

## Research

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**The effects of continuous venovenous hemofiltration on coagulation activation**

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**Abstract**

**Introduction** The mechanism of coagulation activation during continuous venovenous hemofiltration (CVVH) has not yet been elucidated. Insight into the mechanism(s) of hemostatic activation within the extracorporeal circuit could result in a more rational approach to anticoagulation. The aim of the present study was to investigate whether CVVH using cellulose triacetate filters causes activation of the contact factor pathway or of the tissue factor pathway of coagulation. In contrast to previous studies, CVVH was performed without anticoagulation.

**Methods** Ten critically ill patients were studied prior to the start of CVVH and at 5, 15 and 30 minutes and 1, 2, 3 and 6 hours thereafter, for measurement of prothrombin fragment F1+2, soluble tissue factor, activated factor VII, tissue factor pathway inhibitor, kallikrein-C1-inhibitor and activated factor XII-C1-inhibitor complexes, tissue-type plasminogen activator, plasminogen activator inhibitor type I, plasmin-antiplasmin complexes, protein C and antithrombin.

**Results** During the study period the prothrombin fragment F1+2 levels increased significantly in four patients (defined as group A) and did not change in six patients (defined as group B). Group A also showed a rapid increase in transmembrane pressure, indicating clotting within the filter. At baseline, the activated partial thromboplastin time, the prothrombin time and

the kallikrein-C1-inhibitor complex and activated factor XII-C1-inhibitor complex levels were significantly higher in group B, whereas the platelet count was significantly lower in group B. For the other studied markers the differences between group A and group B at baseline were not statistically significant. During CVVH the difference in the time course between group A and group B was not statistically significant for the markers of the tissue factor system (soluble tissue factor, activated factor VII and tissue factor pathway inhibitor), for the markers of the contact system (kallikrein-C1-inhibitor and activated factor XII-C1-inhibitor complexes) and for the markers of the fibrinolytic system (plasmin-antiplasmin complexes, tissue-type plasminogen activator and plasminogen activator inhibitor type I).

**Conclusion** Early thrombin generation was detected in a minority of intensive care patients receiving CVVH without anticoagulation. Systemic concentrations of markers of the tissue factor system and of the contact system did not change during CVVH. To elucidate the mechanism of clot formation during CVVH we suggest that future studies are needed that investigate the activation of coagulation directly at the site of the filter. Early coagulation during CVVH may be related to lower baseline levels of markers of contact activation.

CRRT = continuous renal replacement therapy; CVVH = continuous venovenous hemofiltration; ELISA = enzyme-linked immunosorbent assay; F1+2 = prothrombin fragment F1+2; tFVIIa = activated factor VII; FXIIIa = activated factor XIII; IL = interleukin; PAI-1 = plasmin-antiplasmin; TFPPI = tissue factor pathway inhibitor; t-PA = tissue-type plasminogen activator.

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## **Table of Contents The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No**

1. Understanding the eBook The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - The Rise of Digital Reading The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Advantages of eBooks Over Traditional Books
2. Identifying The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Exploring Different Genres
  - Considering Fiction vs. Non-Fiction
  - Determining Your Reading Goals
3. Choosing the Right eBook Platform
  - Popular eBook Platforms
  - Features to Look for in an The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - User-Friendly Interface
4. Exploring eBook Recommendations from The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No

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**8. Staying Engaged with The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No**

◦ Joining Online Reading Communities

- Participating in Virtual Book Clubs
- Following Authors and Publishers The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
- 9. Balancing eBooks and Physical Books The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Benefits of a Digital Library
  - Creating a Diverse Reading Collection The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
- 10. Overcoming Reading Challenges
  - Dealing with Digital Eye Strain
  - Minimizing Distractions
  - Managing Screen Time
- 11. Cultivating a Reading Routine The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Setting Reading Goals The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Carving Out Dedicated Reading Time
- 12. Sourcing Reliable Information of The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Fact-Checking eBook Content of The Hemostatic System And Continuous Venovenous Hemofiltration Mutual Effects Acta Biomedica Lovaniensia No
  - Distinguishing Credible Sources
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  - Utilizing eBooks for Skill Development
  - Exploring Educational eBooks
- 14. Embracing eBook Trends
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