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Review Article

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Endothelial dysfunction and the glycocalyx in major surgery: Emerging mechanisms of capillary leak, microcirculatory failure, and organ dysfunction

Scott Reynolds^{1*}

¹Professor, Neurosurgery, Pacific Neuroscience University, Seattle, USA.

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Abstract

Post-surgical Systemic Vascular Stress Response (SVVR) is more than a BP, CO or circulating volume response. Breakdown of the endothelial glycocalyx at the blood-vessel interface can result in endothelial activation, elevated permeability, adhesion of leukocytes, coagulation disturbance, and development of heterogeneous microvascular flow – all transforming inflammatory, haemodynamic, oxidative and ischaemia-reperfusion signals. This is a narrative review of the evidence for glycocalyx injury in major surgical contexts, looking at the development of capillary leak, tissue oedema, microcirculatory failure and postoperative organ dysfunction. Special focus is placed on haemorrhage, cardiopulmonary bypass, inflammatory activation, ischaemia-reperfusion, transfusion, fluid loading and vasopressor exposure. Preclinical investigations show that biomarkers, such as syndecan-1, heparan sulfate and hyaluronan are all elevated during the peri-operative period and are associated with changes in sublingual microvascular perfusion. Elevation of the biomarkers is not, however, equivalent to actual loss of the glycocalyx at the tissue level, and a bedside gold standard for assessment of the glycocalyx integrity does not exist. Current evidence thus favors a vascular-protective approach to the periop period, instead of a one-and-done glycocalyx-repair approach. Future studies should include endothelial markers, direct microcirculatory imaging, haemodynamic phenotyping, and organ-pathology outcome measurements.

Keywords: Endothelial dysfunction, Glycocalyx injury, Capillary leak, Organ pathology

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1. Introduction

Major surgery shouldn't only be interpreted as a macrocirculatory challenge. Tissue perfusion relies on the need for a functional microvascular network, for blood to be able to flow through, for the exchange of oxygen

* Corresponding author: Scott Reynolds, Professor, Neurosurgery, Pacific Neuroscience University, Seattle, USA.

and solutes across an intact endothelial barrier and for appropriate interstitial fluid balance. The endothelial glycocalyx, the carbohydrate-rich coating on the luminal surface of vascular endothelial cells, is of key interest in these functions, as well as being a mechanosensor and a regulator of the interactions leucocytes and platelets have with the endothelial cell surface (Reitsma *et al.*, 2007; Curry and Adamson, 2012; Weinbaum *et al.*, 2007; Curry and Michel, 2000). This layer can be altered during times of major surgery through inflammatory signaling, oxidative stress, haemorrhage, ischaemia/reperfusion, cardiopulmonary bypass as well as fluid and vasopressor disturbances (Becker *et al.*, 2010; Johansson *et al.*, 2017; Chappell *et al.*, 2014; Annecke *et al.*, 2010).

More than a protective coating, the consequences of a degraded glycocalyx are: Loss of anti-adhesive and anticoagulant endothelial surfaces (ECS) function and increased permeability of endothelial surfaces (ECS) allows extravasation of plasma water and macromolecules, allowing the leukocytes and platelets to interact with endothelial surfaces (ECS). These processes may be happening while arterial pressure and/or cardiac output remain seemingly adequate, but do not necessarily reflect tissue level parameters and, therefore, a supposedly acceptable level of tissue-level perfusion (Ince, 2015; De Backer *et al.*, 2010; Donati *et al.*, 2013; De Backer *et al.*, 2013). This idea has been reinforced by recent investigations in the peri-operative period which have shown an increase in syndecan-1 and other markers of endothelial injury following major abdominal, cardiac, hepatic, pancreatic and thoracic surgery, sometimes preceding measurable microcirculatory damage (Microvasc Res, 2023; Trajectory of plasma syndecan-1 and heparan sulphate during major surgery: Retrospective observational study, 2022/2023; Microvasc Res, 2019; Postoperative microcirculatory perfusion and endothelial glycocalyx shedding following cardiac surgery with cardiopulmonary bypass, 2019; Endothelial glycocalyx shedding and hemodynamic variables during hepatic and pancreatic resection surgery, 2025; Mass intraoperative endothelial glycocalyx shedding affects postoperative systemic inflammation response, 2024).

2. The endothelial glycocalyx: Structure, function, and the vascular barrier

The endothelial glycocalyx consists mainly of membrane bound proteoglycans, glycosaminoglycans, glycoproteins, glycolipids and plasma proteins related to them. Syndecans and glypicans anchor to the membrane, and heparan sulfate, hyaluronan and chondroitin sulfate help to build up the hydrated matrix extending into the vascular lumen. It is not a static structure, but continually changes depending on circulating proteins and soluble components that interact with it, and which affect the endothelial surface continually as it changes in accordance with the flow, inflammation, and metabolic state.

From a physiological point of view the glycocalyx is a component of the endothelial permeability barrier, prevents unwanted adhesion of cells, plays a role in shear stress sensing and coagulation, and is involved in inflammatory signaling (Korean J Anesthesiol, 2018). The layer is also in contact with the cytoskeleton and junctional complexes of the endothelia. As a result glycocalyx damage can exacerbate endothelial dysfunction without additional intercellular junctions being initially disrupted. This is significant because the changes to

Table 1: Major perioperative insults, mechanisms of glycocalyx injury, and vascular consequences

Perioperative insult	Principal mechanism	Vascular consequence
Surgical trauma/inflammation	Cytokines, proteases, endothelial activation	Shedding and permeability increase
Haemorrhage/hypoperfusion	Altered shear, catecholamines, oxidative stress	Barrier disruption and abnormal microvascular flow
Ischaemia-reperfusion	Reactive oxygen species and inflammatory activation	Glycocalyx degradation and endothelial leak
Cardiopulmonary bypass	Contact activation, inflammation, altered shear	Syndecan-1/heparan sulfate release and microcirculatory impairment
Fluid overload	Mechanical/haemodynamic stress	Interstitial oedema and barrier stress
Vasopressors	Intense vasoconstriction and flow redistribution	Potential macro-microcirculatory mismatch

the endothelium that happen perioperatively occur in a spectrum of stages from reversible surface perturbation to shedding to endothelial activation to disruption of the endothelial junctions to progressive and persistent microvascular dysfunction.

Another effect is on the relationship between the intravascular volume and the interstitial compartment, through the loss of integrity of the glycocalyx. The endothelial surface layer (ESL) is involved in fluid and protein barrier regulation in the intact barrier. Following degradation there will be increased permeability and altered oncotic handling which can promote extravascular fluid accumulation. This offers a possible link between risky peri- and postoperative fluid management and postoperative tissue oedema, especially in the case of inflammatory endothelial cell activation ([Perioperative fluid therapy for anaesthetists and intensivists, 2024](#); [Myburgh and Mythen, 2013](#)).

3. Why major surgery damages the endothelium and glycocalyx

The peri-operative setting represents a mélange of several known factors that are harmful to the endothelium. Trauma to tissues elevates levels of cytokines and activates innate immune responses, while blood loss causes shear/loss of perfusion, anaesthetic status affects vascular tone, periods of hypoperfusion followed by reperfusion cause an additional inflammatory and mechanical stimulus and can be an added inflammatory and mechanical stimulus during cardiopulmonary bypass. All these insults act through endothelial signalling pathways of reactive oxygen species, proteolytic enzymes, inflammatory mediators, and cytoskeletal remodelling pathways ([Ostrowski and Johansson, 2012](#); [Nieuwdorp et al., 2006](#)). Enzymatic degradation of glycocalyx components can be triggered by inflammatory mediators which activate endothelial cells. Meanwhile there can be damage to the structure of the endothelial membrane and glycocalyx by oxidative stress. This induces the syndecan-1, heparan sulfate, hyaluronan, and other fragments to get released into plasma and should therefore be interpreted as an indirect quantitative measurement of the remaining glycocalyx at each vascular bed rather than a quantitative measurement ([Uchimido et al., 2019](#); [Multimodal measurement of glycocalyx degradation during coronary artery bypass grafting, 2022](#)). Let's not forget haemodynamic management. Both too little and too much fluid can decrease tissue perfusion, thereby increasing the stress on the endothelial surface, and excessive fluid administration can increase the hydrostatic and interstitial pressure and result in oedema. Likewise, vasopressors can also be needed to restore perfusion pressure; their use may cause redistribution of microvascular blood flow, which does not reflect the arterial pressure ([Brandstrup et al., 2012](#); [Ospina-Tascón et al., 2022](#)). Too little or too much fluid or too little or too much vasopressor therapy is therefore not the clinically relevant question anymore, but how to maintain coherence between the macro and micro circulation.

4. From glycocalyx shedding to capillary leak and microcirculatory failure

After injury of glycocalyx, there is a sequence of mechanism connecting endothelial surface damage with the capillary leak. Altered barrier function and exposure of adhesion molecules and the endothelial cell membrane to circulating cells and plasma proteins, resulting from loss of the negatively charged, hydrated surface layer, affect barrier function. The activated endothelium, then, can facilitate cascade events of leukocyte and platelet adhesion, enzymatic activation of the coagulation apparatus as well as additional inflammatory events. This creates a self-reinforcing vascular phenotype, in which permeability and inflammation can work together, to affect flow.

Microcirculatory dysfunction is of special significance since the capillary perfusion is in space heterogeneous. These may be well perfused capillaries or capillary stagnant intermittently or persistently. This is due to the heterogeneity as it leads to a longer diffusion pathway and tissue hypoxia without an obvious global oxygen supply deficit ([Ince et al., 2018](#)). Therefore, it should not be inferred that a mean arterial pressure that is, or appears to be, normal is associated with normal tissue perfusion.

A good clinical example is cardiac surgery. In the course of cardiopulmonary bypass the expression of syndecan-1 increased and the perfused vessel density and the related sublingual microcirculatory indices decreased. During major abdominal surgery postoperative increases in syndecan-1 and other markers of endothelial injury were also found to be related to inflammatory activation and postoperative morbidity. These observations suggest a potential link between the release of glycocalyx and endothelial dysfunction in

the micro circulation and reinforced the idea that glycocalyx shedding and microcirculatory dysfunction are part of a perioperative endotheliopathy, though there is still some uncertainty regarding causality and direction.

5. Endothelial dysfunction and postoperative organ injury

Capillary leak has clinical relevance as interstitial oedema may disturb oxygen diffusion, interfere with the microcirculation and affect organ-specific architecture. The lung, in particular is also very susceptible: disruption of endothelial and epithelial barrier can lead to an increase in extravascular lung water and lead to postoperative respiratory dysfunction. Renal injury also can result from endothelial dysfunction, renal microcirculatory changes, inflammation and tubular stress alone or in combination, and not from systemic hypotension alone ([Zarbock *et al.*, 2014](#); [Poston and Koyner, 2019](#); [Cannesson *et al.*, 2011](#); [Dane *et al.*, 2015](#)).

Here the kidney is a good example of how endothelial injury can have organ specific consequences. The glomerular filtration barrier is dependent on a specialised endothelial glycocalyx and glomerular microvascular function can be impaired by perturbation of the systemic endothelial glycocalyx ([Endothelial glycocalyx layer shedding following lung resection, 2016](#)). Postoperative perfusion is also dependent on the local microvascular anatomic organization and the endothelial responses in the liver, intestine, the heart and in the brain. So, systemic changes of biomarkers need not necessarily be equated with uniform vascular bed injury.

Importantly, there are likely to be bidirectional links between injury to the glycocalyx and organ dysfunction. Although surgery may compromise the endothelial barrier, organ injury can aggravate the systemic inflammation, oxidative stress and endothelial activation. This feedback loop can at least partly explain that early vascular injury can occur and subsequently go on to result in ‘sustained postoperative endotheliopathy’ within high-risk patients.

6. Clinical evidence across major surgical settings

Based on current patient surgical cohorts, there is a growing body of evidence that supports glycocalyx degradation in the peri-operative setting. Recently, syndecan-1 and heparan sulfate was serially evaluated during major hepatic and pancreatic surgery to monitor the shedding of the glycocalyx. Results were findings of measurable postoperative changes and the reality that it was not a simple relationship with low-central-venous-pressure management, meaning it was challenging to distinguish whether it was a result of fluid-restriction, of vasopressors or of a combination of surgical manipulation or ischaemia-reperfusion.

In a large study of 112 patients undergoing major abdominal surgery, the levels of interleukin-6, syndecan-1, soluble VEGFR1 and thrombomodulin were all increased after surgery, with a correlation between richer endothelial-damage and inflammatory cytokines and higher postoperative morbidity. A further study was carried out on 72 patients undergoing major surgery, which revealed individual biomarker (syndecan-1 and heparan sulfate) temporal trends with blood loss, surgical type, and surgical duration. Taken together, these findings counsel against thinking of a universal response of the endothelia to the surgical event.

Perhaps some of the best evidence linking biochemical glycocalyx injury with functional microcirculatory change is seen in cardiac surgery. These glycocalyx associated biomarkers are elevated during cardiopulmonary bypass and continue to be correlated with decreased perfused-vessel measures. In a similar fashion, a prospective study of off-pump coronary surgery revealed increasing levels of syndecan-1 and IL-6 which correlated with both markers of SIR and severity of surgery post operatively.

Glycocalyx perturbation likewise occurs after thoracic surgery! One pilot study after lung resection found postoperative increases of syndecan-1 level—but there were only 5 patients and no conclusions could be drawn on relationships with the level of oxygenation ([Chappell *et al.*, 2008](#)). Collectively these surgical studies provide a good biological plausibility and association to the concept of glycocalyx injury, yet there is a significant lack of evidence that selectively preventing glycocalyx shedding will impact on hard clinical outcomes.

7. Can perioperative management preserve the glycocalyx?

Perioperative management should focus on minimizing any avoidable endothelial stress at present, as a proven-electrocardiograph pharmacologic glycocalyx-repair therapy is unavailable. Unconfirmed by clinical

Table 2: Clinical evidence linking surgery with endothelial/glycocalyx injury and microcirculatory dysfunction			
Setting	Population	Main finding	Interpretation
Major abdominal surgery	112 patients	IL-6, syndecan-1, sVEGFR1 and sTM increased	Endothelial injury accompanies inflammation
Major abdominal surgery	72 patients	Distinct syndecan-1 trajectories; HS increased in a subgroup	Response varies with operative factors
Cardiac surgery with CPB	17 patients	Perfused-vessel measures decreased while shedding markers increased	Biochemical injury parallels microvascular dysfunction
Hepatic/pancreatic resection	36 patients	Syndecan-1 and HS changed perioperatively	Haemodynamic strategy may modify endothelial cost
Off-pump coronary surgery	50 patients	Syndecan-1 and IL-6 rose and related to severity measures	Glycocalyx injury may amplify inflammation
Lung resection	16 patients	Postoperative syndecan-1 increased	Thoracic surgery can induce shedding

evidence, suggested glycocalyx-directed treatment options are the protective effects of corticosteroid signalling, antioxidant pathways, albumin, careful fluid management, but it has not been established whether there is universal value to these therapies.

This is especially important for the fluid therapy as hypovolaemia and fluid overloading could be harmful respectively via different mechanisms. Too much crystalloid can lead to extra fluids in the tissues and too little can lead to not enough fluid to the organs. Fixed fluid targets are thus likely not the most informative of parameters, and dynamic assessment of fluid responsiveness, perfusion endpoints, lactate trends, urine output, peripheral perfusion and, where available, microcirculatory measurements, may be more useful.

Vasopressor therapy should be tailored in a similar fashion. During vasodilatory hypotension, it is imperative that arterial pressure should be restored, although there is no guarantee of consistent improvements in capillary flow with excess vasoconstriction. The ultimate therapeutic goal will be to achieve haemodynamic coherence – adequate systemic pressure and adequate cardiac output, as well as maintenance of tissue level perfusion. Other aspects, such as temperature, transfusion or avoiding unnecessary hyperoxia, glycaemic control and minimising prolonged ischaemia may provide further possibilities of reducing endothelial stress, though evidence for glycaemic control is heterogeneous.

8. Biomarkers, current gaps, and future directions

Despite the most commonly used circulating marker of glycocalyx shedding being syndecan-1, it should not be interpreted as a single indicator of integrity of glycocalyx. Heparan sulfate and hyaluronan are complementary pieces of information and soluble thrombomodulin, angiopoietin-2, E-selectin and other markers of endothelial activation provide information on more general endothelial activation ([Reitsma *et al.*, 2007](#)). Functional data on the endothelial surface and microcirculation can be obtained by either a direct or semi-direct method and include sublingual microscopy and perfused boundary region measurements, both of which have methodologic and anatomical limitations.

As for a measurement of human glycocalyx, there are no gold standard universally accepted. Multimodal studies are thus important because circulating markers of disease, thickness estimates of the glycocalyx, endothelial activation markers and microvascular flow parameters all capture aspects of the same biological system. Assuming a raised biomarker is indicative of treatment responsive injury, future perioperative studies should incorporate these with clinically meaningful endpoints.

Patients phenotyping is next. A patient with massive blood loss, elevated inflammatory markers, long surgeries, and persisting microvascular changes may be a different endotheliopathy phenotype than a patient with a simple surgery and elevated inflammatory markers for a brief period of time. Future research is needed to determine if additional studies using this method of endothelial phenotyping at an early patient stage can help identify acute kidney injury, pulmonary complications, delirium, long-term intensive-care stay or any

other organ-specific outcomes, and if haemodynamic or pharmacological interventions targeting this phenotyping affect these outcomes or not.

Finally, intervention trials should differentiate between prevention of shedding of the glycocalyx and repair of damaged glycocalyx. Thus the combination in a single bundle of individualised fluid and vasopressor strategies and minimising ischaemia-reperfusion burden, optimising inflammatory control and real-time assessment of microvascular perfusion may be most promising. The paradigm change would transform peri-operative vascular protection from biomarkers to clinically testable treatments.

9. Conclusion

Major surgery can cause “dynamic endotheliopathy” characterized by glycocalyx degradation, inflammation, haemodynamic stress, capillary leak and microcirculatory dysfunction. This triggers an apparent phenomenon of “macro-microcirculatory uncoupling” which may constitute a possible mechanism for a tissue oedema, associated with impaired oxygen utilization and organ dysfunction shortly after surgery. There’s currently lots of evidence that the glycocalyx hurt is biologically relevant, but no single test nor glycocalyx-specific therapy has been explored and proven in the clinic to improve outcomes. Future perioperative management should therefore involve concerted haemodynamic management to maintain endothelial integrity, and a strategy involving biochemical and microcirculatory supervision in research and, if proved valid, in clinical decision making.

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