Platelets

ISSN: 0953-7104 (Print) 1369-1635 (Online) Journal homepage: www.tandfonline.com/journals/iplt20

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To cite this article: Per Sandgren , Shaikh Faisal Zaman & Ina Laura Perkins (2026) Functionality of platelets after in vitro testing of pulsatile total artificial hearts, Platelets, 37:1, 2741743, DOI: 10.1080/09537104.2026.2741743

To link to this article: https://doi.org/10.1080/09537104.2026.2741743

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PLATELETS
2026, VOL. 37, NO. 1, 2741743
https://doi.org/10.1080/09537104.2026.2741743

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BRIEF REPORT

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Functionality of platelets after in vitro testing of pulsatile total artificial hearts

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ABSTRACT

Hemolysis is a metric of hemocompatibility in total artificial hearts (TAHs) but provides limited insight into platelet biology and thrombogenicity. We demonstrated reduced hemolysis with Realheart® TAH (RH) compared to FDA-approved SynCardia TAH (Sync). Here, we evaluated platelet function following exposure to both devices in a whole-blood in vitro system using a standardized ASTM F1830 protocol. Human blood ($n = 5$) was circulated for 360 min under controlled conditions (5 L/min, 100 mmHg, 37 °C) in RH, Sync, while matched static controls were maintained under identical temperature conditions. Platelets were assessed using metabolic (ATP, mitochondrial potential), phenotypic (surface markers, microparticles), and functional (agonist response, thromboelastometry) assays. Differences were observed between circulating and static conditions, including higher pH, increased lactate, and reduced platelet surface receptor expression, consistent with the non-physiological nature of static controls. Despite these changes, no evidence of increased platelet activation (CD62P), thrombogenicity ($CD61^+$ microparticles), or mitochondrial damage (ATP, JC-1) was observed, and platelet function remained preserved by agonist stimulation and thromboelastometry. SynCardia increased mean platelet volume and reduced ATP levels, suggesting altered particle composition rather than intrinsic platelet dysfunction. These findings indicate preserved platelet integrity under clinically relevant TAH conditions and highlight that hemolysis and platelet responses represent distinct aspects of device hemocompatibility.

ARTICLE HISTORY
Received 18 June 2026
Revised 18 August 2026
Accepted 25 September 2026

KEYWORDS
Biocompatibility; Hemocompatibility; platelets; thrombogenicity; total artificial heart

Introduction

Heart failure is a global health burden affecting tens of millions of people, with prevalence expected to exceed 74 million by 2030,[1] and ~50% 5 years mortality.[2] With only ~10,287 donor hearts transplanted each year,[3] demand for Total Artificial Hearts (TAHs) is growing. SynCardia TAH (Sync) implanted in >2,100 patients, has demonstrated clinical utility but is associated with blood-related complications including bleeding, anemia, and infection.[4]

The pulsatile 4-chamber Realheart® TAH (RH) mimics native atrioventricular plane displacement[5] aiming to reproduce physiological flow patterns. Previous CFD,[6] hybrid simulation,[7] and animal studies,[8] have characterized the hemodynamic behavior of RH and demonstrated the ability to modulate cardiac output via beat rate, stroke volume, and systolic/diastolic timing, maintaining left-right balance.[7]

Hemolysis testing is a regulatory endpoint, with Sync representing the only FDA-approved TAH comparator. We previously demonstrated that RH produces significantly lower hemolysis under in vitro physiologically relevant left-sided (systemic) conditions,[9] while clinical studies have reported substantial hemolysis in Syncardia-supported patients.[4,10] However, hemolysis provides only a partial assessment of hemocompatibility and does not capture device-induced effects on platelet biology or thrombogenicity.

Platelet activation is a key driver of thrombotic complications in mechanical circulatory support, yet standardized evaluation methods in TAH systems are lacking. Studies of Sync used isolated platelets and shear-dependent thrombin generation, demonstrating increased activation at higher operating conditions with levels exceeding those of mechanical valves alone.[10] While informative, these approaches do not reflect the integrated response of platelets with whole blood.

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Supplemental data for this article can be accessed online at https://doi.org/10.1080/09537104.2026.2741743

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In this study, we extend our prior hemolysis findings by assessing platelet function in a clinically relevant whole-blood circulation model following a standardized ASTM F1830 protocol. The study objective was to determine whether total artificial heart circulation impairs platelet functionality and whether platelet responses differ between Realheart® TAH and SynCardia TAH.

Materials and methods

TAH systems and blood loop testing

Two pulsatile TAHs were evaluated: the clinically established SynCardia TAH driven by the Freedom™ driver (Syncardia Systems LLC, Tucson, AZ, USA) and a prototype of the Realheart® TAH (V11.2) (hereafter RH; Figure 1). Testing was performed under physiologically relevant systemic circulation conditions using a validated in vitro blood loop model.[9,11–13]

Human blood preparation

Fresh human non-leukoreduced whole blood units anticoagulated with citrate–phosphate–dextrose (CPD) was obtained from healthy male donors via the Blood Center at Karolinska University Hospital following informed consent for research use. Platelet counts between 130–240 $\times 10^9$/L were required for inclusion. Blood from three to four ABO-compatible donors (B or AB, identical RhD status) was pooled and stored overnight at 4–8°C. Pooling was performed to reduce donor variability and obtain sufficient blood volume for testing. Information regarding individual donor medication use was not available to the investigators, as anonymized blood units were obtained through routine blood-center procedures.

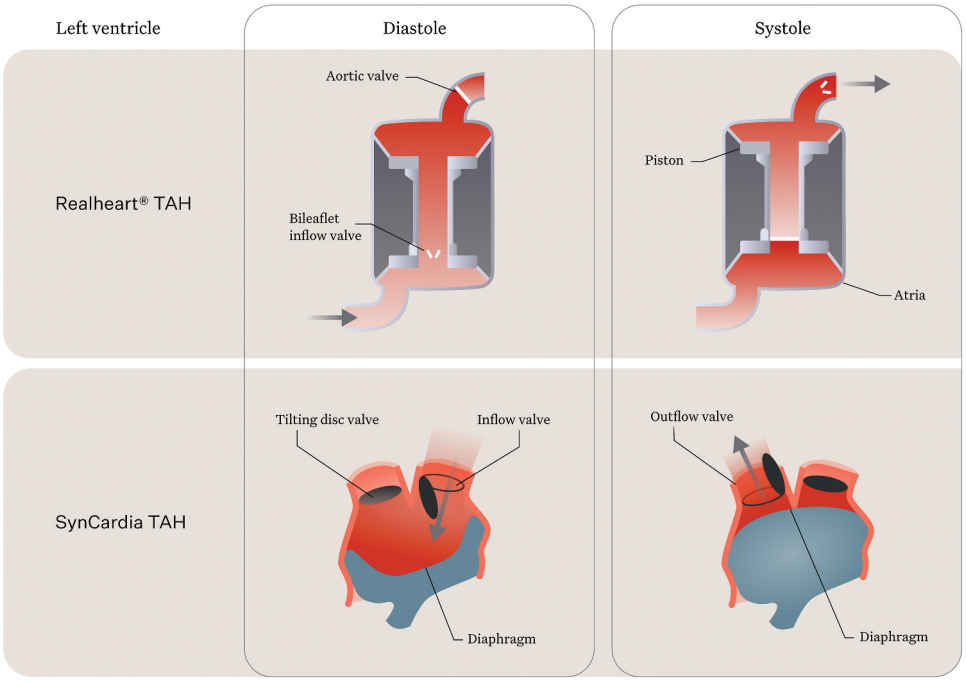

Figure 1. Schematic of TAH devices. the Realheart® TAH acts as a piston pump in which, during diastole, blood enters the artificial atrium and passes through the bileaflet mitral valve to fill the atrioventricular (AV) plane cylinder and artificial ventricle. During systole, the AV-plane moves toward the ventricle, increasing ventricular pressure, which leads to closure of the mitral valve and opening of the aortic bileaflet outflow graft valve. The reciprocating motion of the AV-plane is actuated by an electrically driven rack-and-pinion system. The SynCardia TAH ventricle is filled through a tilting-disc inflow valve during diastole. During systole, the pneumatically driven diaphragm inflates, increasing ventricular pressure and causing closure of the inflow valve and opening of the aortic outflow valve, thereby ejecting blood.

Prior to testing, blood was diluted to a hematocrit of 29–32% in accordance with ASTM F1830-17 using phosphate-buffered saline, sodium bicarbonate for pH adjustment, and antibiotic/antimycotic supplementation to prevent contamination in the non-sterile system, as described previously.[9]

Blood loop configuration and experimental design

Pulsatile blood loops incorporating atrial and arterial compliance chambers were filled with 550 ml of blood and configured to achieve a target differential pressure of 100 ± 3 mmHg at a flow rate of 5.0 ± 0.25 L/min (ASTM F1841-97). Temperature was maintained at 37 ± 1°C using a circulating heated water bath connected to the compliance chamber. Loop temperature was monitored using a thermocouple probe within the circulation circuit.

Seven independent pooled-blood preparations were used: four were tested in non-paired circulation pilot experiments (two in the SynCardia TAH and two in the Realheart® TAH), while three were tested sequentially in paired tests in both devices. This resulted in five independent circulation experiments (biological repeats) for each TAH group. Static controls consisted of blood from the same pools maintained at 37 ± 1°C for 360 minutes without exposure to pumping or circulation, to distinguish device-induced effects from ex vivo incubation changes.

Platelet-rich plasma preparation

Whole blood samples were collected at baseline (blood pool prior to circulation) and after 360 min of circulation. Platelet-rich-plasma (PRP) was obtained within 30 minutes of collection by differential centrifugation (200 × g and 800 × g), with minimal agitation to avoid artefactual activation, and used for all platelet analyses.

In vitro testing

Platelet count and mean platelet volume (MPV) were determined in platelet-rich plasma (PRP) using an automated hematology analyzer (Cellguard CA-620, Boule Medical AB, Solna, Sweden). The extracellular metabolic environment was assessed using a clinical blood gas analyzer (ABL800 FLEX, Radiometer Medical ApS, Copenhagen, Denmark), measuring pH, pCO_2, pO_2, glucose, lactate, and bicarbonate at 37°C.

Platelet ATP content was quantified using a luciferase-based luminescence assay (BioThema AB, Handen, Sweden) following protein precipitation and normalization to platelet count. Mitochondrial membrane potential ($\Delta\psi$) was assessed in unfixed platelets using the JC-1 assay (ImmunoChemistry Technologies, Bloomington, MN, USA), with intact $\Delta\psi$ expressed as the percentage of JC-1–positive cells.

Flow cytometric analyses were performed on a CytoFLEX flow cytometer (Beckman Coulter Life Sciences, Indianapolis, IN, USA). Platelet responsiveness was assessed on unfixed platelets. Platelets were identified using a predefined FSC/SSC gate, after which PAC-1 binding (Becton Dickinson, Franklin Lakes, NJ, USA) was measured following stimulation with ADP or collagen. Surface receptor expression (CD62P, CD42b, CD61/CD41; Beckman Coulter Life Sciences, Indianapolis, IN, USA, GPVI; BD Pharmingen/Becton Dickinson, San Diego, CA, USA, and PECAM-1; Sigma-Aldrich, St. Louis, MO, USA) was evaluated on fixed platelets using fluorochrome-conjugated monoclonal antibodies. Microparticles were analyzed using CD61 (Beckman Coulter).

Global platelet contribution to coagulation was assessed using rotational thromboelastometry (ROTEM delta 3000, TEM International GmbH, Munich, Germany) performed on PRP with EXTEM activation to focus on platelet-dependent clot formation, recording clotting time, clot formation time, and maximum clot firmness.

Statistical analysis

All data are presented as mean ± standard deviation (SD). Group comparisons were performed using one-way analysis of variance (ANOVA). Homogeneity of variances was confirmed using Brown–Forsythe and Bartlett's tests. Three groups were analyzed ($n = 5$ per group; 15 total observations). A $p < .05$ was

considered statistically significant. Analyses were performed using GraphPad Prism (GraphPad Software, San Diego, CA, USA).

Results and discussion

Platelet responses were assessed following circulation of human whole blood in two total artificial heart (TAH) systems—the clinically established SynCardia TAH (Sync) and the Realheart® TAH prototype V11.2 (RH)—and compared with static controls. Across metabolic, phenotypic, and functional endpoints, platelet behavior was largely preserved under circulatory conditions, with no evidence of increased activation or functional impairment in either TAH system. The concordance of findings across independent analytical approaches strengthens the interpretation that platelet compatibility is maintained during pulsatile TAH circulation under the tested conditions.

Platelet characteristics nevertheless displayed selective alterations across the experimental groups. Although platelet counts in platelet-rich plasma (PRP) were comparable between RH, Sync, and static conditions (Figure 1A), indicating similar platelet recovery following circulation, this observation contrasts with previously reported whole-blood hematology findings obtained from the same circulation experiments. In those analyses, SynCardia-treated blood exhibited significantly elevated platelet counts compared with both Realheart® and static controls.[9]

The increased platelet counts previously observed in whole blood following SynCardia circulation are consistent with previous reports describing leukocyte fragmentation and platelet-sized cellular debris during mechanical circulatory support.[14–16] Such findings should, however, be interpreted cautiously and may not represent a true increase in circulating platelets. Rather, these findings may point toward an altered composition of the cellular or particulate fraction. Supporting this interpretation, mean platelet volume (MPV) was significantly increased in SynCardia-derived PRP (Figure 2B), consistent with a shift toward larger particles and a broader size distribution within the PRP fraction.

ATP concentrations were significantly lower in SynCardia samples despite unchanged platelet counts (Figure 2C). This may reflect dilution of platelet-derived ATP within a heterogeneous particulate population, although no direct assessment of cellular fragmentation was performed.

Mitochondrial membrane potential ($\Delta\psi$), assessed by JC-1 positivity, remained preserved across all groups (Figure 2D), indicating maintained platelet bioenergetic integrity and argues against substantial platelet damage (since mitochondrial depolarization constitutes an early marker of irreversible platelet injury). Collectively, these findings suggest that the MPV and ATP changes more likely attributable to non-platelet material within the PRP fraction than intrinsic platelet dysfunction.

The extracellular metabolic environment differed primarily between circulating and static conditions. Blood exposed to TAH circulation maintained pH within the physiological range at 37°C, whereas static blood showed a significant reduction in pH (Figure 3A). This was accompanied by numerically higher pCO_2 and altered pO_2 in static samples (Figure 3B–C), although these differences did not reach statistical significance. The observed acidification under static conditions is consistent with ongoing cellular metabolism in the absence of convective gas exchange. Lactate levels were modestly increased following circulation (Figure 3E), while glucose and bicarbonate concentrations remained unchanged (Figure 3D,F), indicating preserved buffering capacity and substrate availability. Together, these findings suggest that the observed metabolic differences primarily reflect the non-physiological nature of static conditions rather than device-specific effects.

Phenotypic analysis revealed no increase in platelet activation following TAH exposure. CD62P expression was comparable across RH, Sync, and static samples (Figure 3G), indicating similar levels of platelet surface activation across conditions. In contrast, differences were observed in surface expression of CD42b (Figure 3H), CD61, CD41, GPVI, and PECAM-1 in circulating samples compared with static controls (Figure 4A–D). These receptors are known to be regulated by shear and contact with artificial surfaces.[17] Together with the absence of concomitant CD62P expression, no increase in CD61-positive microparticles (Figure 4E), and preserved platelet function, the findings suggests minor receptor modulation rather than pathological platelet activation. Stimulation with ADP or collagen elicited the expected increases in platelet activation relative to unstimulated samples, with no differences between groups (Figure 4F). This demonstrates intact agonist-dependent signaling and integrin activation following TAH exposure. Similarly,

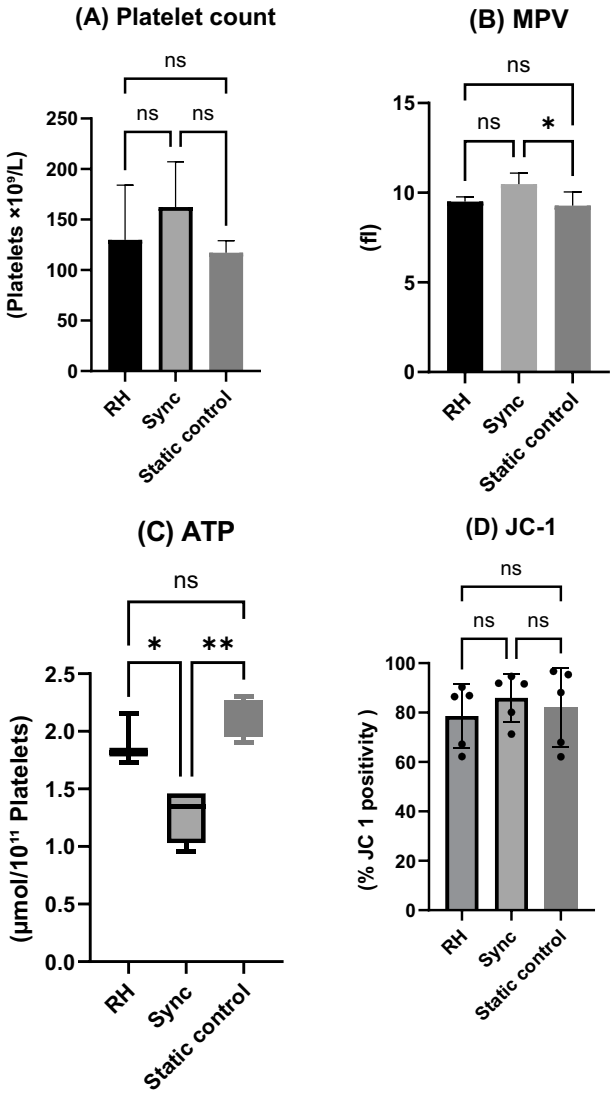

Figure 2. Basic platelet characteristics following circulation in total artificial heart (TAH) systems. Human blood was exposed to either the clinically established SynCardia TAH (Sync) or a newer prototype of the Realheart® TAH (RH; V11.2) and compared with static controls. Data are presented as mean ± SD ($n = 5$) and include platelet count (A), mean platelet volume (MPV; B), intracellular adenosine triphosphate (ATP; C) levels, and mitochondrial membrane potential assessed by JC-1 positivity (D). Statistical analysis was performed using one-way ANOVA. *$p < .05$; **$p < .01$; ns, not statistically significant.

viscoelastic testing demonstrated comparable clotting time, clot formation time, and maximum clot firmness between groups (Figure 4G–I), indicating preserved platelet contribution to global clot formation and mechanical clot stability. Concordant flow-based responses and viscoelastic parameters support preserved platelet function.

Notably, the absence of functional differences between RH and Sync was observed despite previous reports of higher hemolysis associated with the SynCardia TAH.[9] This dissociation supports the concept that erythrocytes and platelets represent distinct hemocompatibility endpoints with different susceptibility thresholds. Platelet dysfunction typically requires higher or more sustained non-physiological shear or metabolic stress than that required for red cell lysis.[14,18] The present data therefore indicate that both TAH systems operate within a shear environment permissive for platelet integrity, even if other blood elements are differentially affected.

Taken together, the consistency of platelet responses across metabolic, phenotypic, and functional assays indicates that exposure to TAH circulation does not compromise platelet function under the

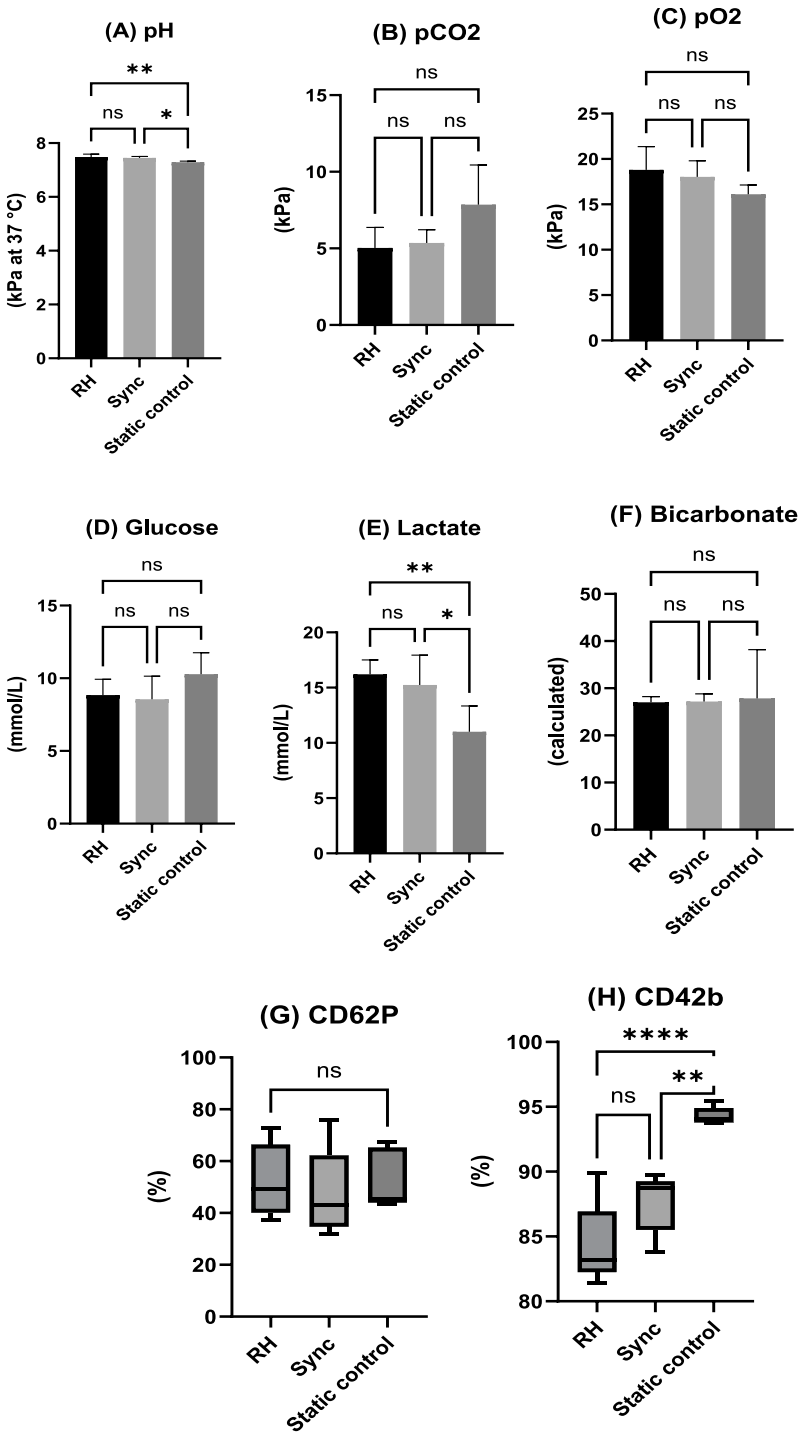

Figure 3. Extracellular metabolic profile and platelet surface markers in human blood exposed to total artificial heart (TAH) circulation. Human blood was circulated in either the SynCardia TAH (Sync) or the Realheart® TAH prototype (RH; V11.2) and compared with static controls. Measured parameters included pH (A), partial pressure of carbon dioxide (pCO_2; B), partial pressure of oxygen (pO_2; C), glucose concentration (D), lactate concentration (E), and bicarbonate concentration (F) at 37°C, as well as CD62P expression (G) and CD42b expression (H). Data are presented as mean ± sd ($n = 5$). Statistical analysis was performed using one-way ANOVA. *$p < .05$; **$p < .01$; ***$p < .001$; ns, not statistically significant.

conditions tested. The minor phenotypic differences observed were not associated with altered activation or hemostatic performance. In this context, the Realheart® TAH prototype demonstrated platelet compatibility comparable to the clinically established SynCardia TAH. Importantly, inclusion

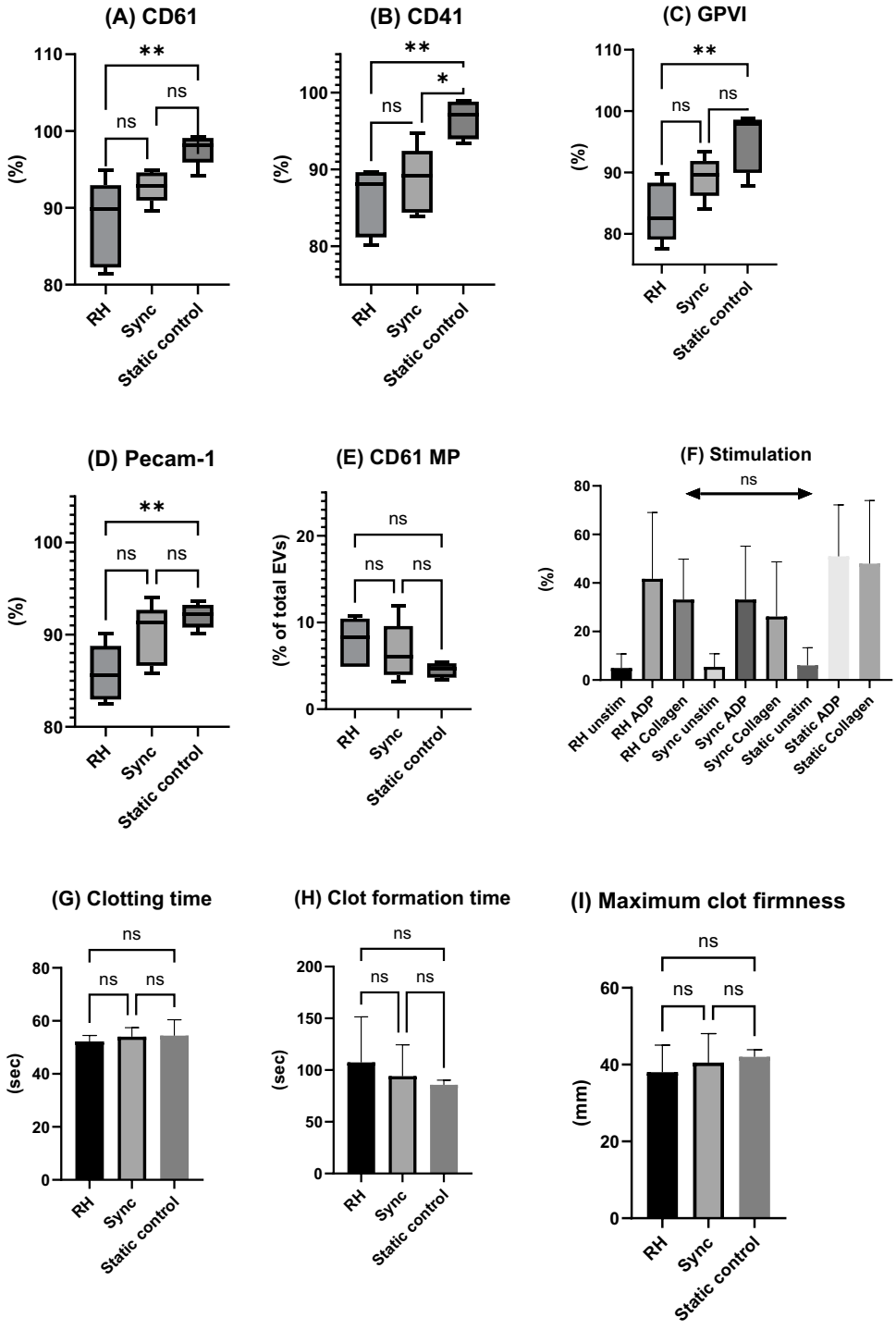

Figure 4. Platelet surface markers, microparticle (MP) release, agonist-induced responsiveness, and viscoelastic properties following exposure to total artificial heart (TAH) systems. Human blood was circulated in the SynCardia TAH (Sync) or the Realheart® TAH prototype (RH; V11.2) and compared with static controls. Platelet surface expression of CD61 (A), CD41 (B), GPVI (C), and PECAM-1 (D), CD61-positive microparticles (E), platelet responsiveness to stimulation (F), as well as clotting time (CT; G), clot formation time (CFT; H), and maximum clot firmness (MCF; I) were assessed. Data are presented as mean ± sd ($n = 5$). Statistical analysis was performed using one-way ANOVA. *$p < .05$; **$p < .01$; ***$p < .001$; ns, not statistically significant.

of static control provides a necessary baseline, enabling interpretation of observed effects relative to unexposed blood and thereby strengthening the assessment of device-induced changes in platelet behavior.

Study limitations

The present study was conducted under controlled in vitro conditions using pooled healthy donor blood and a 6-hour circulation protocol. While this approach follows established ASTM hemocompatibility testing standards and allows direct device comparison, it does not reproduce the complex inflammatory, endothelial, coagulation, and hematologic responses present in TAH patients. Consequently, the findings should be interpreted as an assessment of relative device effects under standardized conditions rather than a direct prediction of clinical outcomes. This study was limited to systemic (left pump) circulation to maintain consistency with prior methods,[9,11,12] and future work should include combined systemic and pulmonary loops for a more comprehensive assessment of TAH hemocompatibility. The mechanism underlying the observed changes in platelet count and MPV was not directly assessed. Although consistent with reports that leukocyte fragmentation during mechanical circulatory support may generate platelet-sized particles evidenced by regular/imaging flow cytometry that are counted as platelets by hematology analyzers,[19] confirmatory analyses were not performed, thus interpret with caution. Despite the modest sample size yielding statistically significant differences for several endpoints, larger studies would improve precision and possibly detect more subtle effects.

Acknowledgments

This study was funded by Scandinavian Real Heart AB. The authors acknowledge Tatiana Khudur and the R&D department at Scandinavian Real Heart AB for their contribution to this study.

Conception and design of the work as well as the analysis and interpretation of the data – all authors. Drafting of the paper, revising it critically for intellectual content – Per Sandgren and Ina Laura Perkins. All authors have approved of the final version to be published and agree to be accountable for all aspects of the work.

Author contributions

CRediT: **Per Sandgren:** Conceptualization, Data curation, Formal analysis, Investigation, Methodology, Writing – original draft, Writing – review & editing; **Shaikh Faisal Zaman:** Data curation, Formal analysis, Writing – review & editing; **Ina Laura Perkins:** Conceptualization, Formal analysis, Funding acquisition, Supervision, Writing – original draft, Writing – review & editing.

Disclosure statement

P Sandgren, SF Zaman and IL Perkins are employees and/or shareholders of Scandinavian Real Heart AB.

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Data availability statement

Data can be shared upon request.

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