

# Development and Validation of a Hybrid AI Expert System for Coagulation Analysis: Integrating Rule-Based Logic with Generative AI for Intelligent Interpretation

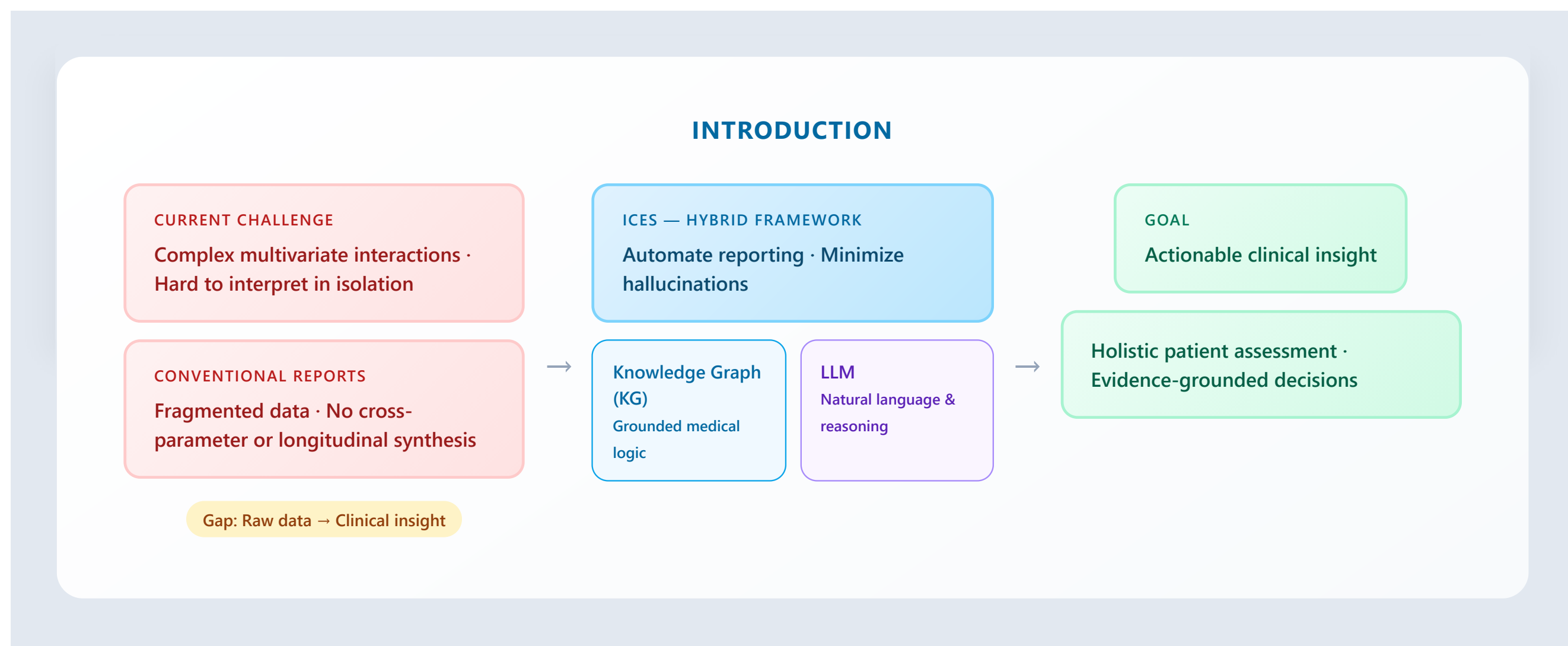
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## Introduction

Coagulation diagnostics play a crucial role in clinical decision-making but involve complex multivariate interactions that are challenging to interpret in isolation. Conventional laboratory reports often provide fragmented data without synthesizing cross-parameter relationships or longitudinal trends, thereby limiting their value for holistic patient assessment. To bridge the gap between raw data and actionable clinical insight, this study aims to develop an Intelligent Coagulation Expert System (ICES). We propose a hybrid framework that integrates a domain-specific Knowledge Graph (KG) with a Large Language Model (LLM) to automate clinical reporting and minimize AI hallucinations through grounded medical logic.

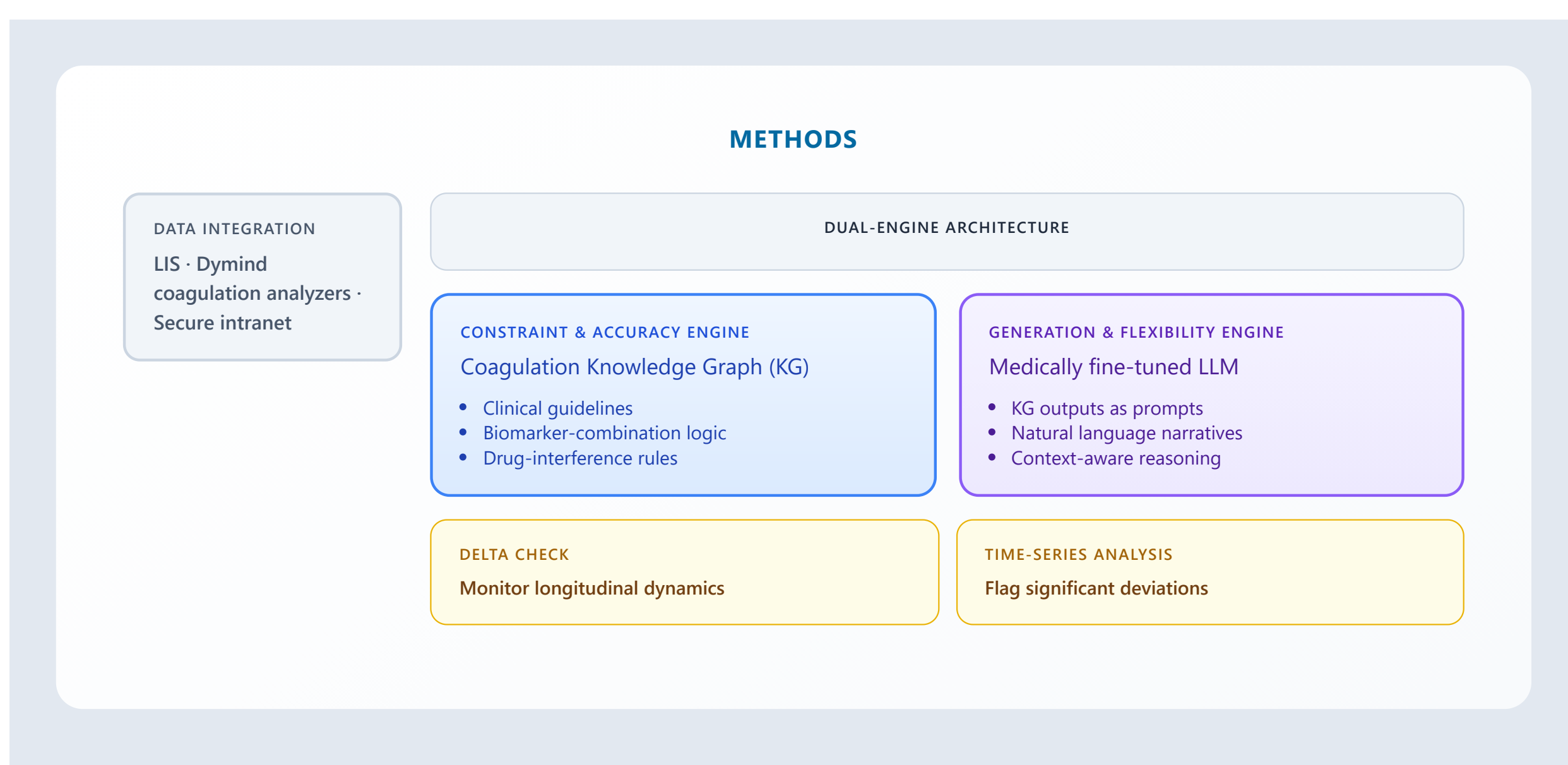


## Objectives

To develop an Intelligent Coagulation Expert System (ICES) that integrates a Knowledge Graph with an LLM, to automate clinical reporting and to reduce AI hallucination through evidence-grounded interpretation.

## Methods

The ICES was integrated with the Laboratory Information System (LIS) and Dymind automated coagulation analyzers via a secure intranet interface. It adopts a dual-engine hybrid architecture: (1) a Constraint & Accuracy Engine based on a standardized Coagulation Knowledge Graph encoding clinical guidelines, biomarker-combination logic, and drug-interference rules to ensure medical accuracy; and (2) a Generation & Flexibility Engine, where a medically fine-tuned LLM uses structured KG outputs to generate natural-language interpretations and perform context-aware reasoning. The system also incorporates delta-check algorithms and time-series analysis to monitor longitudinal changes and flag significant deviations.



## Results

Preliminary validation on 50 clinical scenarios (common coagulopathies & anticoagulant monitoring) showed ICES achieved 90% concordance with senior hematologists. The hybrid model reduced clinically relevant hallucinations by 13.5% vs standalone LLMs and outperformed template-based reports in readability, coherence, and contextualization. >80% of interpretations were anchored to verifiable knowledge graph nodes, ensuring evidence-grounded outputs.



## Representative Case

Parameter Name	Parameter Abbreviation	Result	Unit	Reference Range
Prothrombin Time	PT	15.8	s	11-14
Activated Partial Thromboplastin Time	APTT	35.2	s	28-43
Fibrinogen	FIB	1.9	g/L	2-4
Thrombin Time	TT	20.5	s	13.5-20
D-Dimer	D-Dimer	8.2	mg/L	0-0.5
Fibrinogen Degradation Products	FDP	12.4	µg/mL	0-5
Antithrombin III Activity	AT-III	58.0	%	80-120
International Normalized Ratio	INR	1.3		0.8-1.2

Note Type: KG, LLM

Main abnormality: D-Dimer significantly elevated (severe). Fibrinogen decreased (moderate). AT-III reduced (moderate), suggesting consumptive coagulopathy with bleeding/thrombosis risk. Tests show D-Dimer declining but abnormality persists. Fibrinogen and AT-III have not recovered—immediate attention to AT-III and Fibrinogen required. Focus monitoring on Fibrinogen (prevent bleeding), AT-III (prevent thrombosis), INR (coagulation status assessment).

**I. Suspected Disease Analysis and Inference**

**1. Most Probable Disease List**

Disease Name	Inference Reason (Mechanism Basis)	Pathological Mechanism	Typical Clinical Manifestations/High-Risk Groups
Disseminated Intravascular Coagulation (DIC)	PT prolonged, INR elevated, FIB decreased, D-Dimer/FDP significantly elevated, AT-III reduced → consumption of coagulation factors + activation of fibrinolytic system → concurrent bleeding and thrombosis.	Extensive microthrombus formation → consumption of coagulation factors + activation of fibrinolytic system → concurrent bleeding and thrombosis.	Severe infection, trauma, malignancy, obstetric complications, etc.
Liver Disease-related Coagulopathy	PT prolonged, INR elevated, FIB decreased → impaired hepatic synthesis of coagulation factors and fibrinogen.	Hepatocellular injury → decreased synthesis of coagulation factors (e.g., II, VII, IX, X) and FIB.	Chronic hepatitis, cirrhosis, acute liver failure patients.
Antithrombin III (AT-III) Deficiency	AT-III activity significantly reduced (41%) → insufficient anticoagulant effect, prone to thrombosis.	AT-III cannot effectively inactivate thrombin and factor Xa, promoting coagulation cascade.	Hereditary AT-III deficiency or secondary to liver disease, DIC, etc.
Secondary Fibrinolysis Hyperactivity	FDP significantly elevated (12.4 µg/mL), D-Dimer significantly elevated → massive degradation of fibrinogen.	Excessive plasmin activation → accumulation of fibrin degradation products, may accompany consumptive coagulopathy from various infections or other stress states (e.g., DIC).	Surgical procedures, trauma, malignancy, infection, and other stress states.

**II. Next Steps**

**Further Examinations**

- Coagulation factor activity test (e.g., Factor II, VII, IX, X)**  
Rationale: Differentiate liver disease-related coagulopathy from DIC, clarify extent of factor consumption.
- Complete liver function panel (ALT, AST, Bilirubin, albumin)**  
Rationale: Assess whether hepatic synthetic function is impaired, aid in diagnosing liver disease-related coagulation abnormalities.
- Fibrinolysis function test (e.g., plasminogen, α<sub>2</sub>-antiplasmin)**  
Rationale: Clarify degree of fibrinolytic system activation and mechanism.

**Lifestyle Recommendations**

- Diets:** Avoid high-fat diets (reduce atherosclerosis risk); supplement vitamin K (e.g., leafy greens) to improve PT/INR abnormalities.
- Severities:** Limit prolonged sitting or bed rest; but monitor for bleeding tendency (e.g., gingival bleeding, skin ecchymosis).
- Contraindications:** Do not use anticoagulants (e.g., warfarin) or antiplatelet drugs (e.g., aspirin) without medical supervision; adjust under physician guidance.

**III. Summary**

**1. Main Abnormal Mechanisms and Risks:**

- Significant hyperfibrinolysis (D-Dimer/FDP elevated) + consumption of coagulation factors (FIB decreased, PT prolonged) + anticoagulant dysfunction (AT-III decreased), highly suggestive of Disseminated Intravascular Coagulation (DIC) or severe liver disease-related coagulopathy.
- Concurrent bleeding tendency (low FIB, prolonged PT) and thrombosis risk (low AT-III, hyperfibrinolysis) may accompany microthrombi—close monitoring required.

**2. Urgent Concerns:**

- Significantly reduced AT-III activity (41%) may rapidly progress to life-threatening thrombotic events; prioritize ruling out DIC or hereditary AT-III deficiency.

## Conclusion

The proposed expert system effectively reconciles the determinism of knowledge-based logic with the generative flexibility of AI. It offers a robust and interpretable solution for coagulation reporting, enhancing both diagnostic efficiency and quality management. Future work will involve multi-center validation to further quantify its impact on clinical outcomes.

## Reference

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