation centrifugal devices, such as the HeartMate 3, with some studies demonstrating decreased depletion of high molecular weight multimers of vWF, as well as decreased rates of GI bleeding.[38,46-48] Other studies have demonstrated reduced GI bleeding rates in cohorts with relatively higher PI.[49] These data suggest that reduced continuous flow as compared to native flow may mitigate some of the high-shear effects on vWF degradation seen with continuous flow

devices operated at high rotational speeds. In fact, the relative absence of gastrointestinal bleeding in completely pulsatile devices indicates that the mechanism of generating flow may be the cause of this serious complication.

Thrombosis

As with most prostheses involving the cardiovascular system, there remains a significant risk of in-device or in-conduit thrombosis with MCS devices. Furthermore, stagnant flow in the aortic root can predispose to thrombosis. Such events can lead to clinical or subclinical cerebrovascular accident, systemic embolic disease, cardio-embolic event, or overt device failure. These events account for significant morbidity and mortality among patients with implanted MCS devices, especially those on destination therapy.[50,51] While some level of thrombosis is believed to be unavoidable given the interaction with blood and artificial surfaces, there remains a significant contribution of the hemodynamic milieu to this phenomenon. High shear zones can lead to vWF elongation and conglomeration, forming vWF nets and subsequent platelet-rich thrombus.[52] Such high-shear zones are generated about the vanes of both axial and centrifugal flow pumps. Similar regions of high shear are developed along the bearings of centrifugal flow devices and have been shown to be niduses of platelet-rich thrombus in extracorporeal circuits.[53] As MCS devices become further miniaturized in subsequent iterations, their smaller dimensions, as well as the increased rotational speeds required to maintain adequate pump flow, may lead to even higher shear rates, and thus, in situ thrombosis.

There remains little literature studying the difference in thrombosis rates between pulsatile and continuous flow devices as the former have largely fallen out of common use. However, as the blood in these systems is exposed to high shear rates for relatively short period of time, it is reasonable that these devices are less likely to generate long vWF fibers and thus platelet-rich thrombus. However, newer continuous-flow devices, such as the Heartmate 3, claim to reduce in-device thrombosis by rheological means. By magnetically levitating the pump head, a thin layer of blood is able to accumulate between the pump head and pump housing. This serves to coat these surfaces with a semiendothelial layer, creating a biological antithrombosis coating. Furthermore, this device makes use of a frequent, miniscule change in rotational speed to promote wash out of stagnant blood. In fact, recent study has demonstrated that these devices suffer from less device-related thrombosis, with patients experiencing fewer thromboembolic events and less pump thrombosis.[48,54] These results point to pulsatile flow, even if miniscule in amplitude, as a mediator of pump washout and reduced thrombosis.

Pump mechanics and design challenges

Biologic basis for pulsatile flow

As mentioned, human arteries have developed to possess varying levels of elasticity across the cardiovascular system, impacting their distensibility in various arterial beds. This important adaptation may have resulted from the need for biological flow to be generated via pulsatile means. While man-made pumps can generate continuous flow much more reliably than pulsatile flow, these require free-standing or freely rotating members. Additionally, continuous motion of such a member is dependent on constant energy input to maintain rotation or reciprocation. Living organisms, alternatively, depend on contractile elements made of biological tissue, such as myofibrils, to generate motion.[55] As such, their pumping mechanisms require periods of relaxation, restoration of energy sources, and multiple conformations to achieve fluid propulsion. By the very nature of these limitations, living tissues are unable to generate truly steady, continuous flow. Whether the compliant conduits and perfusion mechanisms evolved around these pump qualities, or vice-versa, it is understood that the human body attains normal perfusion characteristics under pulsatile flow conditions.

Perhaps in response to these requirements, the peripheral vasculature has developed to accommodate and sense pulsatile flow. There is increasing evidence indicating that mechanotransduction on endothelial cell surfaces affords these cells the ability to sense changes in shear stresses. Additionally, a component of radial force due to the transmitted pressure waveforms may play a role. In fact, much in vitro study has demonstrated sensitivity of these cells to pulsatile flows leading to improved vasodilation due to endothelium-derived relaxing factor and nitric oxide signaling.[56-59] Studies in humans have shown that this relaxability may be reduced in patients supported with continuous flow LVADs, even compared to those with advanced heart failure not receiving mechanical support.[60-61]

Prior art and current state of affairs

Generating pulsatility with speed modulation of continuous flow devices.

Speed modulation modes

With renewed interest in delivering pulsatile flow for long-term support, there has been much work over the past several years aimed at repurposing clinically-available continuous flow devices to generate such flow. As seen prior, there are significant mechanical challenges in creating such systems. Some groups have used PI controllers to modulate driver output for continuous flow devices and generate pulsatile flow profiles based on reference waveforms.[62] Others have employed prescribed pump speed profiles regardless of associated serial loads to enhance pulsatility.[30,63,64] While all of these approaches have been able to enhance pulsatility at higher levels of support as compared to continuous modes, they have not shown significant pulse pressure beyond native function. Additionally, these studies have not been performed over

prolonged periods of time. As such, the ultimate benefit of these strategies is unclear, suggesting that attempting to develop pulsatile flow with continuous flow devices may not provide the proposed benefits of true pulsatile flow.

Overcoming inertial forces

Generating pulsatile flow in viscous fluids with substantial density, such as blood, incurs challenges beyond those encountered in continuous flow modes. Before truly steady flow is developed, the working fluid (i.e., blood) must be accelerated, which requires substantial force to overcome fluid inertia. Additionally, the pumping components have inertia of their own which must be overcome to provide adequate impetus for fluid flow. These can be described as the separate inertial moments of both the fluid and the impeller or pump head, and contribute to the impedance of the fluid system. This discrepancy between component and fluid moments in and of itself can lead to flow separation and dyssynchrony. In such fashion, fluid flow may become detached ("separated") from the device components, leading to regions of significant stagnation and drag.

However, these effects are greatly compounded and become problematic in pulsatile flow systems derived from pumps that are not specifically designed to handle such flow (i.e., continuous flow devices). Rapid changes in pump speed can lead to worsening dyssynchrony between pump input and flow output. Any fluid with substantial mass (such as blood) requires substantial time for full acceleration and deceleration to appropriate velocity. The phase delays incurred as a result of fluid inertia can lead to significant conditioning of the output pressure and flow waveforms, with washout of the final signal. This conditioning here refers to a low-pass filtering effect in which the high-frequency components of a signal, such as a square wave, are filtered out, resulting in smoothing of the output waveform with loss of sharp features such as corners. With adequate enough filtering, this may result in a continuous signal without any pulsatility. Sufficient enough delays, especially with increasing frequency of pulsatility, may lead to superposition of waveforms with further washing out of pulsatile behavior (Figure 4). At high enough frequency, this may render a completely continuous signal or eventual stagnation of the pump head as its own movement is impeded by the stagnant fluid it is attempting to accelerate. Such modulation of speed and subsequent shear can also contribute to significant shear-thinning and changes in viscosity of non-Newtonian fluids, such as blood, which may further exacerbate hemolysis and signal washout.

Furthermore, as significant torque is required to overcome these inertial moments and change directions rapidly, speed modulation of continuous flow devices requires substantial power beyond that used in normal continuous-flow modes. This increased power consumption is sure to limit the lifetime of device battery, and the high torques required likely to hasten deterioration of pump components.

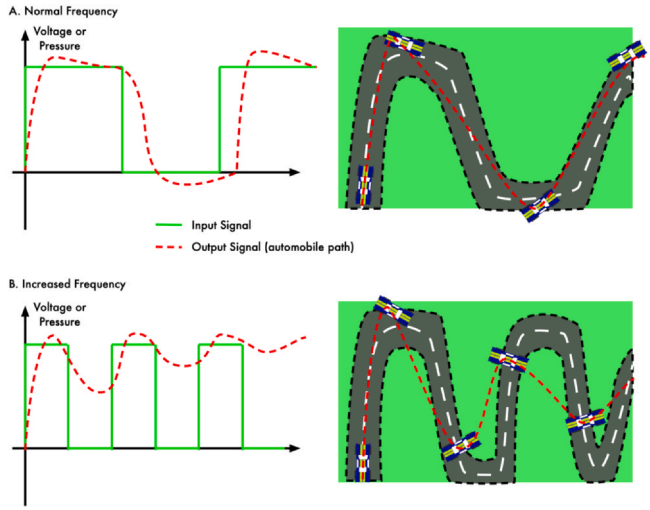

Figure 4 Conditioning of fluid waveforms with square wave applied with normal (A) and higher frequency (B) pulsatility. We can liken this to an automobile attempting to make sharp turns to remain on a winding road. As the frequency of turns increases, the driver has to change direction more often and is unable to reach full speeds in one direction or the other. Thus, over time, the "signal" (i.e., the path of the vehicle) loses its pulsatility.

Unnatural forcing functions

In conjunction with the rapid changes in rotational speed required to achieve pulsatility, unnatural forcing functions with discrete changes in motor speed have a significant impact on pump components. While some of these changes are conditioned by the fluid itself, the abrupt change in speed can lead to shearing of components. Additionally, the cyclical nature of loading, with changing loading conditions and stresses, can lead to dissociation between motor and shaft/impeller, or may simply surpass the working conditions of the motor. These loads may ultimately lead to device wear and failure.

Effects on blood

As mentioned, rotational speeds beyond normal continuous-flow conditions are necessary to achieve adequate pulsatility with these devices. As such, peak flows and resultant shear are far greater in speed-modulation modes. This high shear regime may exceed typical shears experienced with continuous flow devices, which may lead to vWF elongation and high-shear thrombosis as mentioned prior.[65] Furthermore, the rapid deceleration phase necessary to create diastole may lead to adverse pressure gradients resulting in flow reversal, oscillatory shear, and areas of stagnation, predisposing certain features of the pump, such as the hollows under the vanes of the pump head, to low-shear coagulation. These variable effects have yet to be studied in controlled settings and are likely to become more apparent when speed modulation is used for long-term support. Current work has yet to subject these devices to longer runs under pulsatile conditions.

Future technologies

Pulsatile flow devices

Positive displacement

With Gibbon's first heart-lung machine, positive displacement pumps have maintained permanence in the field of cardiopulmonary support. These pumps may come in a variety of configurations, such as the commonly used roller pump, and work by mechanically displacing a certain volume of blood with each stroke of the actuator or rotation of the pump head. As the inlet flow relies on the suction forces generated by pump rotation and equivalent outlet flow, these devices suffer less from changes in inertia compared to centrifugal or axial flow pumps. The TORVAD device (Windmill Cardiovascular Systems) is a valve-less positive displacement pump that makes use of 2 independent pistons to generate separate strokes with unidirectional control of flow.[66] It is able to efficiently deliver a fixed stroke volume with tight modulation of rotational speed in time to generate pulsatility with far lower shear than experienced in available continuous flow devices. The individual strokes can be synchronized at will to underlying cardiac rhythm to achieve desired pumping characteristics. Though yet to be approved for human use, significant study has shown improved ventricular mechanics with improved hemocompatibility.[67-70]

Pulsed membrane

Unlike traditional positive displacement pumps, pulsed membrane devices create small, fixed displacements of fluid with actuation of a levitating membrane. By changing the frequency of these displacements, the flow rate of the device can be modulated. The CorWave device (CorWave, Clichy, France) utilizes such a mechanism with a small profile allowing for intrapericardial implantation much like the HeartMate 3. Given the small displacements and necessary fluid speeds, these devices have lower overall shear profiles, even with pulsatile peak flows, and thus improved hemocompatibility. Additionally, as with the TORVAD device, newer sensing technologies allow for adaptations in flow delivery to meet physiologic demands. Furthermore, this device can achieve true pulse pressure with higher fidelity to native waveforms. Unfortunately, there is little data on hemocompatibility with longer pump runs, as well as no in-human data at present. However, this type of device has some promise in delivering physiologic flow to patients needing long-term support.

Clinical trends

As the newest pulsatile devices are yet to be made available for clinical use, there remains continued use of standard continuous flow LVADs. However, demonstration of the potential superiority of pulsatile flow remains elusive without use of such devices in patients, further preventing their wide adoption. Despite this, the number of patients needing cardiac support far surpasses the hearts available for donation, and long-term mechanical support will continue to be required.

Conclusions

Use of mechanical circulatory support devices continues to increase as further patients require augmentation of native function. Pulsatile devices have the potential to promote improved microcirculatory and ventricular mechanics, as well as reduced pump thrombosis and associated consequences. Attempts to deliver pulsatile flow with continuous flow devices are limited by the mechanics of these pumps and are inadequate in reaching the characteristics desired with true pulsatile flow. Thus, the need for modern pulsatile flow devices is apparent, and the latest offerings show promise in achieving more physiologic mechanics with fewer patient- and device-sided complications.

Disclosure statement

The authors declare that they have no known competing financial interests or personal relationships that could have appeared to influence the work reported in this paper.

Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.jhlto.2023.100032.

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