Biomechanics of Arteriovenous Fistula: An Overview of Hemodynamic and Remodeling Mechanisms

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ABSTRACT

Arteriovenous fistulas (AVFs) are widely accepted as the gold standard for vascular access in chronic hemodialysis patients due to their superior long-term patency and low complication rates. However, up to half of all created AVFs fail to mature adequately, and many develop late complications such as stenosis, thrombosis, or aneurysmal degeneration. This review provides an overview of the AVF development process, focusing on the biomechanical forces that drive vascular remodeling and contribute to maturation and pathological changes. We summarize the role of endothelial mechanotransduction and insights from recent studies that reveal how wall shear stress (WSS) patterns and oscillatory flow relate to the genesis of neointimal hyperplasia and later stenosis. Understanding these mechanobiological processes has led to novel surgical techniques, anastomosis design optimization, and better cannulation strategies. Although the knowledge of hemodynamic-biological interactions remain limited, further research can offer directions for better AVF performance.

KEYWORDS

arteriovenous fistula; hemodynamics; vascular access; remodeling; wall shear stress

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INTRODUCTION

The prevalence of patients with end-stage renal disease (ESRD) requiring regular dialysis has been growing, with approximately 550,000 people in Europe being dependent on this treatment (1). The rising count is associated mainly with demographic changes – population aging, and the increasing incidence of diabetes mellitus and hypertension, which are both major contributors to the development and progression of chronic kidney disease (CKD). In patients who advance to ESRD, renal replacement therapy becomes essential to sustain life (2, 3). Today's medical science offers three main treatment options for renal replacement: hemodialysis, peritoneal dialysis, and kidney transplantation (3, 4). While in the past ESRD used to be considered fatal, developments in therapeutic strategies, such as strict treatment-monitoring protocols and advanced modern hemodialysis machines, have made the whole treatment process safe and serious complications rare. Hemodialysis is generally the most commonly used modality, typically performed in specialized outpatient departments of hospitals or clinics, three times per week in sessions lasting approximately four hours. However, the exact regimen serves individual patient needs. Peritoneal dialysis, in contrast, is conducted daily, usually at the patient's home. For example, automated peritoneal dialysis (APD) commonly takes place overnight.

HEMODIALYSIS (HD)

Hemodialysis, a life-sustaining extracorporeal treatment for patients with ESRD, has become a routine procedure. With modern dialysis equipment, which is accessible worldwide, and established treatment protocols, the incidence of severe complications has markedly decreased (5). In this complex procedure, uremic toxins and excess fluid from the bloodstream are filtered through a semipermeable membrane using counter-current flow of dialysate (5, 6). Small waste products are removed primarily through diffusion, osmosis, and ultrafiltration. During diffusion, waste products move from areas of higher concentration in the blood to areas of lower concentration in the dialysate. Osmosis helps balance fluid levels by allowing water to move across the membrane. Ultrafiltration, generally driven by transmembrane pressure differences, can efficiently extract excess fluid from the vascular system. These combined mechanisms ensure toxin removal and fluid balance maintenance (6).

DIALYSIS ACCESS

Dialysis access is essential to dialysis, providing a reliable and durable site through which blood can be removed and returned during each procedure. The ideal vascular access should be technically easy to construct, ready for immediate use after construction, and low-maintenance. Unfortunately, none of the available options meet all the criteria mentioned above. There are three main types of dialysis access: arteriovenous fistulas (AVF), arteriovenous grafts (AVG), and central venous catheters (CVCs) (7, 8). To compare these types of vascular access, CVCs can be used immediately, AVGs either also immediately or within two weeks, depending on the chosen prosthesis material, and AVFs require the longest period for maturation, which is four to six weeks (8).

Both AVFs and AVGs are technically demanding to create, while inserting a CVC is considered relatively easy and less invasive. These procedures also differ in terms of patient administration, medical procedure planning, and length of hospital stay. The initial success rate of AVFs is low, but after maturation, they offer high long-term patency. In contrast, AVGs and CVCs have high initial success rates but lower long-term patency, with central vein catheters having the worst performance (8, 9). Blood flow rates are high in AVGs and matured AVFs, but low in CVCs. The probability of infection is lowest with AVFs, higher with AVGs, and highest with CVCs. CVCs also have a high probability of central vein thrombosis (10). AVFs are noted for their high primary patency rates, remaining functional for extended periods without requiring intervention. Long-term, AVFs generally provide better outcomes than AVGs and CVCs. In terms of maintenance, arteriovenous fistulas require fewer interventions to maintain their functionality, whereas arteriovenous grafts and CVCs usually need more frequent upkeep. The selection of the appropriate access type depends on patient-specific factors, including vascular anatomy, existing health conditions, and the expected duration of dialysis. Hemodynamic benefits are significant with AVF as they support higher blood flow rates and have less negative inflow arterial pressure, which contributes to more effective dialysis treatment (11).

DEVELOPMENT AND MATURATION OF AVF

An AVF is constructed by creating a direct anastomosis between an artery and an adjacent vein, typically in the forearm or upper arm. The arterial blood flow is redirected into the venous system, resulting in elevated flow and pressure that induce progressive vein remodeling. The vein subsequently undergoes hypertrophy and dilation to accommodate repeated cannulation and hemodialysis's high blood flow demands (12).

After surgery arteriovenous fistulas require a maturation period of four to six weeks. However, once matured, they demonstrate superior long-term outcomes, including prolonged patency and lower infection rates with rare need for interventions (8). The mature AVF ought to keep flow rates of approximately 350–450 mL/min to support adequate dialysis. In clinical practice, forearm AVFs commonly achieve flows between 500 and 2000 mL/min, while upper arm AVFs may reach 3000 mL/min (12, 13). Despite proper surgical technique, failure of maturation occurs in approximately 20–50% of AVFs (12, 14). One potentially serious complication is distal ischemic steal syndrome, in which retrograde flow into the venous system deprives the distal extremity of arterial perfusion. In severe cases, this may necessitate surgical revision or ligation of the fistula (15).

Long-term complications, even in well-functioning AVFs, are frequent. Most common complication include stenosis, thrombosis, and aneurysmal degeneration (14, 16).

These complications often arise at sites of repeated puncture or in regions subject to disturbed flow. Aneurysm formation is typically localized to the venous segment of the arteriovenous fistula and may be preceded by intimal hyperplasia and vessel wall thickening (16, 17). Segmental venous dilation may disrupt laminar flow, promoting thrombogenesis. Narrowing of the outflow tract with stenosis results in increased wall tension and may contribute to aneurysmal progression. AVF aneurysms frequently exhibit tortuous morphology, with thinned, friable walls that are prone to rupture if left untreated (16, 17).

HEMODYNAMIC FORCES AND VASCULAR REMODELING IN AVF MATURATION AND COMPLICATIONS

Processes determining the success or failure of fistula maturation and the patency of the connected vessels are not yet fully understood. However, it is certain that numerous biomechanical factors significantly influence the long-term performance of the vessel junction. Reasons for the principal changes happening to the vein, that is the increase in its diameter and wall thickness, have a clear mechanobiological explanation. The vein, suddenly exposed to arterial, e.g. significantly higher, blood pressure, starts the process of remodeling so that it can withstand the new loading conditions. While the need for change in geometry of the vessel is easy to comprehend, the processes leading to it are not so much. Nevertheless, it has been shown that endothelial cells (ECs) in the intimal layer of the vein play a pivotal role in the intricate mechanism. Forming a contact layer between the blood and the vessel, these cells transcript the mechanical stimuli from the bloodstream into biochemical signals decisive for the vessel's response.

In this paper, the mechanism of remodeling is only discussed from the mechanical point of view, reviewing the literature on the relationship between the properties of blood flow and the vessel's response. For a thorough analysis of the biochemical side of the problematic, readers are referred to other literature, such as Sakurai and Sawamura (18), Pries, Reglin, and Secomb (19), González and Maldonado-Agurto (20), and sources cited therein. From biomechanical perspective, there are two forces acting on the ECs. The force exerted by the blood pressure, causing the vessel wall to dilate and stretch in circumferential direction, and the force arising from the viscous properties of the blood, causing the ECs to be stretched in the direction of the flow. The mechanosensitive nature of the ECs then allows to transduce information of the cell's deformations into chemical signals. The mechanical stimuli in the form of EC stretch change from their physiological state as the vein of AVF is exposed to arterial blood flow, initiating the vessel's remodeling (21, 22).

Since there is a direct relation between the blood-vessel wall interactions and the vessel's pathophysiological response, computational biomechanics have been employed in studies concerning the performance of the AVF. While it is deformations of the ECs that drive the remodeling process, stresses on tunica intima are usually assessed in such studies. The reason why stress is commonly in the focus of the research is that it can be computed without extensive knowledge of the vessel's mechanical properties, at least to a reasonable degree of accuracy (23, 24). Although the radial component of the blood pressure acting on the vessel wall might influence its response, it is the wall shear stress (WSS) that plays the major role in remodeling process (22, 25).

The WSS is caused by the viscous forces exerted by the streaming blood and is defined by its magnitude and direction. Laminar flow can be observed in majority of the circulatory system (26). For both steady and pulsatile laminar flow, the direction of the WSS remains aligned with the axis of the vessel in its straight segments (27). The WSS induced by undisturbed laminar flow is believed to maintain functional homeostasis in the vessel and inhibit EC and vascular smooth muscle cell (VSMC) proliferation (28). Geometric irregularities such as branching disturb the flow, changing its profile. Laminar vortex structures are usually observed in places of irregular geometry. As a result, drops in WSS may be observed at certain spots of vascular branching. Atherosclerotic lesions often develop in areas of low WSS, as has been observed in aortic bifurcations (29,30). Irregularities in the flow (and thus WSS) are introduced as AVF is created, which directly affect the process of vein remodeling and the development of various pathologies.

In principle, the venous part of the AVF is affected by two major changes upon the fistula creation – increase of blood pressure and geometric irregularity. Blood pressure and the average WSS in the vein increase as the vascular junction is created. Elevated WSS can be observed in the vein several centimeters distal to the junction, as demonstrated by numerical simulations (31, 32). In the past, high WSS was associated with the development of intimal hyperplasia, a common cause of AVF failure. However, it has been demonstrated that WSS remains elevated in matured fistulae, regardless of the AVF's outcome (33–35). In contrary, it is believed that steadily high WSS propagates outward remodeling, e.g. increase in diameter and media thickening, as opposed to the unfavorable inward remodeling in the form of VSMCs migration and intimal hyperplasia development (36, 37).

Increase of WSS can be observed in the distal segments of the connected vein. However, more complex stress profile is to be expected at the place of anastomosis and the adjacent swing segment of the vein. Nonuniform disturbed flow develops in area near the junction, with places of high, low, and oscillating WSS present in the region. A high-speed jet strikes the toe region of the vein opposite the anastomosis, inducing WSS of magnitude several times higher than on the surrounding tissue (31, 38). The increased WSS gradient has been shown to correlate with locations of calcified plaque in both artery and vein of the AVF (39).

Although significant increase of WSS is observed at the impact zone of the pulsating blood opposite the anastomosis, a low-velocity region is found in near proximity of the arteriovenous junction (40, 41). A correlation between low average WSS and intimal hyperplasia development was observed in these regions, leading to the belief that it is the decrease of WSS that promotes intimal thickening (42).

However, more recent studies have pointed out that it is not the average magnitude of WSS but rather its change in time that seem to be essential for the ECs biochemical reaction. It has been suggested in computational studies that the place of stenosis is often found in place of oscillatory WSS (32, 40, 43). The oscillations in the WSS direction are caused by the pulsatile nature of the flow, as the vein is introduced to the arterial blood flow pattern. The place of oscillatory WSS coincides with low average WSS as at the pulses only cause changes in magnitude rather than direction in the high-velocity regions. However, although both low and oscillatory WSS can be associated with intimal hyperplasia, it has been demonstrated in an in-vitro study that oscillatory flow leads to a disorganized intima structure development, promoting the inward remodeling that causes stenosis (44). In a unique longitudinal study, Soliveri et al. (45) show that stenosis develops at a place of disturbed flow in a patient over time. They suggest that the observed intimal thickening was caused by WSS instability resulting from disturbances in flow, reinforcing the idea that time-varying WSS is a key predictive marker of the pathological condition.

In summary, using biomechanical tools such as computational simulations, the insight into the pathophysiological processes in AVF has increased significantly in the past two decades. The main research focus has shifted from assessment of the WSS magnitude to the study of its disturbance and oscillations as its instability is linked to the development of vascular stenosis in AVF. The advances in computational methods have also allowed for the newly obtained knowledge on the matter to be used to enhance the techniques and methods used during the surgery and the hemodialysis process. Numerous studies have for example assessed the anastomosis angle and its effect on the blood flow parameters, suggesting that an acute angle of approximately 45 to 75° potentially leads to a more favorable flow pattern in the vein swing segment (46–48). Furthermore, the effect of the blood stream injected during the hemodialysis process at the place of the vascular access has been thoroughly studied, suggesting that further complications can be promoted by the hemodynamic conditions generated around the needle tip (49–51). Thus, although the precise connection between the mechanical stimuli, and the remodeling and pathological processes of the AVF are not yet fully understood, the results of the lates biomechanical studies have already proven to be invaluable for both understanding the mechanobiological principles and enhancing the performance of the arteriovenous junction.

CONCLUSION

Maintaining the arteriovenous fistula at the desired flow levels for dialysis with minimum complications after years of regular cannulations is still a clinical challenge despite decades of experience, research and upkeep optimizing. It is known that the surgical technique itself and individual patient-related factors, such as vessel quality or local anatomy, play crucial roles in determining AVF outcomes. According to our findings, recent studies of AVF biomechanics have revealed that the wall shear stress, with pressure gradients and flow turbulence, influences endothelial behavior and causes vessel wall remodeling, mainly with disturbed or oscillatory WSS. These underlying conditions initiate intimal hyperplasia and stenosis, which can also lead to aneurysm formation.

Computational modeling has brought detailed mapping of WSS distribution and identified high-risk zones for potential stenosis. Described revelations affected surgical planning of AVF creation and have led to improvements for example in optimizing anastomosis angles for better flow geometry and cannulation techniques for hemodialysis.

Optimizing arteriovenous fistula performance requires thoroughly understanding how hemodynamic forces drive vascular remodeling. Recent progress in biomechanics and computational methods has clarified the role of shear stress, especially its disturbances, in both the maturation process and pathological outcomes. Joining biomechanical understanding with the best AVF design strategies and a correct clinical approach will ensure prolonged AVF functioning and a better life for ESRD patients.

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