Detroit Red Wings Alumni Association vs UCP Pucksters

Saturday, March 21, 2015  Viking Arena, Hazel Park, MI
Silent Auction begins at 3:30pm  Sled Hockey 4pm  Alumni Game 6pm

$10444.36
This is Blood
Welcome to Detroit

Photo credit: Dr Steven Moore, MD as a brand-spanking new intern (June 30, 2012)
A 56-year-old alcoholic with MELD score of 26 presents to your urgent care with distended abdomen and a fluid wave. He is uncomfortable appearing and short of breath. He states he gets a therapeutic paracentesis every 10 days but missed his appointment last week because he was out of town. Except a respiratory rate of 24, his vitals are stable and he is otherwise in his normal state of health. Before performing this procedure, you check labs and learn his INR is 4.3. This means he is:

• Hypocoagulable. (Of course he’ll bleed.)
• Hypercoagulable. (Nah, he’ll be fine.)
• Hemostatic. (Let’s call it “eucoagulable”.)
• It depends. (Stop asking me IM questions)
A 37-year-old with cirrhosis secondary to chronic hepatitis C presents to the emergency department after a car accident in which she was a restrained back seat passenger. She has a positive FAST exam and CT reveals a splenic laceration. Her **INR is 4.3**. The surgery team is prepping for the OR. You decide to share your opinion and state the patient is:

- Hypocoagulable. (It’s gonna be a bloodbath.)
- Hypercoagulable. (It’s only a flesh wound.)
- Hemostatic. (“Eucoagulable” sounds made up.)
- It depends. (Seriously. Knock it off.)
A 64 year old with end stage liver disease secondary to NAFLD presents to the emergency department with temp 39.3 degC, HR 106 bpm, BP 84/46, and RR 32. He has a tender and distended abdomen with a fluid wave – you suspect SBP. You initiate EGD and are in the process of placing a central line. Your med student helpfully informs you the patient’s **INR is 4.3** just as you notice bright red pulsatile blood in the syringe. You tell the med student this INR value means the patient is:

- Hypocoagulable. (Starting to second guess...)
- Hypercoagulable. (Am I using INR correctly?)
- Hemostatic. (Here’s hoping!)
- It depends. (...oh god, please don’t be arterial...)


My Cirrhotic Patient’s INR is Eleveneen!

Michael Harrison, MD PhD
PG-Y3 (EM/IM/CCM)
December 4, 2014
1630
Objectives

• Was the med school clotting cascade sufficient?
• How do we currently measure coagulopathy?
• What happens when the liver doesn’t work?
• What happens to our liver patients?
• How should we measure coagulopathy in our liver patients?
Table 1. Contributors to the Perception of a Bleeding Tendency in Patients with Acute and Chronic Liver Disease

<table>
<thead>
<tr>
<th>Contributor</th>
<th>Cirrhosis</th>
<th>Acute liver failure</th>
</tr>
</thead>
<tbody>
<tr>
<td>Elevated international normalized ratio</td>
<td>Mild-moderate</td>
<td>Moderate-severe</td>
</tr>
<tr>
<td>Thrombocytopenia</td>
<td>Moderate-severe</td>
<td>Mild-moderate</td>
</tr>
<tr>
<td>Portal hypertension</td>
<td>Moderate-severe</td>
<td>None-mild</td>
</tr>
</tbody>
</table>

Stravitz, 2012
“An old dogma is being dispelled in favor of the newly emerging concept that blood coagulation in such patients is rebalanced, owing to the parallel reduction of procoagulant and anti-coagulant factors.”

- Hemorrhagic events after liver biopsy?
- Hemarthrosis is liver patients?
- Portal vein thrombosis and Budd-Chiari Syndrome
Clinics in Liver Disease
Vol 13(1):1-166

- The coagulation cascade in liver cirrhosis
- The platelet and platelet function testing in liver disease
- Hyperfibrinolysis in liver disease
- Superimposed coagulopathic conditions in cirrhosis: infection and endogenous heparinoids, renal failure, and endothelial dysfunction
- Heparin-like effect in liver disease and liver transplantation
- Tests of coagulation in liver disease and liver transplantation
- **Tests of coagulopathy in liver disease**
  - The international normalized ratio of prothrombin time in the model for end-stage liver disease score: a reliable measure
  - International normalized ratio of prothrombin time in the model for end-stage liver disease: an unreliable measure
  - Blood products, volume control, and renal support in the coagulopathy of liver disease
  - The role of anti-fibrinolytics, rFVIIa and other pro-coagulants: prophylactic versus rescue
- **Coagulopathy of acute liver failure**
- Hypercoagulation in liver disease
- Parenchymal extinction: coagulation and hepatic fibrogenesis
- Portal vein thrombosis and Budd-Chiari Syndrome
- Bleeding in liver surgery: prevention and treatment
- Coagulation disorders and bleeding in liver disease: future directions
Nuts and Bolts of INR

• International Normalized Ratio of prothrombin time

\[ \text{INR} = \left( \frac{\text{PT}_{\text{test}}}{\text{PT}_{\text{normal}}} \right)^{\text{ISI}} \]

• Developed for standardization of therapeutic anticoagulation with Vitamin K antagonists
  – Reflection of PT ratio
  – Does not account for endothelial damage, Protein C level, platelet function, fibrinogen, plasminogen...
The Clotting Cascade

Contact activation (intrinsic) pathway
- Damaged surface
  - XII → XIIa → XI → IX → IXa → VIIIa → VIII → VIIa → VII

Tissue factor (extrinsic) pathway
- Trauma → Tissue factor
  - TFPI
  - VIIa → VII

Prothrombin (II)
- Xa
  - Thrombin (IIa)
  - Va

Active Protein C
- Protein S
- Protein C + thrombomodulin

Fibrinogen (I)
- Fibrin (Ia)
- XIIIa → XIII

Common pathway
- Cross-linked fibrin clot
What Maintains The Balance?

Procoagulant
• Factors I, II, III, IV, V, VI, VII, VIII, IX, X, XI, XII
• Platelets
• Fibrin

Anticoagulant
• Protein C
• Protein S
• Antithrombin
• TFPI
• Plasminogen
And If The Liver Doesn’t Work?

Procoagulant

• Factors I, II, III, IV, V, VI, VII, VIII, IX, X, XI, XII
• Platelets
• Fibrin
• Elevated antiphospholipid antibodies

Anticoagulant

• Protein C
• Protein S
• Antithrombin
• TFPI
• Plasminogen
• Thrombocytopenia
"...rebalanced state...some patients with advanced liver disease have a loss of balance towards the clotting side...and conversely, some patients are clearly more prone to bleeding. These tendencies are not static and may change with the clinical status of the patient, perhaps over hours...The clinical challenge is determining which patients fall into which group before an event (bleeding or clotting) occurs."
Q: What if I’m an EM doctor and I’m poking the patient with something sharp?

Dogma: Watch where you’re pointing that thing.
Central venous cannulation in patients with liver disease and coagulopathy – a prospective audit

- **1°**: Incidence of vascular complications following CVC placement in liver patients with INR >1.5 and/or platelets <150 000
  - MAJOR: any hemodynamically significant hemorrhage
  - MINOR: hematoma, superficial oozing
- Internal jugular (n=352) or subclavian (n=306)
  - No ultrasound guidance
Central venous cannulation in patients with liver disease and coagulopathy – a prospective audit

Table 1  INR and platelet values in subclavian and internal jugular cannulations (IQ interquartile)

<table>
<thead>
<tr>
<th></th>
<th>Median</th>
<th>IQ range</th>
<th>Range</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Subclavian</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INR</td>
<td>2.4</td>
<td>1.7–3.9</td>
<td>1–16</td>
</tr>
<tr>
<td>Platelets</td>
<td>81</td>
<td>51–133</td>
<td>9–1088</td>
</tr>
<tr>
<td><strong>Internal jugular</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>INR</td>
<td>2.7(^a)</td>
<td>1.8–4.7</td>
<td>1–17</td>
</tr>
<tr>
<td>Platelets</td>
<td>83</td>
<td>53–133</td>
<td>10–425</td>
</tr>
</tbody>
</table>

\(^a\) INR values in SC and IJ groups compared; \(p < 0.05\), Mann-Whitney U test
• Only 1 significant event (INR 1.5)
  – Subclavian artery puncture
    • Hemothorax that caused “respiratory embarrassment”; treated with chest tube
“...in patients with liver failure, the presence of raised INR or PT ratio should not be considered an absolute contra-indication to CV cannulation...it is prudent for cannulations in patients with coagulopathy to be carried out by experienced practitioners only.”
Q: What if I’m an IM doctor and my milestones include VTE prophylaxis?

Dogma: They’re already anticoagulated. They don’t need not heparin.
• 1°: Development of symptomatic DVT or PE confirmed with imaging
• 2°: Length of stay and in-hospital mortality
• 193 patients divided into quartiles by INR
  – <1.4
  – between 1.4 – 1.69
  – between 1.7 and 2.19
  – >2.2

Unfortunately, included patients with malignancy (n=44), prior history of VTE (n=5), and exogenous estrogen (n=1)
Coagulopathy Does Not Protect Against Venous Thromboembolism in Hospitalized Patients With Chronic Liver Disease

Ousama Dabbagh, MD, MSPH, FCCP; Aabha Oza; Sumi Prakash, MD; Ramez Sunna, MD; and Timothy M. Saettele, MD

• Incidence of VTE: 6.3%

Table 3—Primary and Secondary Outcomes

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>First Quartile (INR &lt; 1.4)</th>
<th>Second Quartile (1.4 ≤ INR &lt; 1.7)</th>
<th>Third Quartile (1.7 ≤ INR &lt; 2.2)</th>
<th>Fourth Quartile (INR ≥ 2.2)</th>
<th>All (N = 190)</th>
<th>P Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>In-hospital VTE</td>
<td>3 (6)</td>
<td>3 (5)</td>
<td>4 (11)</td>
<td>2 (5)</td>
<td>12 (6.3)</td>
<td>.665</td>
</tr>
<tr>
<td>Hospital mortality</td>
<td>2 (4)</td>
<td>2 (3)</td>
<td>5 (13)</td>
<td>14 (32)</td>
<td>23 (12.1)</td>
<td>&lt;.001</td>
</tr>
<tr>
<td>Hospital LOS, d (IQR)</td>
<td>3 (5)</td>
<td>4 (6)</td>
<td>4 (10)</td>
<td>5 (8)</td>
<td>4 (6)</td>
<td>.221</td>
</tr>
<tr>
<td>Diagnostic testing</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>VD-US</td>
<td>10 (21.3)</td>
<td>19 (31.1)</td>
<td>12 (31.6)</td>
<td>19 (43.2)</td>
<td>60 (31.6)</td>
<td>.168</td>
</tr>
<tr>
<td>Spiral CT scan</td>
<td>10 (21.3)</td>
<td>16 (26.3)</td>
<td>11 (28.9)</td>
<td>9 (20.5)</td>
<td>46 (24.2)</td>
<td>.763</td>
</tr>
<tr>
<td>VQ scan</td>
<td>2 (4.3)</td>
<td>1 (1.6)</td>
<td>1 (2.6)</td>
<td>1 (2.3)</td>
<td>5 (2.6)</td>
<td>.864</td>
</tr>
<tr>
<td>DVT prophylaxis</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>None</td>
<td>33 (70)</td>
<td>46 (75)</td>
<td>29 (76)</td>
<td>34 (77)</td>
<td>142 (74.7)</td>
<td>.603</td>
</tr>
<tr>
<td>Pharmacologic</td>
<td>7 (15)</td>
<td>5 (8)</td>
<td>1 (3)</td>
<td>4 (9)</td>
<td>17 (9)</td>
<td>.603</td>
</tr>
<tr>
<td>Mechanical</td>
<td>7 (15)</td>
<td>10 (16)</td>
<td>8 (21)</td>
<td>6 (14)</td>
<td>31 (16.3)</td>
<td>.603</td>
</tr>
</tbody>
</table>

Data are presented as No. (%) unless otherwise indicated. VD-US = venous Doppler ultrasound. VQ = ventilation-perfusion. See Table 1 for expansion of other abbreviations.
• Retrospective; Small N (190); Included patients with malignancy (n=44), prior history of VTE (n=5), and exogenous estrogen (n=1)

“...An elevated INR in the setting of CLD does not appear to protect against the development of hospital-acquired VTE. The notion that ‘auto-anticoagulation’ protects against VTE is unfounded...”
Risk of Venous Thromboembolism in Patients With Liver Disease: A Nationwide Population-Based Case–Control Study

Kirstine Kobberøe Søgaard, MD1,2, Erzsébet Horváth-Puhó, MSc1, Henning Grønbæk, MD, PhD3, Peter Jepsen, MD1, Hendrik Vilstrup, MD, PhD, DMS3 and Henrik Toft Sørensen, MD, PhD, DMS1,2

METHODS: We conducted a nationwide Danish case–control study of incident cases of venous thromboembolism from 1980 to 2005 using population-based data from the National Registry of Patients, and from the Civil Registration System. We used conditional logistic regression to compute the relative risk of venous thromboembolism in patients with liver disease compared to population controls. We then excluded patients with known malignancy (diagnosed either before or up to 3 months after the venous thromboembolism) or fractures, trauma, surgery, or pregnancy within 90 days before the venous thromboembolism to estimate the risk associated with unprovoked venous thromboembolism.

RESULTS: A total of 99,444 patients with venous thromboembolism and 496,872 population controls were included in the study. Patients with liver disease had a clearly increased relative risk of venous thromboembolism, varying from 1.74 (95% CI, 1.54–1.95) for liver cirrhosis to 1.87 (95% CI, 1.73–2.03) for non-cirrhotic liver disease. The risks were higher for deep venous thrombosis compared with pulmonary embolism. In the analysis, restricted to 67,519 patients with unprovoked venous thromboembolism and 308,614 population controls, we found slightly higher relative risks: 2.06 (95% CI, 1.79–2.38) for liver cirrhosis and 2.10 (95% CI, 1.91–2.31) for non-cirrhotic liver disease.

CONCLUSIONS: Patients with liver disease have a substantially increased risk of venous thromboembolism.

Am J Gastroenterol 2009; 104:96–101; doi:10.1038/ajg.2008.34
So what test can I trust?!
Potential Applications of Thromboelastography in Patients with Acute and Chronic Liver Disease

R. Todd Stravitz, MD, FACP, FACG
Gastroenterology & Hepatology Volume 8, Issue 8 August 2012

- “...(TEG) records the assembly of a clot in whole blood and provides assessment of overall hemostasis, including contributions from plasmatic and cellular components...clot formation, ultimate clot strength, and the stability of the clot...”
• “...standard tests of coagulation, such as PT/INR or aPTT, assess only plasmatic events...these tests omit the contribution of the platelet scaffold...INR and aPTT may, therefore, provide an inadequate and misleading assessment of the bleeding risk of patients with liver disease.”
Table 2. Correlation of Individual Thromboelastography Parameters with Phases of Hemostasis and Standard Hemostatic Laboratory Tests

<table>
<thead>
<tr>
<th>Thromboelastography parameter</th>
<th>Correlations with physiologic phase of hemostasis</th>
<th>Correlations with standard hemostatic laboratory tests</th>
</tr>
</thead>
<tbody>
<tr>
<td>Reaction time (min)</td>
<td>Time between initiation of coagulation cascade to initial formation of fibrin</td>
<td>INR, aPTT, procoagulant factor levels</td>
</tr>
<tr>
<td>Kinetic time (min)</td>
<td>Time between initial formation of fibrin to specific clot firmness (20 mm)</td>
<td>Fibrinogen, platelet count</td>
</tr>
<tr>
<td>α-angle (degrees)</td>
<td>Rate of fibrin formation and crosslinking</td>
<td>Fibrinogen, platelet count</td>
</tr>
<tr>
<td>Maximum amplitude (mm)</td>
<td>Maximum clot strength</td>
<td>Fibrinogen, platelet count</td>
</tr>
<tr>
<td>Lysis-30 (%)</td>
<td>Fibrinolysis 30 minutes after maximum amplitude</td>
<td>Fibrin degradation products</td>
</tr>
</tbody>
</table>

Figure 1. A normal thromboelastogram displaying individual parameters. See the text for details of measurement intervals. k=kinetic time; Ly-30=clot lysis in 30 minutes; r=reaction time.

Stravitz, 2012
<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>1.3</td>
</tr>
<tr>
<td>Platelets (x 10⁹/L)</td>
<td>21</td>
</tr>
<tr>
<td>Fibrinogen (mg/dL)</td>
<td>174</td>
</tr>
<tr>
<td>Reaction time (min)</td>
<td>3.5</td>
</tr>
<tr>
<td>Kinetic time (min)</td>
<td>3.8</td>
</tr>
<tr>
<td>α-angle (degrees)</td>
<td>57.0</td>
</tr>
<tr>
<td>Maximum amplitude (mm)</td>
<td>36.8</td>
</tr>
<tr>
<td>Ly-30 (%)</td>
<td>0.6</td>
</tr>
</tbody>
</table>

<table>
<thead>
<tr>
<th>Laboratory test</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>INR</td>
<td>1.9</td>
</tr>
<tr>
<td>Platelets (x 10⁹/L)</td>
<td>8</td>
</tr>
<tr>
<td>Fibrinogen (mg/dL)</td>
<td>88</td>
</tr>
<tr>
<td>Reaction time (min)</td>
<td>4.8</td>
</tr>
<tr>
<td>Kinetic time (min)</td>
<td>NA</td>
</tr>
<tr>
<td>α-angle (degrees)</td>
<td>23.6</td>
</tr>
<tr>
<td>Maximum amplitude (mm)</td>
<td>16.3</td>
</tr>
<tr>
<td>Ly-30 (%)</td>
<td>0.0</td>
</tr>
</tbody>
</table>
Conclusions
- Not Guaranteed to Bleed
Hypercoagulability plays an under-recognized but important role in many aspects of acute and chronic liver disease...often concurrently coagulopathic, hypercoagulable, and hyperfibrinolytic...bleeding is the most common clinical manifestation...However, the lack of control of the enzymatic reactions of the clotting cascade may result in excessive thrombus formation.”
In certain circumstances, the risk of thrombotic events may be greater than the risk of hemorrhage.
Any Questions?

Hey, this is for S. Who I met at the Henry Ford Hospital ICU on Sunday nite. We was talking in the louge, my wife (who I told you I don't get along w/ but we stay 2gether for the new baby) is there after her car acident. You were there for ur grampa who something. You might remembrar that my name begins with D. And I was tall & you liked my tatts on my forearm. I made you laugh, and you will probably remember that I told U that you was Fly. I really liked talking to you, but that dam nurse kept interrupting and finally I couldn't come back because of the feedings tubes problam. When I came back I was like "Dam" bcause U were gone and all. So I am hoping U will see this here. Good news: my wife won't last to much longer, wich means I can b a free man soon, wich means I would like to take you out for a nite on the town if U see this. So get at me because I got what U need and I will treat you right. Not like all those wankstas U was talking about. Really do.

PS - U like Red Lobster?

- Location: Tha D
- it's NOT ok to contact this poster with services or other commercial interests

PostingID: 3116854294
References

- DeSancho & Pastores, 2007
A 56-year-old alcoholic with MELD score of 26 presents to your urgent care with distended abdomen and a fluid wave. He is uncomfortable appearing and short of breath. He states he gets a therapeutic paracentesis every 10 days but missed his appointment last week because he was out of town. **Except a respiratory rate of 24, his vitals are stable and he is otherwise in his normal state of health.** Before performing this procedure, you check labs and learn his **INR is 4.3.** This means he is:

- Hypocoagulable.
- Hypercoagulable.
- Hemostatic.
- It depends.
A 37-year-old with cirrhosis secondary to chronic hepatitis C presents to the emergency department after a car accident in which she was a restrained back seat passenger. She has a positive FAST exam and CT reveals a splenic laceration. Her INR is 4.3. The surgery team is prepping for the OR. You decide to share your opinion and state the patient is:

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A 64 year old with end stage liver disease secondary to NAFLD presents to the emergency department with **temp 39.3 degC, HR 106 bpm, BP 84/46, and RR 32.** He has a tender and distended abdomen with a fluid wave – **you suspect SBP.** You initiate EGD and are in the process of placing a central line. Your med student helpfully informs you the patient’s **INR is 4.3** just as you notice bright red pulsatile blood in the syringe. You tell the med student this INR value means the patient is:

- Hypocoagulable.
- Hypercoagulable.
- Hemostatic.
- It depends.
New concepts of coagulation and bleeding in liver disease

Patrick G. Northup · Stephen H. Caldwell

Abnormalities of hemostasis and bleeding in chronic liver disease: the paradigm is challenged

Armando Tripodi · Massimo Primignani · Pier Mannuccio Mannucci
<table>
<thead>
<tr>
<th>Haemostatic abnormality</th>
<th>Mechanism</th>
</tr>
</thead>
</table>
| Hypocoagulability       | Decreased synthesis of coagulation factors (except VIII and VWF)  
Hypofibrinogenaemia (endstage liver failure)  
Vitamin K deficiency (II, VII, IX, X)  
Decreased clearance of degraded coagulation factors |
| Hypercoagulability      | Decreased synthesis of natural anticoagulant proteins  
antithrombin (AT), proteins C, S and Z  
Decreased clearance of activated coagulation factors |
| Dysfibrinogenaemia      | Synthesis of abnormal fibrinogen |
| Hyperfibrinolysis       | Increased levels of circulating tPA activity due to impaired hepatic clearance  
Decreased synthesis of fibrinolytic inhibitors (PAI-1 and α2-antiplasmin)  
Decreased thrombin-activatable fibrinolytic inhibitor (TAFI) |
| Quantitative and qualitative platelet defects: | Decreased bone marrow production (due to decreased thrombopoietin)  
Splenic sequestration |
| Thrombocytopenia         | Immune-mediated platelet destruction |
| Thrombocytopathies       | Folate and vitamin B12 deficiencies  
Direct effect of ethanol  
Non-specific platelet aggregation abnormalities |
Minimal effects of acute liver injury/acute liver failure on hemostasis as assessed by thromboelastography

R. Todd Stravitz¹,*  Ton Lisman³, Velimir A. Luketic¹, Richard K. Sterling¹, Puneet Puri¹, Michael Fuchs¹, Ashraf Ibrahim², William M. Lee⁴, Arun J. Sanyal¹

- $H_0$: Patients with acute liver injury / failure would have normal hemostasis despite elevated INR
- 51 patients w/ acute liver injury and no prior history of liver disease
  - ALI: INR >1.5
  - ALF: ALI with hepatic encephalopathy
Fig. 1. Representative thromboelastogram from a patient with acute liver failure from acetaminophen. The tracing is normal in all of the 5 TEG parameters indicated despite an INR of 4.2: (R-time, 3.8 min; K-time, 1.1 min; α-angle, 73.8°; MA, 63.0 mm; lysis at 30 min, 0.3%). At the time of this TEG, the patient had grade 3 hepatic encephalopathy and venous ammonia of 120 μmol/L. Other coagulation components included: platelet count, $163 \times 10^9$/L; fibrinogen, 189 mg/dl; factor VII, 4% of normal; factor VIII, 558% of normal; protein C, 5% of normal.
Central venous cannulation in patients with liver disease and coagulopathy – a prospective audit
Minimal effects of acute liver injury/acute liver failure on hemostasis as assessed by thromboelastography

R. Todd Stravitz¹,*, Ton Lisman³, Velimir A. Luketic¹, Richard K. Sterling¹, Puneet Puri¹, Michael Fuchs¹, Ashraf Ibrahim², William M. Lee⁴, Arun J. Sanyal¹

Conclusions: Despite elevated INR, most patients with ALI/ALF maintain normal hemostasis by TEG, the mechanisms of which include an increase in clot strength with increasing severity of liver injury, increased factor VIII levels, and a commensurate decline in pro- and anticoagulant proteins.
Central venous cannulation in patients with liver disease and coagulopathy – a prospective audit

Brit-speak
• To whom do we attribute the procedure document?
  – Consultant
  – Registrar or senior registrar
  – Senior house officer
  – House officer

Ameri-speak
• Who dunnit? Write a note, would ya?
  – Attending physician with fellowship training
  – Fellow or senior fellow
  – Senior resident
  – Resident
Anti- “auto-coagulation”

Hypercoagulation and thrombophilia in liver disease


MECHANISMS OF DISEASE

The Coagulopathy of Chronic Liver Disease

Armando Tripodi, Ph.D., and Pier Mannuccio Mannucci, M.D.
[template]