

A Novel In-Vitro Platform to Answer How, When, and Why Clots Embolize

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Abstract

Purpose: Blood clots that form intra-vascularly can break off, or embolize, and occlude a distal site, leading to deadly sequelae such as strokes and pulmonary embolisms. Embolization is a highly mechanical phenomenon during which hemodynamic forces overcome a clot's resistance to wall delamination or bulk fracture. While we and others have characterized these resistances, no prior work has experimentally demonstrated their role in actually promoting embolization. Thus, our objective was to build an in vitro platform to study how, when, and why clots embolize.

Methods: We designed a pulsatile flow loop in which clot mimics made from human blood are coagulated onto a porcine aortic substrate, creating a realistic clot/vascular interface. We prepared clots at 20%, whole blood, and 60% hematocrit from six female and six male donors and subjected them to a scaled physiological venous flow profile. We measured intra-cycle shear strain and

cycles-to-embolization and used a Monte Carlo approach to correlate both with previously published clot stiffness and fracture toughness data.

Results: With increasing hematocrit (20%, whole blood, and 60%), intra-cycle shear strain increased from 0.276 ± 0.031 to 0.320 ± 0.037 and 0.426 ± 0.051 , while cycles-to-embolization decreased from $13,206 \pm 2,072$ to $8,618 \pm 1,570$ and $4,253 \pm 1,425$, respectively. Neither sex nor clot height had a consistent effect. Furthermore, shear strain was negatively correlated with clot stiffness, while cycles-to-embolization were positively correlated with fracture toughness.

Conclusion: Our platform enables the experimental study of clot embolization. We show that increased hematocrit leads to larger clot deformation and faster embolization, likely by decreasing clot stiffness and toughness.

Keywords: Stiffness, Toughness, Stroke, Heart Attack, Pulmonary Embolism, Thrombus, Thrombosis

1 Introduction

Blood clots are both physiologically critical and pathologically devastating [1]. When clots form under physiological conditions, they seal vascular injuries and provide a scaffold for wound healing [2]. In other words, they are vital to our survival. However, under pathological conditions — that is, when forming intra-vascularly — they can lead to vessel occlusion and deadly sequelae such as strokes, heart attacks, and pulmonary embolisms [3, 4]. In the latter case, clots often form at one site, break off or embolize, and occlude a distal site [5]. Embolization itself is a highly mechanical phenomenon during which external (hemodynamic) forces overcome the clots’ resistance to vascular wall adhesion or bulk fracture [6]. Very little is understood about the mechanics of embolization, limiting our ability to fundamentally understand and predict one of the most common causes of morbidity and mortality.

We and others have previously investigated cohesive and bulk clot fracture [7–10]. The consensus is that a clot’s ability to resist wall delamination or internal fracture depends on a number of compositional and physiological factors [11, 12]. For example, it can be shown that clot hematocrit (i.e., the volume fraction of red blood cells) critically drives clot resistance to cohesive and bulk fracture [12–14]. In addition, we have investigated demographic and physiological factors such as sex, age, and biomarkers of inflammation that play measurable roles in regulating a clot’s ability to resist external forces [12, 14].

However, much of this work has been circumstantial. That is, while we understand that cohesive strength and fracture toughness are critical material properties influencing a clot’s propensity to embolize, we have failed to demonstrate their role in actually promoting embolization. While recent work has shown this computationally [15, 16], for example, Ref. [17] has used a fluid-structure interaction framework based on peridynamics to simulate both fracture- and delamination-based embolization [17]. There are no prior accounts of experimental work doing the same.

Thus, the objective of our work is to address this critical gap by experimentally advancing our understanding of clot embolization mechanics. Specifically, we built an

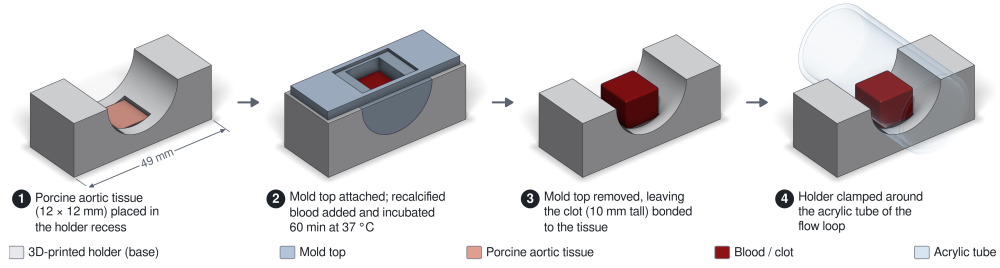
in vitro embolization experiment in which we mount clot mimics made from human blood to a vascular segment and control a pulsatile pump to create a vasculature-equivalent hemodynamic load. Subsequently, we measure the flow-induced clot strain and the number of cycles to failure. We do so for clots of three hematocrit levels and for both female and male blood donors.

2 Materials and Methods

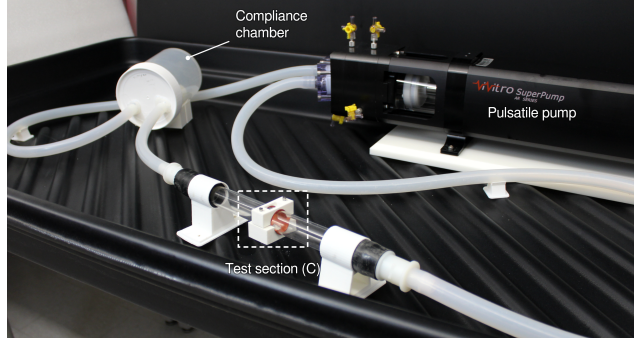
2.1 Flow Loop

We designed a versatile flow loop with the ability to mimic pulsatile flow through a variety of vascular segments. To this end, we combined a pulsatile pump (ViVitro Labs Inc., Victoria, BC, Canada, SuperPump AR Series), a custom compliance chamber, and our sample holder; see Figure 1B for a schematic of our loop. The latter forms the central experimental innovation of our work. Specifically, we built a 3D-printed cylindrical section that hosts a biological vascular segment. This segment serves as a physiological substrate for our in vitro coagulated clot samples. A detailed plan of this segment is depicted in Figure 1A. Procedurally, the clot is coagulated onto the vascular segment, and the clot/wall/holder complex is subsequently mounted to our flow loop. Our clot sample creation is described below.

A Clot sample preparation on a vascular substrate



B Pulsatile flow loop



C Test section

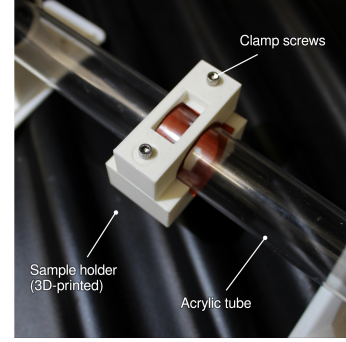


Fig. 1 Experimental setup and design. A) Clot sample preparation on a vascular substrate: porcine aortic tissue is placed in a 3D-printed holder, recalcified blood is added and incubated for 60 minutes at 37°C, and the mold top is removed to leave a 10 mm tall clot adhered to the tissue. B) The pulsatile flow loop, consisting of the ViVitro pump, a compliance chamber, and the test section. C) Detailed view of the test section, showing the 3D-printed sample holder clamped around the acrylic tube.

2.2 Blood Collection and Preparation

Following the guidance and approval of the Institutional Review Board at The University of Texas at Austin, we collected venous blood samples from healthy volunteers. Written informed consent was obtained from all participants prior to blood collection. We drew blood into acid citrate dextrose (ACD) anticoagulant and stored it at 4°C until further processing. Please note that all samples were prepared and tested within 24 hours of collection. In total, we included six male and six female subjects in this study.

We prepared clots across three red blood cell (RBC) compositions per subject: 20% RBCs, 60% RBCs, and whole blood (WB), with the latter reflecting each subject's native hematocrit (35% to 45%). To prepare the 20% and 60% clots, we centrifuged blood at 3200 RPM for 10 minutes to separate the plasma and RBCs, then recombined them at the desired volume fractions.

Prior to clot formation, we lined custom molds with a 12 × 12 mm section of porcine aortic tissue, see Figure 1A. The tissue was stored at -80°C and thawed prior to use. Then, to initiate coagulation, we added calcium chloride to blood aliquots to a final concentration of 20 mM and gently mixed. We then added blood to the mold

and incubated samples at 37°C for 60 minutes. This resulted in 10 mm tall samples, which were then inserted into the flow loop.

2.3 Experimental Design

Each subject yielded three clot samples at 20%, WB, and 60% hematocrit. After mounting each sample in our flow loop, we imposed a physiological flow profile representing that of a common iliac vein. Given the differing physical dimensions of our setup, we used Strouhal, Reynolds, and a form of the Ohnesorge number to scale all pump parameters.

Upon mounting each sample, we monitored its flow-induced deformation using recordings from an in-loop baroscope (Stone Digital Company, Kowloon, Hong Kong). To this end, we manually marked the clot height in all images at three distinct points and computed the average. Based on the intra-cycle displacement of our samples, we then computed clot shear strain as per Equation 1. In this equation, H represents the height at rest, while h represents the deformed height. Here, we assume that the clots undergo simple shear and behave incompressibly.

$$\gamma = \frac{\sqrt{h^2 - H^2}}{H} \quad (1)$$

We subsequently monitored the experiment visually until we observed clot embolization and noted the cycle-to-embolization for each clot.

2.4 Statistical Analysis

We conducted statistical analysis in R (The R Foundation, Vienna, Austria, Version 4.1.2). We conducted three independent tests. First, we tested the relationship between clot composition and clot deformation (via shear strain). Second, we tested the relationship between clot composition and cycle-to-embolization. We did so using a linear mixed model in which subject served as a random effect, and in which composition and sex served as fixed effects. In addition, we included clot height (measured before the start of the experiment) as a potential confounding effect and also as a fixed effect.

To mechanistically explore the relationship between clot composition and strain, as well as cycle-to-embolization, we also quantified the relationships between clot composition, clot stiffness, and fracture toughness, along with our two primary outcomes. To do so, we used data from a previous study and a Monte Carlo approach. Specifically, we previously collected data on how hematocrit (20%, WB, and 60%) affected various measures of clot mechanics, including stiffness and fracture toughness. For a detailed explanation of the meaning of these measures and how we measured and computed them, please see our prior work [cite]. Because those data were collected on a similar population but different subjects, we used a Monte Carlo sampling approach to estimate the correlation between these mechanical measures and our two outcomes: intra-cycle shear strain and time-to-embolization. To this end, in each of 5,000 iterations, we randomly paired each subject in our study with a subject of the same sex from the prior study. Subsequently, we fitted a linear regression to the paired data points in each iteration.

For all statistical tests, we defined significance as $p < 0.05$ and we report all data as mean $\pm$ standard deviation.

3 Results

3.1 Effect of composition on embolism risk

In our experiment, we measured two outcomes: i) shear strain, and ii) cycles-to-embolization. Figure 2A shows shear strain for the 20%, WB, and 60% hematocrit groups. Overall, we find that an increased RBC count is associated with an increase in shear strain. Specifically, the 20% group yielded an intra-cycle shear strain of 0.276 ± 0.031 , while the WB and 60% groups yielded 0.320 ± 0.037 and 0.426 ± 0.051 , respectively. Similarly, increased hematocrit results in fewer cycles-to-embolization. That is, the 20% group resulted in $13,206 \pm 2,072$ cycles-to-embolization, while the WB and 60% groups resulted in $8,618 \pm 1,570$ and $4,253 \pm 1,425$ cycles-to-embolization, respectively.

Interestingly, we found that sex did not have a significant impact on most shear strain or cycles-to-embolization groups. The only group to have a significant difference by sex was the whole blood shear strain group ($p = 0.013$). Similarly, clot height did not impact either shear strain ($p = 0.07$) or cycles-to-embolization ($p = 0.96$).

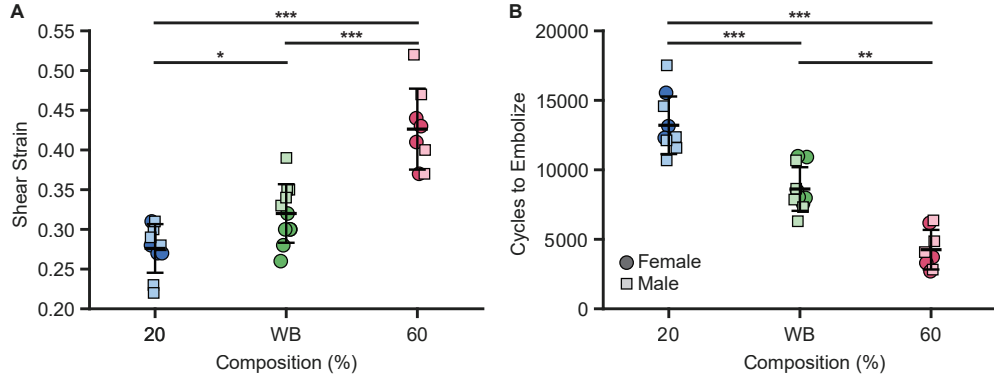


Fig. 2 A) Shear strain increased with increasing hematocrit. There was no significant difference in sex in the 20% or 60% RBC group, but there was a significant difference in the whole blood group ($p = 0.013$). B) Cycles to embolize decreased with increasing hematocrit, meaning samples with a higher hematocrit embolized sooner than those with a lower percentage of RBCs. Asterisks indicate statistical significance (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$).

3.2 The relationship between mechanical properties, composition, and embolism risk

We used a Monte Carlo simulation where subjects were randomly paired with each other between two different mechanical studies (ours and a previously published study) to find correlations between these different parameters. From our data, we used shear

strain and cycles-to-embolization. In the previous data, we took stiffness and fracture toughness. As seen Figure 3, stiffness and fracture toughness differed significantly between hematocrit groups, except between the whole blood and 60% RBC groups for stiffness. Figure 4 shows the two relationships of interest. We found that intra-cycle shear strain was negatively correlated with stiffness, with a slope of -0.021 kPa^{-1} . Conversely, cycles-to-embolization were positively correlated with fracture toughness, with a slope of 2,952 cycles per J/m^2 . Both relationships were significant across all Monte Carlo iterations.

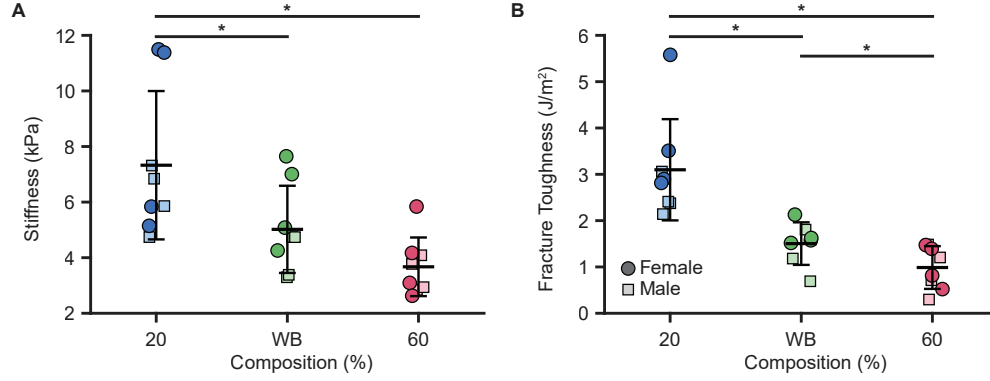


Fig. 3 Mechanical data from a previous study showed that (A) stiffness and (B) fracture toughness decreased with increasing hematocrit. Asterisks indicate statistical significance (* $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$).

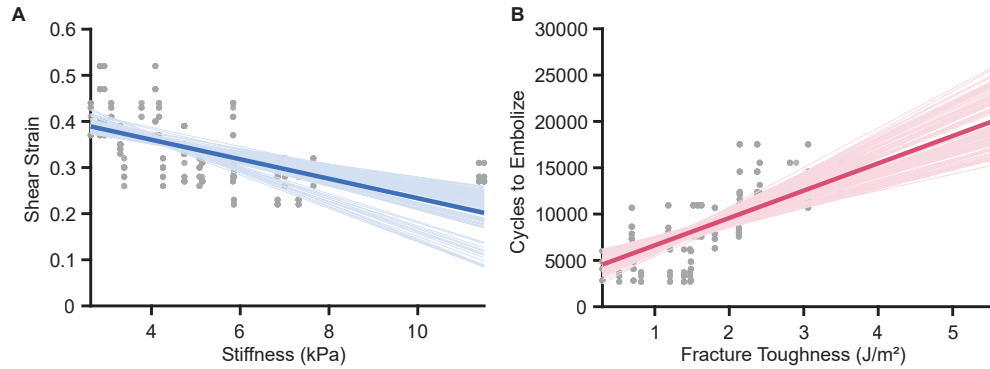


Fig. 4 Results of Monte Carlo simulations ($n = 5,000$). In each iteration, individuals were randomly paired with another individual of the same sex, and a linear regression was fitted to the paired data. We found a negative correlation between (A) stiffness and shear strain and a positive correlation between fracture toughness and cycles to embolize (B). All results from Monte Carlo simulation were significant.

4 Discussion

Our goal was to create a versatile experimental platform to study blood clot embolization. To test our platform, we investigated the role of hematocrit on both intra-cycle strain and time-to-embolization. Additionally, we used previously published mechanical data correlating clot composition and clot mechanical properties to mechanistically explore the role of hematocrit on our two primary outcomes. In summary, we found that composition significantly increases intra-cycle strain and reduces time-to-embolization. Furthermore, we found significant correlations between clot stiffness and fracture toughness, and intra-cycle shear strain and time-to-embolization, respectively.

First, our successful experiments confirm that our *in vitro* experimental framework can be effectively used to study and understand clot embolization. Among our primary innovations, we highlight our vascular substrate, which creates a realistic clot/vascular interface. In contrast, others have coagulated clot against artificial surfaces that likely yield unrealistic surfaces and possibly confound physiological measurements, such as time-to-embolization [18–20]. Additionally, we want to highlight that the controllability of our pulsatile pump, in combination with our non-dimensional analysis, allows the replication of many flow profiles. That is, while we focused on flows representing those of the common iliac vein, changes to pump parameters allow mimicking flows in other vital veins and arteries.

Second, and to our specific findings, it has been shown by us and others that clot composition affects clot mechanical properties [12–14]. However, how changes in these properties affect intra-cycle strain and time-to-embolization may have been suspected, but never experimentally shown. Thus, our work fills a specific knowledge gap that could critically support our understanding of embolization. For instance, clot composition may serve as a surrogate for embolization risk [21, 22]. This is particularly useful as we and others have demonstrated that clot composition may be non-invasively estimated through various imaging modalities including computed tomography and magnetic resonance imaging [23–27].

Our findings are also well supported by prior computational studies. For example, computational frameworks using peridynamic and fluid-structure interaction models have shown that clot fracture and delamination resistance govern embolization onset under flow [17]. Further, other work has shown that an embolus’s material properties influence the hemodynamic stresses it experiences. In turn, this impacts its likelihood of further fracture as it travels through downstream vasculature [28]. In that, our work not only demonstrates the technical value of our setup and fills direct experimental knowledge gaps, but it also serves to validate previously unvalidated computational studies and may serve future computational studies. To this end, all of our data will be made openly available through the Texas Data Repository upon acceptance.

It is important to note the limitations of our study. First, our clots do not represent the vast diversity of *in vivo* clots. That is, our clots are coagulated from fresh venous blood under static conditions. Thus, they lack the anisotropy and heterogeneity induced under shear flow [29–31]. Additionally, our clots lack the compositional complexity of *in vivo* clots that may contain lipids, calcium, collagen, and many other byproducts of clot maturation [4, 32, 33]. Future research – using our setup – should explore the role of clot heterogeneity and compositional complexity on intra-cycle

shear strain and time-to-embolization. Second, our study included only healthy and young subjects. This is important as we and others have shown that age and disease significantly affect blood clot mechanics and thus, likely, its embolization risk [34–36]. Future studies should include a more diverse subject pool with a higher sample size.

In conclusion, we designed an in vitro experiment to study blood clot embolization. We tested our setup by investigating the impact of clot composition on intra-cycle shear strain and on cycle-to-embolization. We found that increased hematocrit leads to larger clot deformation and faster embolization. In addition, we combined our data with prior findings from our lab to show that increased intra-cycle shear strain is likely due to increasing hematocrit decreasing stiffness, and that faster embolization likely stems from increasing hematocrit decreasing toughness. Our findings shed new experimental light on compositional mechanisms driving clot embolization.

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Declarations

Manuel K. Rausch has a speaking agreement with Edwards Lifesciences. The other authors have no conflicts to declare.

Data availability. All data supporting this study will be uploaded to the Texas Data Repository upon acceptance.

Use of AI tools. Generative AI tools were used to assist with editing the manuscript text, to support code development for data analysis, and to assist in preparing Figure 1. All AI-assisted content was reviewed, verified, and revised by the authors, who take full responsibility for the content of this manuscript.

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