Measurement of Tissue Damage from Grasper Trauma in Minimally Invasive Surgery

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Motivation

- Surgeons have a limited sense of touch to safely manipulate tissues in MIS
  - Inadvertent tissue damage from grasping
  - Obvious injury-Bleeding, Perforation
  - Little data on damage due to less severe grasper trauma
    - Possible consequences-

      Scar tissue formation, pain, adhesions
- MIS simulators - no instruction on the consequences of excess stresses during tissue manipulation

Goal

- Measure tissue damage in order to identify stress thresholds for soft tissues that cause significantly increased tissue damage
  - Allow for safer tissue handling during surgery and improved simulator-based training.

Previous studies

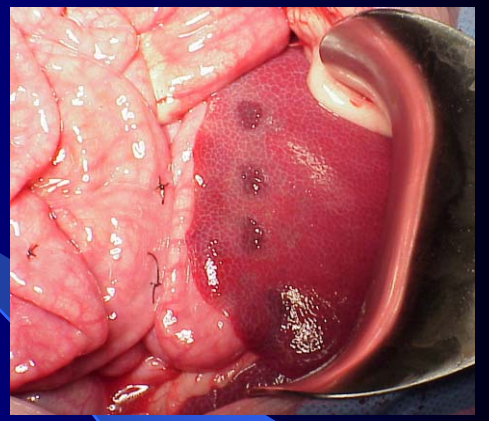
- Grasper trauma on human gall bladder was a function of stress duration (Marucci et al.)
- Acute injury from basic surgical maneuvers measured by histology correlates to chronic effects (Elkins et al.)
- Surgical stress correlates with tissue trauma (Yuen, Miyake)
- Stress modeling suggests injury occurs at stress concentrations (Ressler, Gunter)

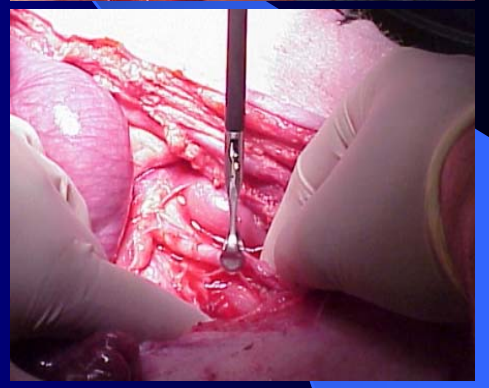
Hypotheses

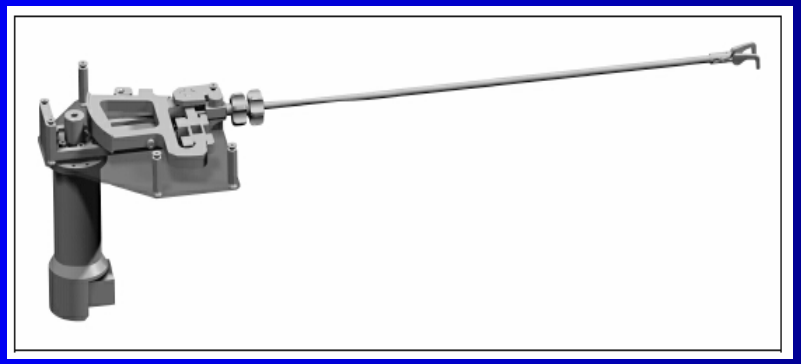
- Grasping during MIS results in cellular death, inflammation, and activation of the coagulation cascade.
- At sub-failure compression stresses, acute tissue damage is a predictable, non-linear function of stress magnitude and duration
- Finite element modeling of compression stresses will calculate stress distribution maps and may indicate mechanisms for mechanically induced damage.

Animal Experiments

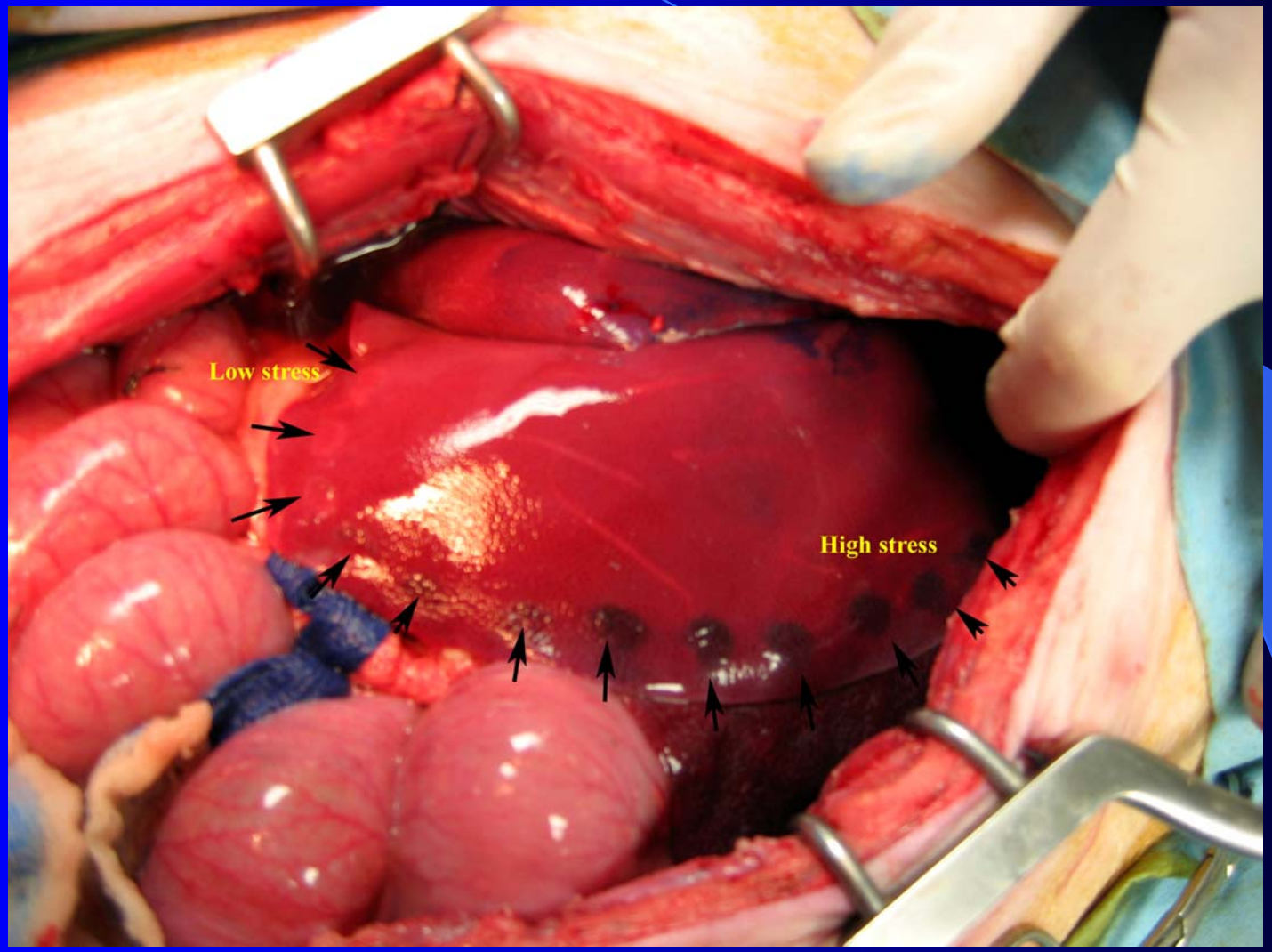
- Animal Experiments using porcine model
- Apply compression stresses in the range of MIS relevant stress magnitudes and durations using the motorized endoscopic grasper (MEG)
- Liver, Ureter, Small bowel
- Allow acute injury to develop
- Harvest compressed tissues





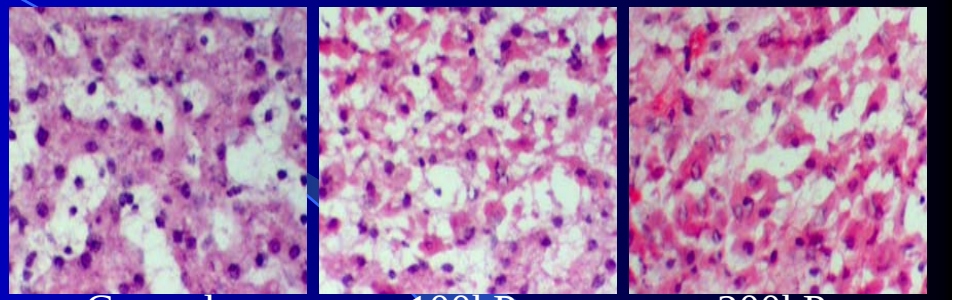


Surface Color Changes



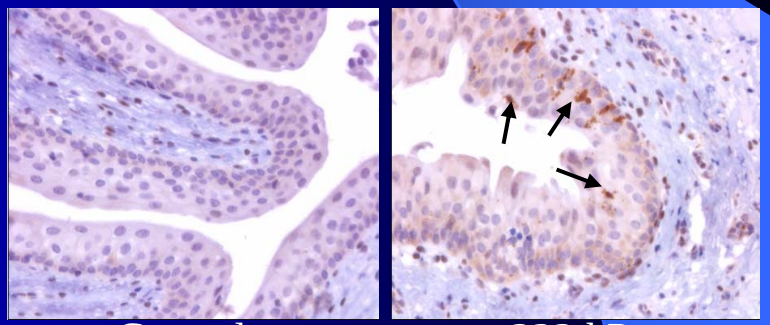
Histology

- Changes in architecture and morphology
  - H&E
- Damage parameters to be quantified
  - Cellular death
    - Anti-activated caspase-3 IHC
  - Inflammatory cell counts
    - Leder stain (granulocytes), CD45 (lymphocytes)
  - Coagulation
    - Anti-fibrin antibody IHC



Control 100kPa 200kPa

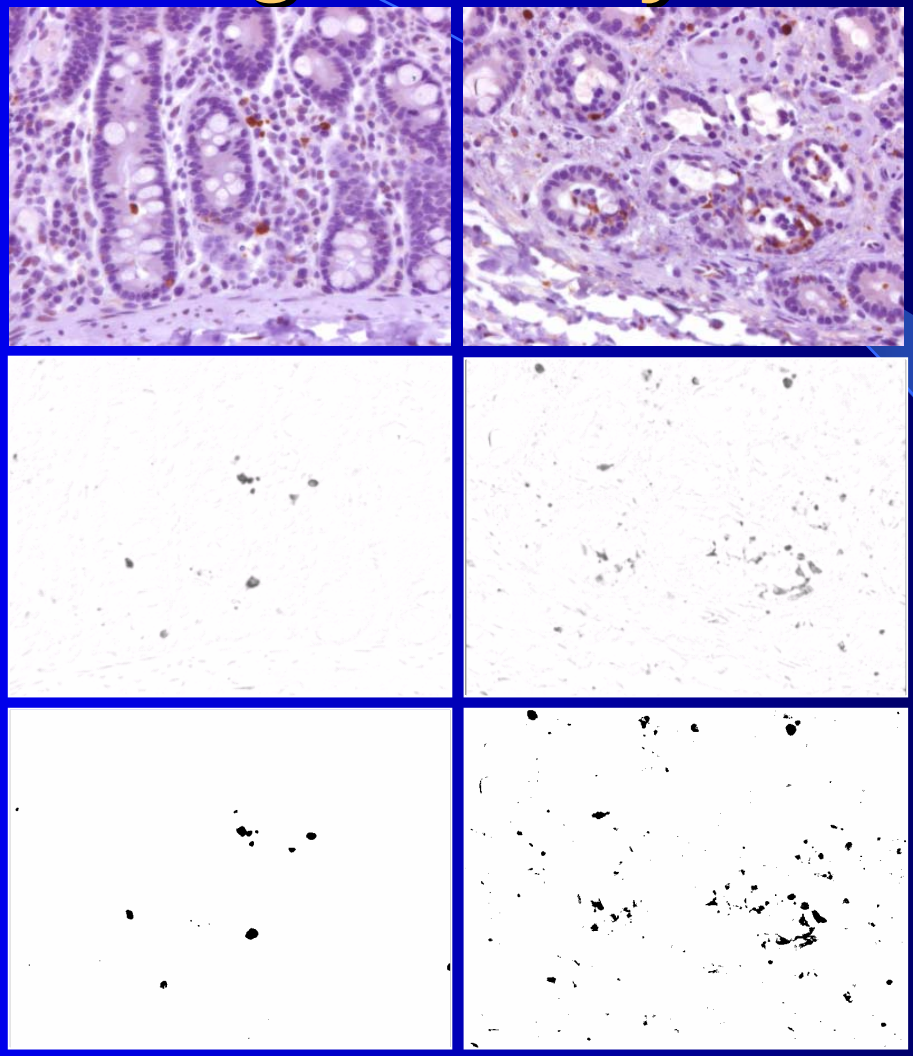
Liver, 400X



Control 222 kPa

Ureter, 400X

Image Analysis



Original image

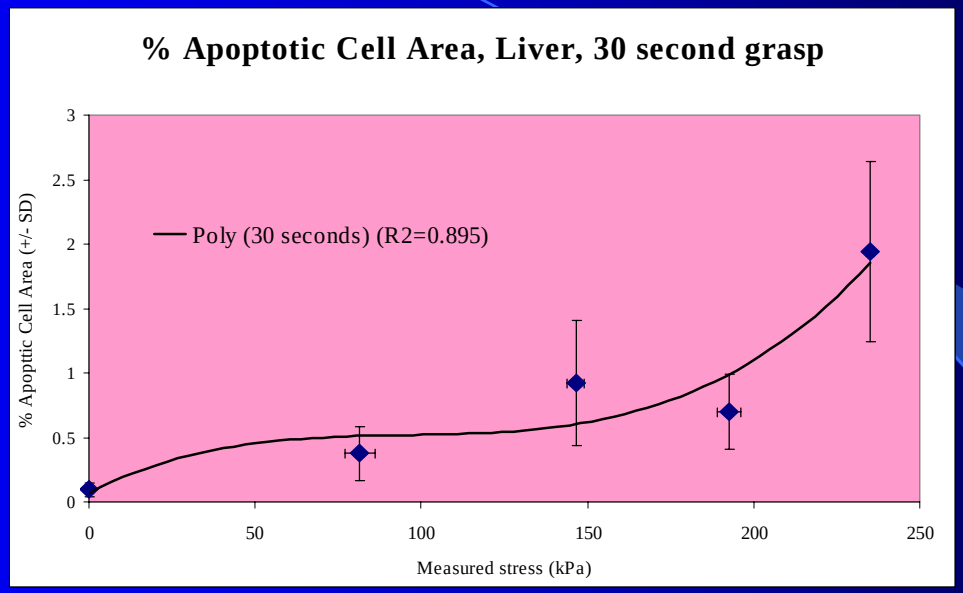
Background removed, Blue channel

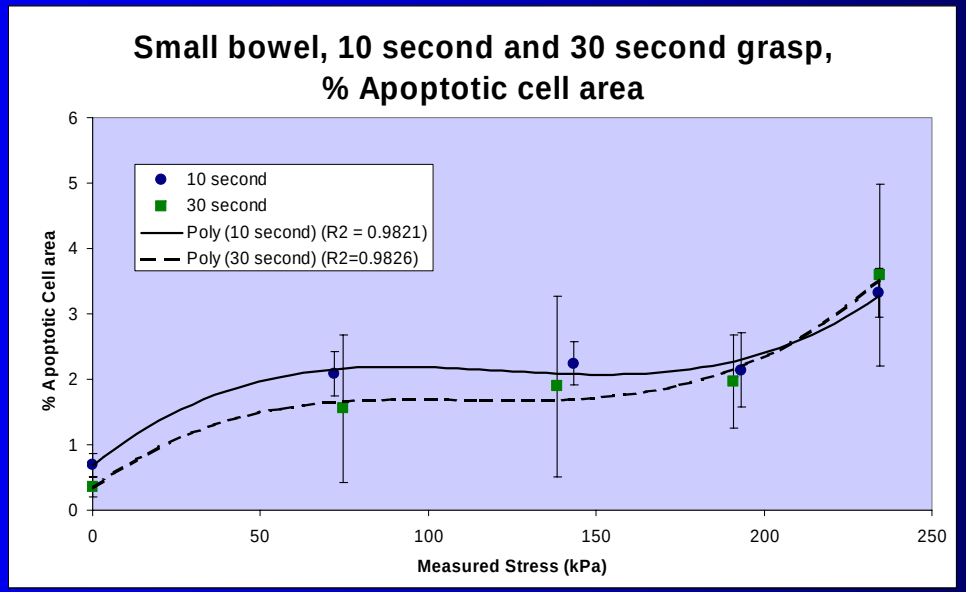
Thresholded image

Small bowel, 400X.

Control (0.38%) 285kPa (1.51%)

Preliminary Results



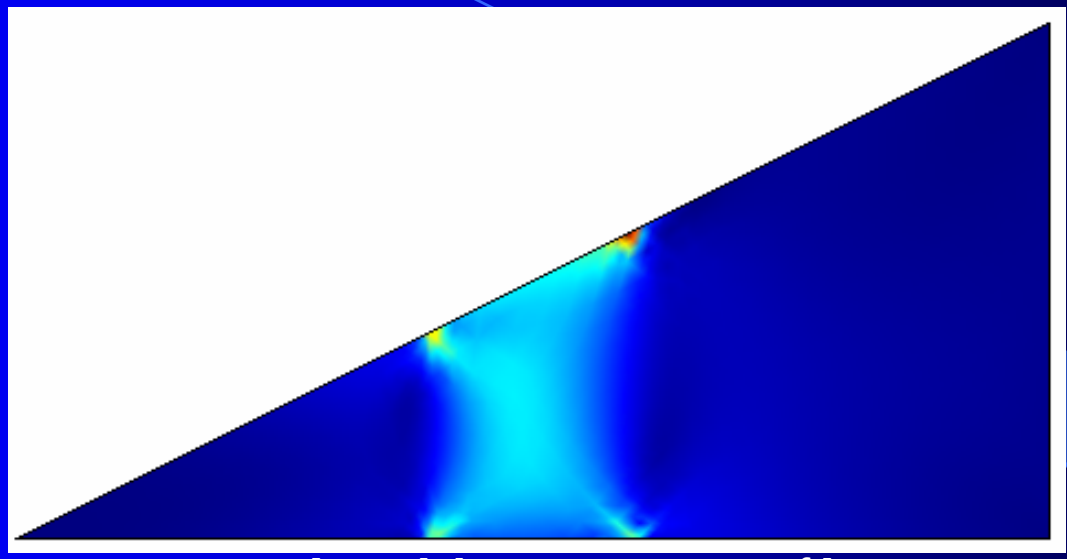


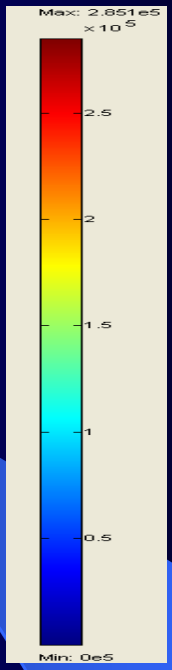
Follow-up with ANOVA and post-hoc analyses

FEM

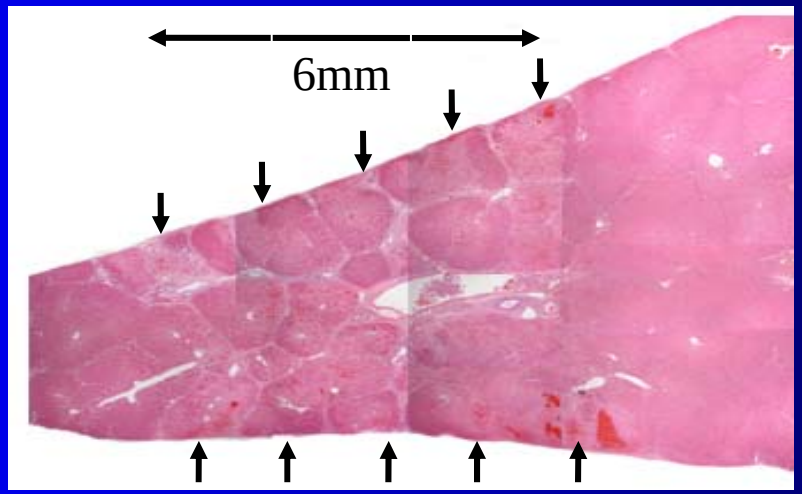
- Stress distribution varies under grasper jaws
- FEM will allow us to compare localized stress values to localized tissue damage
- Comparison of modeling and *in vivo* results may indicate mechanism of injury

FEM





Example with basic properties of liver



Liver (20X), 180 kPa.

Summary

- *In vivo* application of stresses similar to those applied in MIS result in visible tissue damage 3 hours post-injury using histology methods
- A non-linear response damage was seen in small bowel and liver with respect to stress magnitude,
  - May allow for threshold determination
- Basic FEM shows varied stress distribution with expected stress concentrations under grasper jaws with *in vivo* vascular damage greatest at these points

Future Work

- Continue with animal experiments
  - Assess damage based on cellular death, coagulation, and inflammation
  - Use statistical analysis to identify safe ‘threshold’ stress values
- Improve FEM analyses
  - Mimic *in vivo* tissue shapes
  - Incorporate non-linear and nonhomogeneous tissue properties

Acknowledgements

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