



**ED Neurological
Emergencies Patients:
*Neuroresuscitation Update
for Ischemic Stroke &
Intracerebral Hemorrhage***

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Acute Care Medicine Conference**


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Disclosures

- FERNE Chairman and President
- No individual financial disclosures

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**Ischemic Stroke
Patient Care:
*tPA Use in 2007***

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Clinical Situation

- tPA has been approved for 10+ years
- There is still much discussion, if not outright controversy
- It is the standard of care
- When is it the standard of care?
- Why is it the standard of care?
- How should it be used in clinical EM practice?

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Clinical tPA Facts

- tPA has proven clinical efficacy based on paired phase III clinical trials

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Clinical tPA Facts

- tPA has proven clinical efficacy based on paired phase III clinical trials
- tPA has proven clinical effectiveness based on multiple phase IV reports of clinical use

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Clinical tPA Facts

- tPA has proven clinical efficacy based on paired phase III clinical trials
- tPA has proven clinical effectiveness based on multiple phase IV reports of clinical use
- tPA effectiveness is suggested by publications of meta-analysis data

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Clinical tPA Facts

- tPA has proven clinical efficacy based on paired phase III clinical trials
- tPA has proven clinical effectiveness based on multiple phase IV reports of clinical use
- tPA effectiveness is suggested by publications of meta-analysis data
- Reanalysis of the NINDS clinical trials confirms initial clinical efficacy report

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Clinical tPA Facts

- Emergency Medicine organizations suggest that there is likely clinical efficacy in selected patient populations

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Clinical tPA Facts

- Emergency Medicine organizations suggest that there is likely clinical efficacy in selected patient populations
- Legal input suggests that patients, in general, understand this therapy to be the standard of care that offers benefit

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Clinical tPA Facts

- Emergency Medicine organizations suggest that there is likely clinical efficacy in selected patient populations
- Legal input suggests that patients, in general, understand this therapy to be the standard of care that offers benefit
- Many institutions and EM physicians successfully use this therapy

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Clinical Questions

- Based on these facts, is there still concern about the use of tPA in selected patients?
- What is the basis for this concern?
- What more can be studied or taught regarding this stroke therapy?
- What could FERNE specifically do in order to improve your EM clinical practice for these patients?

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ED Stroke Patient Management:

What must we be able to do in order to provide tPA in the ED (mimickers, stroke scales, and CT interpretation)?

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Key Clinical Questions

- You are obliged to be able to give tPA...
- What diagnostic skills?
- What use of stroke scales?
- What CT interpretation skills?
- What IV tPA use skills?

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Diagnostic Skills

- Identify a stroke
- Start with the Cincinnati stroke scale
- Identify speech and language deficit
- Identify hemiparesis
- Identify CN deficits c/w stroke
- Consider mental status changes

FERNE/EMRA



Diagnostic Skills

- Exclude toxic/metabolic causes
- Exclude seizure syndromes
- Exclude TIAs
- Is the deficit significantly improving during the time that you are preparing to give IV tPA?



Stroke Scales Use

- Estimate the severity of the stroke
- Know what patients were treated in the NINDS clinical trials
- Be able to identify significant or moderate stroke
- Consider use in elderly pts with severe stroke (NIHSS > 20) and AFib



NIHSS: LOC

- LOC overall 0-3 pts
- LOC questions 0-2 pts
- LOC commands 0-2 pts

- LOC: 7 points total



NIHSS: Cranial Nerves

- Gaze palsy 0-2 pts
- Visual field deficit 0-3 pts
- Facial motor 0-3 pts

- Gaze/Vision/
Cranial nerves: 8 points total



NIHSS: Motor

- Each arm 0-4 pts
- Each leg 0-4 pts

- Motor: 8 points total
(8 right, 8 left)



NIHSS: Cerebellar

- Limb ataxia 0-2 pts

- Cerebellar: 2 points total



NIHSS: Sensory

- Pain, noxious stimuli 0-2 pts
- Sensory: 2 points total



NIHSS: Language

- Aphasia 0-3 pts
- Dysarthria 0-2 pts
- Language: 5 points total



NIHSS: Inattention

- Inattention 0-2 pts
- Inattention: 2 points total



NIHSS Composite

- CN (visual): 8
- Unilateral motor: 8
- LOC: 7
- Language: 5
- Ataxia: 2
- Sensory: 2
- Inattention: 2



Four Main NIHSS Areas

- CN/Visual: Facial palsy, gaze palsy, visual field deficit
- Unilateral motor: Hemiparesis
- LOC: Depressed LOC, poor responsiveness
- Language: Aphasia, dysarthria, neglect
- 28 total points



NIHSS ED Estimate

- CN (visual): 8
- Unilateral motor: 8
- LOC: 8
- Language/Neglect: 8
- Mild: 2, Moderate: 4, Severe: 8
- +/- Incorporates other elements



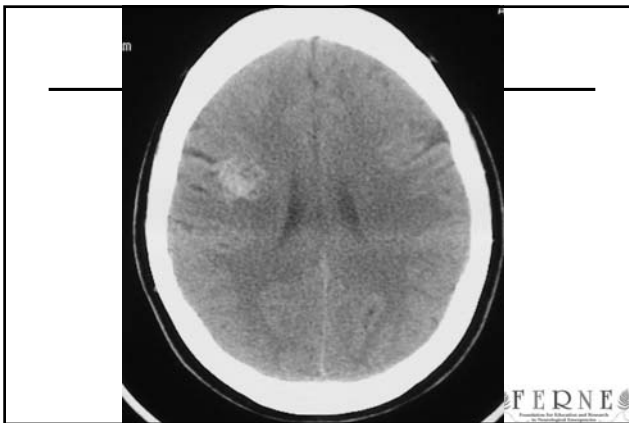
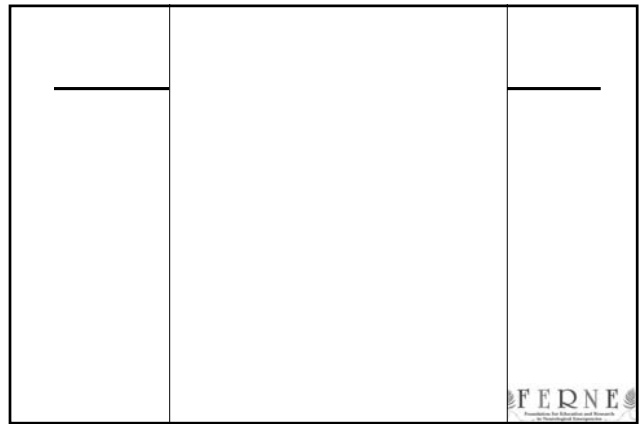
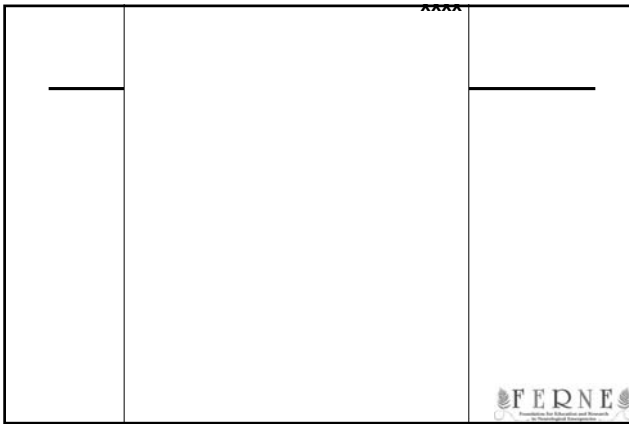
NIHSS Patient Estimate

- CN/Visual: R vision loss, no fixed gaze 4
 - Unilateral motor: hemiparesis 8
 - LOC: mild decreased LOC 2
 - Language: speech def, neglect 4
- Approx 18 points total
• Moderate to severe stroke range



CT Interpretation Skills

- No insular ribbon or MCA sign
- No detailed assessment
- Identify asymmetry and edema
- Identify blood, mass lesion
- Identify any area of hypodensity c/w a recent stroke of many hours duration that precludes IV tPA use



IV tPA Use Skills

- Identify indications, contraindications
- Quickly get the tests and consults
- Communicate with the neurologist
- Obtain consent with family and know what statistics are relevant
- Document the interaction
- Maintain BP below 185/110 range
- Follow the NINDS protocol closely

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ED tPA Documentation

- With tPA, there is a 30% greater chance of a good outcome at 3 months
- With tPA use, there is 10x greater risk of a symptomatic ICH (severe bleeding stroke)
- Mortality rates at 3 months are the same regardless of whether tPA is used
- What was the rationale, risk/benefit assessment for using or not using tPA?
- What was done to expedite Rx, consult neurology and radiology early on?

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Conclusions

- The IV tPA skill set is identified, limited, and manageable
- It is possible to provide quality emergency services with IV tPA
- Identify good patient candidates
- Make it happen quickly
- Document the ED management

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The Neurological Exam in ED Stroke Patients

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Motor Exam

- Is there hemiparesis & how severe?
- Motor: Upper & lower ext
 - Upper: Pronator drift, pull fingers out of hand
 - Lower: Leg lift, hip flexion push against hand

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Sensory Exam

- Is there a loss of light touch?
- Sensory: Light touch, pinprick graphesthesia

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Reflex Exam

- Are there pathologic reflexes?
- Is there a gag reflex?

- Normal vs. pathologic
 - Normal: Corneals, gag, DTRs
 - Pathologic: Babinski, Chaddock
 - Dec LOC, loss of airway control
 - Loss of UMN control

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Cerebellar Exam

- Is finger to nose, heel to shin OK?
- Can the patient sit in the cart?

- Extremity motor cerebellar function
- Truncal ataxia and ataxic gait
- Positive Romberg

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Visual/Neglect Exam

- Does the patient gaze to one side?
- Is there a loss of vision on one side?
- Does the patient neglect one side?

- Persistent gaze to side of ischemic CVA
- Homonymous hemianopsia
- Neglect of one side

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Language Exam

- Is the patient dysarthric?
- Does the patient have an aphasia?

- Dysarthria: Poor mouth motor function
- Aphasia: Disturbed language processing
 - Expressive: can't speak the right words
 - Receptive: can't process what is heard

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Mental Status Exam

- Is there an alteration in mental status?

- Level of consciousness (AVPU)
 - Alert
 - Responds to verbal
 - Responds to painful
 - Unresponsive
- Glasgow Coma Scale Score

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Case History

- 62 yo F with sudden onset paralysis, aphasia at 6:30 pm, no trauma
- No history of similar symptoms in past
- Patient apparently was normal prior
- No known risk factors (DM, HTN)
- No Hx surgery, bleed that would preclude tPA use

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Case Physical Exam

- **Vital signs:** Hypertension noted, pulse ox OK, POC glucose OK
- **HEENT:** Pupils midrange, reactive, no papilledema, airway OK
- **Neck:** No Bruits, no nuchal rigidity
- **Chest:** BSBE No Rales
- **Cardiac:** No afib, no gallops or murmurs

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Case Physical Exam (Con't)

- **Abd:** No evidence of AAA, peritonitis
- **Ext:** No DVT or pedal edema evident
- **Skin:** No cellulitis or wounds
- **Neuro:** Please see below

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Case Neuro Exam

- **CN:** R mouth droop, no lid weakness
- **Motor:** R hemiparesis, flaccid
- **Sensory:** No light touch of R extremities
- **Reflex:** No DTRs RLE, upgoing great toe R
Normal corneals, normal gag reflex

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Case Neuro Exam (Con't)

- **Cerebellar:** Slight truncal ataxia, to R
- **Visual/Neglect:** Lost vision & neglect, R
- **Language:** Dysarthria, expressive aphasia
No receptive aphasia
- **LOC:** Slightly somnolent, responds to verbal stimuli, GCS=13
- **Approximate NIHSS:** 18

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Clinical Case: CT Result



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Clinical Case: ED Rx

- **CT:** no low density areas or bleed
- **No contraindications to tPA, BP OK**
- **NIH stroke scale:** approx 18-20
- **Neurologist said OK to treat**
- **No family to defer tPA use**
- **tPA administered, no complications**

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tPA Use & Repeat Exam

- tPA dosing:
 - 8:21 pm, approx 1'45" after CVA sx onset
 - Initial bolus: 5 mg slow IVP over 2 minutes
 - Follow-up infusion: 40 mg infusion over 1 hour
- Repeat neuro exam at 90 minutes:
 - Repeat Exam: Increased speech & use of R arm, decreased mouth droop & visual neglect
 - Repeat NIH stroke scale: approximately 12-14

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ED tPA Documentation

- With tPA, there is a 30% greater chance of a good outcome at 3 months
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- Mortality rates at 3 months are the same regardless of whether tPA is used
- What was the rationale, risk/benefit assessment for using or not using tPA?
- What was done to expedite Rx and to consult neurology and radiology early on?

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ED tPA Documentation

- Patient was explained risks and benefits of tPA use and was able to understand and provide verbal consent (as able), and signature with L hand.
- Risk/benefit favored tPA given clear onset time, young patient with no significant morbidities or factors that would preclude tPA use, and approx NIHSS that suggests OK use.
- Rapid CT obtained, neurology aware of pt status, agreed with expedited tPA use, to follow.

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Hospital Course & Disposition

- Hospital Course: No hemorrhage, improved neurologic function
- Disposition: Rehabilitation hospital
- 3 Month Exam: Near complete use of RUE, speech & vision improved, slight residual gait deficit
- Able to live at home with assistance

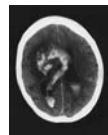
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Conclusions

- The neurological exam can be performed in a way that is easy, understandable to other clinicians
- A well conducted neurological exam allows for good decisions making, and imparts a sense that the work of the emergency physician is compelling
- This will enhance collaboration and satisfaction, enhancing EM practice

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Optimizing ED Ischemic Stroke Patient Care: *Horizons in 2007*



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Ischemic Stroke Pt Care

- Need to utilize tPA when applicable
- No more complicated therapeutic
- Risk of significant hemorrhage 50% that of imparting benefit
- New technologies exist
- Can these new diagnostics improve our ability to utilize this and other therapies?

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Ischemic Stroke Pathophysiology

- Cerebrovascular occlusion
- Core infarct: not salvageable
- Ischemic penumbra: salvageable
- Non-contrast CT cannot distinguish
- MRA and CTA may be able to

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Diagnostics in ED CVA Pts

- Core dead infarct
- Surrounding ischemic penumbra
- Non-contrast CT cannot distinguish these
- MRA and CTA may be able to distinguish
- Therapies based on whether or not there is something to salvage
- This enhances tPA risk/benefit profile

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Key Clinical Questions

- What do MRI and CTA/perfusion offer us when determining optimal ischemic stroke patient therapies?
- Which test will become our standard of care in the future? Why?

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CNS CT, MRI : The Tests

- CT with contrast
- CT angiography (CTA)
- MRI, without or with contrast
- MR angiography (MRA)
- Cerebral angiography

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MRI/MRA

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***Indications for MRI and CT
in Emergent CNS
Illness & Injury:
Beyond the Non-contrast CT***

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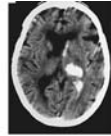
Large, Severe CVAs

- Patients with acute stroke
- Moderate severity
- NIHSS ranges from 10-20?
- Acute hemorrhage must be excluded
- Thrombolytic therapy a consideration
- Can pt selection be optimized?

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Non-Contrast Cranial CT

- Primary use is to rule out acute hemorrhage
 - Contraindication to the use of thrombolytic therapy
 - Identification of potential surgical candidates
- Limited sensitivity for detecting acute cerebral ischemia (31-75%)
- tPA therapy



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Acute Ischemic Stroke CT

- Dense MCA sign
- Decreased gray-white differentiation
 - Especially in the basal ganglia
- Loss of insular ribbon
- Effacement of sulci
- Edema and mass effect *
- Large area of hypodensity* (>1/3 MCA)

*May signify increased risk of hemorrhage with thrombolytic therapy

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Magnetic Resonance Imaging (MRI)

- Multimodal MRI
- Demonstrates hyperacute ischemia
- Considered less reliable in identifying early parenchymal hemorrhage

- What role does MRI play in diagnosis and management of the acute stroke pt?

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MRI: Stroke Center Approaches

- CT acutely with follow-up MRI
 - Late delineation of stroke findings
- Both CT and MRI acutely
 - More expensive, time-consuming
 - Possible enhancements in therapy?
- MRI acutely
 - Is it a reasonable alternative?

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What is Multimodal MRI?

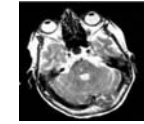
- T1, T2 Imaging: Conventional weighted pulse sequences
- DWI: Diffusion-Weighted Imaging
- PWI: Perfusion-Weighted Imaging
- GRE: Gradient Recalled Echo pulse sequence (T2*-sensitive)
- FLAIR: Fluid-Attenuated Inversion Recovery images

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T1 & T2 Weighted Pulse Sequences

- Sensitive for subacute and chronic blood
- Less sensitive for hyperacute parenchymal hemorrhage?
- Probably adequately sensitive for acute bleed

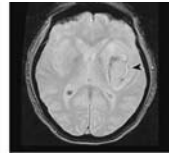


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Gradient Recalled Echo (GRE) Pulse Sequence

- May be sensitive for hyperacute parenchymal blood
- Detects paramagnetic effects of deoxyhemoglobin & methemoglobin as well as diamagnetic effects of oxyhgb

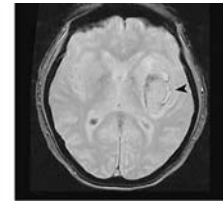


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Gradient Recalled Echo (GRE) Pulse Sequence

- Core of heterogeneous signal intensity reflecting recently extravasated blood with significant amounts of oxyhgb
- Hypodense rim reflecting blood that is fully deoxygenated



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Diffusion-Weighted Imaging

- Ischemia decreases the diffusion of water into neurons
- Extracellular water accumulates
- On DWI, a hyperintense signal
- Present within minutes
- Irreversible damage delineated
- Non-salvageable tissue?
- Infarct core

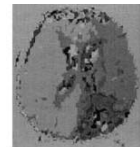


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Perfusion-Weighted Imaging

- Tracks a gadolinium bolus into brain parenchyma
- PWI detects areas of hypoperfusion
 - Infarct core (DWI area) and
 - Ischemic penumbra



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DWI/PWI Mismatch

- Subtract DWI signal (infarct core) from the PWI signal (infarct core and ischemic penumbra)
- DWI/PWI mismatch is the hypoperfused area that may still be viable (ischemic penumbra)

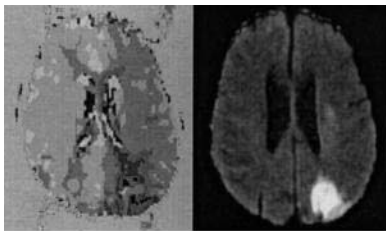
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DWI/PWI Mismatch

- Important clinical implications
- May identify the ischemic penumbra
- If there is a large mismatch, then reperfusion may be of benefit, even beyond the three hour tPA window
- If there is no mismatch, there may be little benefit to thrombolytic therapy, even within the three hour window

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DWI/PWI Mismatch



- PWI hypoperfused area
- DWI signal

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So what is the role of MRI in the ED evaluation of the stroke patient?

- Secondary?
 - Initial CT to rule out hemorrhage
 - Subsequent MRI to fully delineate ischemia, infarct and to follow treatment
- Primary?
 - Initial and possibly only imaging modality

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MRI in Large, Severe CVAs

- Primary MRI not current EM standard
- Logistical, timing issues exist
- MRI likely able to diagnose hemorrhage
- DWI/PWI mismatch a promising exam
- Tailored thrombolytic therapy??
- Improved patient outcome??

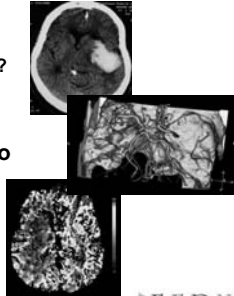
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CT Angiography & CT Perfusion

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CT Angiography and CT Perfusion

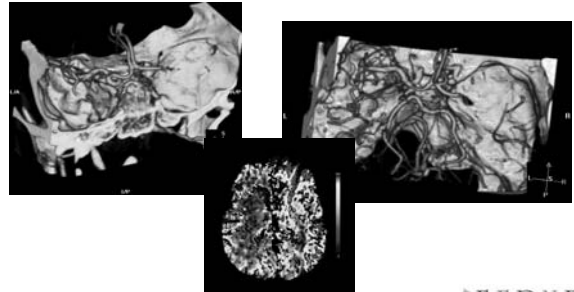
- **Essential questions**
 - Is there hemorrhage?
 - Is there large vessel occlusion?
 - Is there “irreversibly” infarcted core?
 - Is there “at risk” penumbra?
- **One contrast bolus yields two datasets**
 - Vessel patency
 - Infarct versus salvageable penumbra



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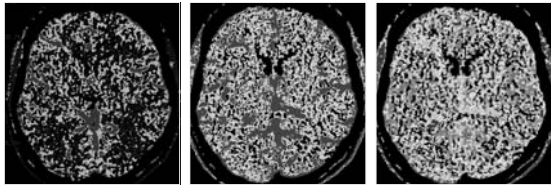
CT Angio & Perfusion



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CT Perfusion Terminology



Blood Flow

Blood Volume

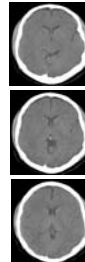
Mean Transit Time
or
Time to Peak

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Case:

Value of CTA/CTP within 3 hour window



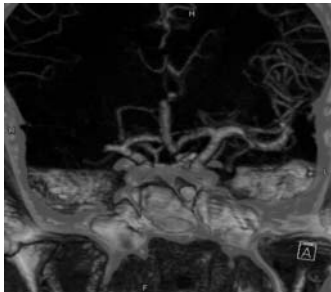
Initial

- 50 yo male
- CT within hour of symptom onset
- Awake, alert, dysarthric
- Fixed right sided gaze
- Left sided weakness

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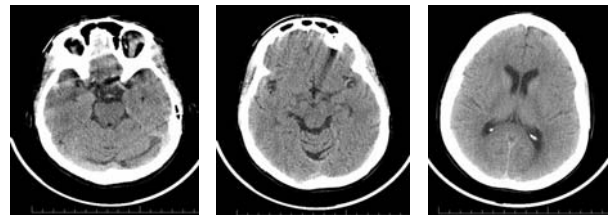
Case:
Value of CTA/CTP within 3 hour window



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Case:
“Wake up” Stroke

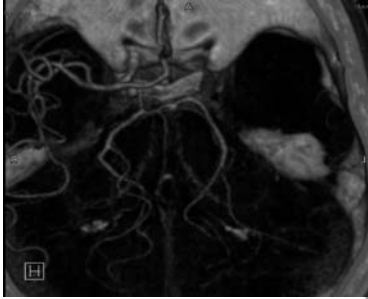


0735 at outside hospital

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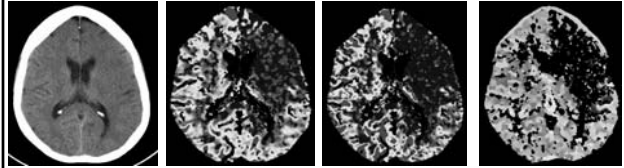
Case:
"Wake up" Stroke



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Case:
"Wake up" Stroke

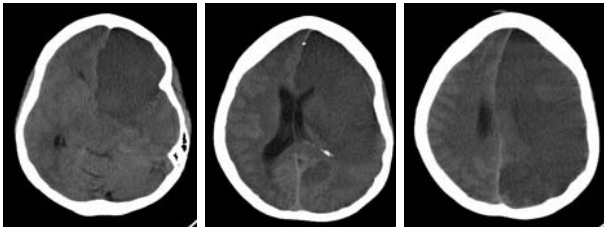


1030 at stroke center

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Case:
"Wake up" Stroke



24 hours later at stroke center

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Conclusions

- Diagnostics may guide future therapies, esp when onset time and penumbra size uncertain
- May be able to maximize benefit and minimize risk through greater understanding of infarct core and salvageable ischemic penumbra
- Future CTA use like non-contrast CT use today
- Software for rapid reconstruction exists
- MRI/MRA still has too many technical hurdles
- EM physicians need to consider next steps

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Treating Intracerebral Hemorrhage in the Anti-coagulated Patient

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Clinical History

- 66 year old male presents with acute onset of aphasia and right sided weakness while eating at home
- Initially complained of a headache
- BP of 220/118 mm Hg
- Accucheck 316
- Initial GCS of 14

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Paramedic's Report

- Patient less responsive than initially
- Aphasia and weakness worsening?
- He is on a "bag o' meds"
 - Per family, started an antibiotic a week ago

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ED Presentation

- ED VS
 - BP 224/124, P 100, RR 16, T 98.8, pulse ox 99%
- Somnolent, but slowly responds to simple commands
- Snores a bit when not stimulated
- Clear lungs and a regular cardiac rate and rhythm
- Neurological screening exam
 - Pupils midpoint, equal and reactive
 - L sided gaze preference
 - R facial weakness
 - R upper > lower extremity weakness
 - Expressive aphasia

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Key Clinical Questions

- What are the key diagnostic issues?
- What are the potential complicating factors?
- What guidelines direct potential therapies?
- What is the urgency of potential interventions?
- What is the relative availability of those therapies in our institution?

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Bag o' Meds



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The Great American Poison



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Which of these belong to this patient?



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Elevated INR Therapy: *The Procedure*

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INR

- Based on the Prothrombin time test
- Sensitive to reductions of Vitamin-K dependent clotting factors II, VII, and X
 - Not factor IX
- Designed specifically for stably anticoagulated patients
 - May be inappropriate test following replacement therapy with either plasma or clotting factor concentrates

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Elevated INR Rx Procedure

- Vitamin K 10 mg by slow IV infusion

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Vitamin K

- Necessary to achieve more than a temporary reversal of anticoagulation
- Adequate response requires at least 2-6 and up to 24 hours
- Anaphylactic or anaphylactoid reactions rarely associated with IV administration
- Safest and most rapidly acting route of administration unclear

Wjasow C, McNamara R. *J Emerg Med* 2003;24(2):169-72.
Fiore LD et al. *J Thrombosis & Thrombolysis* 2001;11(2):175-83.

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Coagulation Factor Replacement

- Options include
 - FFP
 - Prothrombin Complex Concentrates (PCC)
 - Recombinant Factor VIIa
- Normal coagulation achieved more rapidly with PCC, rFVIIa than with FFP

Fredriksson K et al. *Stroke* 1992;23:972-977.
Makris M et al. *Thromb Haemostasis* 1997;77:477-480.

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Bedside Realities:

Can you answer these process questions?

- Is thawed FFP immediately available from your blood bank?
- How long will it take your blood bank to get it to you?
- Does your hospital blood bank or inpatient pharmacy store PCC and rFVIIa?
- What is the relative rapidity of response of each of these agents?

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Elevated INR Rx Procedure

- Vitamin K 10 mg by slow IV infusion
- Fresh frozen plasma (5-8 ml/kg, 1-2 units, 250-500 cc total)

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Elevated INR Rx Procedure

- Vitamin K 10 mg by slow IV infusion
- Fresh frozen plasma (5-8 ml/kg, 1-2 units, 250-500 cc total)
OR
- Prothrombin Complex Concentrate 25-50 IU/kg
 - Dose based on Factor IX units
 - Alternatively, 500 IU initially followed by second administration of 500 IU according to the INR value measured just after the first administration

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Elevated INR Rx Procedure

- Vitamin K 10 mg subq or IVP
- Fresh frozen plasma (5-8 ml/kg) 1-2 units, 250-500 cc total
OR
- Prothrombin Complex Concentrate 25-50 IU/kg
OR
- Recombinant Factor VIIa (40-60 µgr/kg)
 - Usually 3-4 mg total

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Drawbacks to FFP Reversing OAC

- Time-consuming?
 - Can delay neurosurgical evacuation
- May require clinically substantial IV fluid volumes
- Contains a variable content of Vitamin K-dependent clotting factors
- May not completely correct INR
 - May not adequately correct for factor IX
- Risk of viral transmission
 - Not pooled
 - HIV ≈ 1:1,900,000
 - Hepatitis C ≈ 1:1,000,000
 - Hepatitis B ≈ 1:137,000

Makris M et al. *Thromb Haemostasis* 1997;77:477-480. Edward P. Sloan, MD, MPH 

PCC

- Prepared from pooled plasma of thousands of blood donors
 - Less viral transmission risk than FFP
- Contains vitamin K-dependent procoagulant and factors
- Infused over 15 minutes
- Relative thromboembolic risk unclear
- Acquisition cost of usual adult dose ≈ \$450

Abe et al. *Rinsho to Kenkyu* [in Japanese] 1987;64:1327-37.
 Sorensen B et al. *Blood Coagulation and Fibrinolysis* 2003;14:469-477.

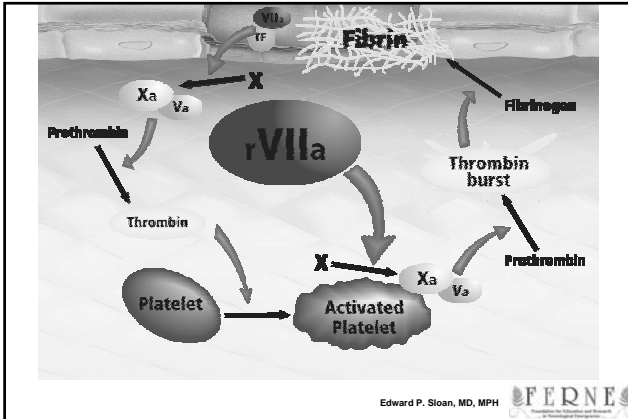
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Recombinant Factor VIIa

- Rapid onset of action
 - Almost immediate
 - Clinically apparent hemostasis in 10 minutes
- Short half life (2.3 hours)
- Relatively high acquisition cost
 - ≈ \$2,500-\$3,500 for 3-4 gm dose

Park p et al. *Neurosurgery* 2003;53:34-39.
 Sorensen B et al. *Blood Coagulation and Fibrinolysis* 2003;14:469-477.
 Novoseven [package insert], Princeton, NJ: Novo Nordisk Pharmaceuticals, Inc; 2003.

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ED Treatment and Patient Outcome

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- ### ED Patient Management
- The BP treated with IV labetalol
 - The INR was noted to be 5.6
 - Vitamin K administered
 - 2 units FFP administered
 - Pt admitted to the neurosurgical ICU
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- ### Patient Outcome
- The hemorrhage size increased slightly on CT with slight intraventricular extension
 - The patient's clinical condition slightly improved gradually
 - Discharged to rehab 10 days after admission
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ED ICH Patient Rx: *A Retrospective*


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- ### OAC Related ICH
- Know the treatment guidelines
 - Know the relative availability at your institution of different coagulation factor replacements
 - Communicate with neurosurgical consultants regarding a potential indication for PCC or rVIIa use
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**FERNE / EMA 2007 Emergency and Acute Care Conference
Neuroresuscitation in Ischemic and Hemorrhagic Stroke
Edward P. Sloan, MD, MPH, FACEP**

Conclusions

- Ischemic stroke and ICH are EM diseases
- Treatment options are easy to identify
- Standards can be met
- Pt outcomes can be optimized
- EM clinical practice can be optimized

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Thank you.

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