CURRICULUM VITAE

Eric Howard Gluck, M.D.

OFFICE ADDRESS: Director of Critical Care Services
Swedish Covenant Hospital
5145 North California Avenue
Chicago, Illinois 60625

OFFICE PHONE: 773.293.3200 egluck@aol.com

MARITAL STATUS: Married, September 6, 1978 (Margaret Ostram)
Children: Heidi Nan, August 1, 1981
Paul Matthew, December 13, 1983
Molly Bea, September 20, 1989

EDUCATION: City College of New York, B.S., 1972
New York Medical College, M.D., 1975

INTERNSHIP: Beth Israel Medical Center, New York, 1975-1976

RESIDENCY: Beth Israel Medical Center, New York, 1976-1978

FELLOWSHIP: Pulmonary Fellowship, University of Utah
College of Medicine, 1978-1980

PROFESSIONAL: January 2000 to present
Director of Critical Care Services
Swedish Covenant Hospital
5145 North California Avenue
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July 1998 to Present
Professor of Medicine
Division Chief Critical Care Medicine
Finch University of Health Sciences/The Chicago Medical School
3333 Green Bay Road
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Sept 1996 to Jan 2000
Chief, Medical Service
North Chicago VA Medical Center
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December 1996 to December 1999
Associate Chair, Department of Medicine
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3333 Green Bay Road
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January 1994 to present
Section Chief Critical Care Medicine
North Chicago VA Medical Center
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January 1994 to July 1998
Associate Professor of Medicine
Chief, Division of Pulmonary-Critical Care Medicine
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North Chicago, Illinois 60064

March 1991 to December 1993
Associate Professor of Medicine
Section of Pulmonary and Critical Care Medicine
Rush-Presbyterian St. Luke's Medical Center
1725 West Harrison Street, Suite 306
Chicago, Illinois 60612

March 1991 - December 1993
Associate Director of Respiratory Therapy
Rush-Presbyterian St. Luke's Medical Center
1725 West Harrison Street, Suite 306
Chicago, Illinois 60612

HOSPITAL APPOINTMENTS:

Professor of Medicine
Department of Medicine
The Chicago Medical School
North Chicago, Illinois
1998

Associate Professor of Medicine
Section of Pulmonary and Critical Care Medicine
North Chicago VA Medical Center
North Chicago, Illinois
1994

Assistant Professor of Medicine
Section of Pulmonary & Critical Care Medicine
Rush-Presbyterian St. Luke's Medical Center
Chicago, Illinois
1991 - 1993

Assistant Professor of Medicine
University of Connecticut Medical School
Farmington, Connecticut
1980 - 1991

Assistant Professor, Department of Biology & Health Science, University of Hartford, Hartford, Connecticut 1986 - 1991

BOARD CERTIFICATION:

National Board of Medical Examiners	1976
American Board of Internal Medicine	1978
Subspecialty, Pulmonary Medicine	1980
Subspecialty, Critical Care Medicine	1991

LICENSURES:

New York	1976 - 1980
Utah	1979 - 1980
Connecticut	1980 – 1991
Illinois	1991 - Present

SPECIAL COURSES:

Laser Workshop, The Institute for Applied Laser Surgery, Inc., 1984

PROFESSIONAL SOCIETIES:

Society of Critical Care Medicine (Fellow)
American College of Chest Physicians (Fellow)
The Chicago Institute of Medicine [Fellow]
American Thoracic Society
Society of Sigma XI
Alpha Omega Alpha
American Society of Law, Medicine and Ethics

AWARDS:

Chemistry Honors Society	1972
Analytical Chemistry Award	1972
Merck Award for Excellence in Medicine	1975
Resident of the Year	1977
John C. Leonard Teaching	1982
Award of Excellence in Teaching	1982
Who's Who of Rising Americans	1990
Governors Community Service Award-ACCP	2000

LECTURES:

Critical Care Nursing Course
University of Utah College of Nursing,
"Anatomy and Physiology of the Respiratory System",
and "Analysis of Blood Gases", September, 1978 and
February, 1979.

LDS Hospital Critical Care Nursing Course,
"Acid Base Metabolism and: Analysis of
Blood Gases", September, 1978.

Connecticut Society for Respiratory Therapy,
"High Frequency Ventilation, When, Why?",
January, 1984.

Emergency Nurses Association, North Central
Connecticut Chapter
"The Physiologic Effects of Asthma and Emergency
Care', November 4, 1987.

Connecticut Society for Respiratory Therapy,
"Jet Ventilation", November 4, 1987.

Symposium - "Use of Inhaled Corticosteroids",
American College of Allergy & Immunology,
March 16, 1988, Anaheim, California.

Connecticut Society of Respiratory Therapy -
"Auto-PEEP and its Clinical Implications".

Niel Institute of Medicine-Lille, France,
"Effect of Ultra High Frequency
Jet Ventilation on Patients with ARDS",
April, 1990.

Resuscitation of Patients with Respiratory Failure,
N.E. Symposium of Emergency Medicine.

New York State Society of Respiratory Care,
Annual Symposium - Alternate Modes of Ventilation
November, 1991.

Pulmonary Grand Rounds/Bay State Medical Center
Ultra High Frequency Jet Ventilation,
November, 1991.

Cook County Medical Society - Review course
in Critical Care Medicine
Emergency Treatment of Asthma
November, 1991.

Mechanical Ventilation - Update ACCP Post Graduate
Course, December 13, 1991.

Pulmonary Grand Rounds - Chicago
Osteopathic Hospital Asthma Update, March, 1992.

Connecticut Society Respiratory Care
Ultra High Frequency Jet Ventilation Update,
April, 1992

Pulmonary Intensive Care Update - National Finish
Meeting.
Tempire, Finland May, 1992.

Fourth Pulmonary Fellows symposium,
Occupation Lung Disease - Ft. Lauderdale, Florida
May, 1992.

Kansas City Society of Respiratory Care
Weaning from Mechanical Ventilation
May, 1993.

Iowa Society Respiratory Care
Ultra High Frequency Ventilation, June, 1993.

Connecticut Society of Respiratory Care,
Helium Therapy, April 1993.

Third International Symposium on High Frequency
Ventilation - Can protocols be written for
ventilator control of gas exchange during HFV?
Dusseldorf, Germany, April, 1993.

Challenges in Critical Care; Respiratory Care
Department of Dartmouth-Hitchcock Medical Center:
Weaning from mechanical ventilation; April, 1994.

Illinois Society of Respiratory Care: Reducing costs
of ventilating patients; High Frequency Ventilation;
June, 1994.

California Society of Respiratory Care: Reducing
costs of ventilating patients; High Frequency Ventilation;
June, 1994.

National Meeting on Monitoring in the ICU, Sapporo,
Japan; Reducing duration of mechanical ventilation;
June, 1994.

Special Symposium on ICU Medicine, Beijing, China;
Reducing stays on mechanical ventilation;
June, 1994.

Respiratory Society Meeting; Taipei, Taiwan;
Optimizing the care of ventilator patients; June,
1994.

Huff and Puff Society of San Francisco: Weaning from
Mechanical Ventilation; May, 1994.

Special Symposium; LSU University Medical Center,
New Orleans; Weaning from Mechanical Ventilation;
May, 1994.

Minnesota Society Respiratory Care - Treatment of
ARDS in 1990's, September, 1994

Course Director- Chicago Critical Care Symposium
July 1995

International Congress of Internal Medicine-Manila Phillipines; COPD - current concepts in management and Iatrogenic respiratory failure; Feb 1996.

University of Southern California- Barlow Respiratory Hospital Symposium on Mechanical Ventilation; The Use of Protocols inWeaning from Mechanical Ventilation. April 1996

University of Miami- 1st Annual Doug Onorato Memorial Lecture; The use of inhaled heliox in the management of acute airway obstruction. June 1996

Course Director-Chicago Critical Care Symposium Aug 1996

Course Director-Chicago Critical Care Symposium July 1997

Computerization of Weaning Protocols-plenary session, American College of Chest Physicians Nov 1999

American Academy of Physician Assistants-Annual Meeting; Chicago 111, June 2000- Diagnosis and treatment of Pulmonary Embolism

American Academy of Physician Assistants-Annual Meeting; Chicago Ill, June 2000- Treatment of Congestive Heart Failure

Illinois Masonic Hospital- Medica1 Grand Rounds- Chicago I11 2000- Use of Non invasive positive pressure ventilation

St Francis Hospital- Medical Grand Rounds- Evanston, I11 2000- Use of Non-Invasive Positive Pressure Mechanical Ventilation

COMMITTEE APPOINTMENTS:

Infection Control Committee
Graduate Medical Education Committee (Swedish Covenant Hospital)
Co-Chairman Hospital Ethics Committee
Chairman VISN 12 Taskforce - Innovations in delivery of health care
House Officer Education October 1997
VISN Quality Council Nov- 1998
Deans committee - CMS January 1997
Finance Committee - American College of Chest Physicians- 1996
Managed Care Committee - NCVAMC-'96
Credentials and Peer Review Comm.- NCVAMC-'96
Executive Committee of Medical Staff - NCVAMC-'96
Research Committee - Chicago Medical School-'95

Medical Education Committee - Chicago Medical School '95
Drug Utilization Committee - NCVAMC
Peer Review Committee, NCVAMC, 1993
Critical Care Committee, NCVAMC, 1993
Utilization Review, Hartford Hospital 1984 - 1991
Computer Committee, Hartford Hospital 1984 - 1991
ICU Planning Committee, Hartford Hospital 1984 - 1991
Emergency Medicine and Educational 1988 - 1991
Liaison Committee
Capital Equipment Comm, Hartford Hospital 1988 - 1991

ABSTRACTS
Eric Howard Gluck, M.D.

1. Brown CC, Gluck EH, Ostram M, "Respiratory support of oleic acid induced adult respiratory distress syndrome with ultra high frequency jet ventilation". Clinical Research, 34:3A, 1986.

2. Gluck EH, Frey TM, 'Measurement of airway pressures in pigs during ultra high frequency jet ventilation". Clinical Research, 34:4A, 1986.

3. Gluck EH, Ostram M, Weinberg B, Use of ultra frequency jet ventilation in patients with ARDS'. Chest, 92:2:67SA.

4. Gluck EH, Mesologites D, Orlando R, "Ultra high frequency jet ventilation in the physiological assessment of pigs with bronchopleural fistula". Chest, 92:2:107SA, August, 1987.

5. Gluck EH, Brown CC, Ostram M, "Ultra high frequency jet ventilation compared to conventional ventilation in the treatment of oleic acid induced ARDS in pigs". Chest, 92:2:67SA, August, 1987.

6. Gluck EH, Frey TM, "Airway pressure measurement in the living pig undergoing ultra high frequency jet ventilation using retrograde catheter technique". Chest, 92:2:109SA, August, 1987.

7. Gluck EH, Renouf R, Shiue ST, Gluck M, 'Effect of ultra high frequency jet ventilation on gas exchange in experimentally induced emphysema. Amer. Rev. Resp. Dis., 137:471A, April, 1988.

8. Shiue ST, Thrall RS, Gluck EH, 'Analysis of bronchoalveolar lavage fluid from rats treated with ultra high frequency jet ventilation'. Amer. Rev. Resp. Dis., 137:374A, April, 1988.

9. Gluck EH, Heard S, Fahey P, "Ultra High Frequency Jet Ventilation in ARDS-Multicenter Results". Chest, 96:2:174SA August, 1989.

10. Winston C, Gluck EH, "Augmented absorption of pneumothoraces using helium-oxygen mixtures. Radiological Society of North America, 1991

11. Keating, Markewitz, Onorato, Gluck EH, "Effect of ventilatory mode on hemodynamics in a porcine trauma model". CCM, Vol. 19, April, 1991.

12. Pettel C, Mohr J, Mathews J, Piraus A, Gluck EH, "Use of UHFJV in management of ARDS; Anesthesiology 73(3):A256, 1990.

13. Lamothe PH, Ujehelyl MR, Gluck EH, "Hemodynamic actions of histamine antagonists during endotoxin induced sepsis; Journal of Clin. Pharm, 32(s):745, August 1992.

14. Gluck EH, Balk R, Casey L, Heydorn P, Nawas Y, Silver M, Bone R, "Esophageal pressure measurements allow more aggressive weaning from chronic mechanical ventilator support. ARRD, 47(4):4:873, April, 1993.

15. Gluck EH, Balk R, Casey L, Nawas Y, Silver M, Bone R, Predicting weanability in chronically ventilated patients", Chest, 48(5):A673, May 1994

16. Gluck EH, Lopez P, Tamul P; The effect of trigger location on the delay between inspiratory effort and gas delivery to the patient. Chest 1995;108;S142

17. Gluck EH, Tamul P; Blasius gas equation can predict the effective endotracheal tube diameter; Chest 1995;108;S142

19. Akbarullah S, Gluck EH; The role of price and study quality on the purchase of medical equipment; Chest 1995;108;S186

20. Gluck EH, Keating K, Kaufman'L, Heard S, Conrad S; Multicenter evaluation of the effectiveness of esophageal balloon manometry during weaning. J of Crit Care Med; 24(l);104A

21. Bader IH, Gluck EH, Rosman J; Comparison of effects of beta 2 agonists nebulizer with oxygen and heliox. Chest 1996; 110; S33

20. Gluck EH, Maldonado, F, Sorresso, D, Gazmuri R. ; Improved care delivery utilizing an intensivist hospitalist system in a Veterans Affairs Hospital. Critical Care Med; 1999; Vol 27, Nol: A155.

PUBLICATIONS:
Eric Howard Gluck, M.D.

1. Gelb A, Gluck EH, Solon A, and Garcia I, "Granulomatous vasculitis of the upper gastrointestinal tract: A case report". Mt. Sinai Journal of Medicine, 45:2, March-April, 1978.

2. Armstrong JD, Gluck EH and Hughes JMB, "Measurement of lung water with helium dilution". Thorax, 1983.

3. Littenberg B, and Gluck EH, "Controlled trial of methylprednisolone in the emergency treatment of acute asthma". New England Journal of Medicine, 314:3, January 16, 1986, pp. 150-152.

4. Nino A, Berman M, Gluck EH, Conway M, Fisher J, Dougherty J, and Rossi M, "Drug-induced left ventricular failure in patients with pulmonary disease". Chest, 94:4, October, 1987.

5. Orlando R, Gluck EH, and Cohen M, "Ultra high frequency and Broncho-Pleural Fistula". Arch. Surgery, vol. 1123, pp. 591, May, 1988.

6. Gluck EH. "Diagnosis of asthma", J of Resp. Dis. 9:S19 - S23, 1988.

7. Schiue ST and Gluck EH, "Use of helium-oxygen mixtures in the support of patients with status asthmaticus and respiratory acidosis". Journal of Asthma, 26(3) pp. 177-180, 1989.

8. Gluck EH, Onorato D, Castriotta R, "On the use of helium oxygen mixtures in intubated patients with status asthmaticus and respiratory acidosis". Chest, 98 pp. 693-698, 1990.

9. Gluck EH, "Use of helium in patients with bronchospasm; clinical advances in the treatment of asthma". 2 (3) pp. 6-9. 1991.

10. Veenstra R, Gluck EH. "A clinical librarian program in the Intensive Care Unit". CCM 20(7); 1038-1042, 1992.

11. Bone R, Eubanks D, Gluck EH. "Beyond the basics: Operating the new generation of ventilators". J Crit. Illness 7(5); pp.- 770-788, 1992.

12. Korst RJ, Orlando R, Yeston N, Molin M, DeGraff A, Gluck EH. "Validation of respiratory mechanics software in microprocessor controlled ventilators". CCM 20 (8) 1152-1156.

13. Gluck EH, Bone RC, Eubanks DH. "The technique of instituting mechanical ventilation"; 7(8):1319-1328, Journal of Crit III. 1992

14. Gluck EH. "Hospital emergency room treatment of acute exacerbations of asthma". Hospital Formulary (27) 1119-1130; November, 1992.

15. Gluck EH, Eubanks D, Bone RC. "Techniques for weaning a patient from mechanical ventilation". J of Crit Illness; 8(l): 121-129; January, 1993.

16. Gluck EH, Heard S, Mohr J, Patel S, Calkins JM. "Ultra high frequency ventilation in the treatment of adults with severe ARDS - a preliminary report"; Chest; 103: 1413; May, 1993.

17. Bone RC, McElwee NE, Eubanks DH, Gluck EH. "Analysis of indications for intensive care unit admission". Chest, 1993; 104:1806-1 1.

18. Bone RC, McElwee NE, Eubanks DH, Gluck EH. "Analysis of indications for early discharge from the intensive care unit". Chest 1993; 104:1812-17.

19. Franklin C, Gluck EH. "The assessment of new technology: who pays for it? Thorax; 48(7):721, July. 1993.

20. Gluck, EH, Barkoviak MJ, Balk R, Casey L, Silver M, Bone R; Medical effectiveness of esophageal balloon pressure manometry in weaning patients from mechanical ventilation: Crit Care Med; 23;504-509; March 1995

21. Gluck EH; Chaos in Research; Thorax; 49;713; July 1995

22. Gluck EH and Keogh B; Application of High Frequency Ventilation in Patients with Acute Respiratoy failure. Clinical Pulmonary Medicine; 1995;2(l):58-65.

23. Gluck EH and Corgian L; Predicting eventual success or failure to wean in patients receiving long term ventilation. Chest 1996; 110(4); 1018-1024

24. Sorresso D, Wagner D, Gluck EH. Postoperative assessment and management of the pneumonectomy patient; Anesthesia Today; 1996; 7(2):14-17

25. Franklin C, Gluck EH. Pitfalls in Ventilator Management before the Patient is Weaned. Hospital E

26. Gluck EH, Sorresso D. Critical Care of the Obsteric Patient. Hospital Physician; April 1998; 3: (1): 1-8.

27. Rodriquez, O, Gluck EH. Sandouk, A. Nosocomial infections in the intensive care unit. Hospital Physician, August 1998; 3: (3): 1-12.

28. Samuel J, Gluck, EH, Upper Airway Obstruction. Hospital Physician, September 1998: 3:(4)

29. Sorresso D, Gluck EH; Hemodynamic Monitoring in the Intensive Care Unit, Hosp Phys, Jan 1999; 4:(1)

30. Sorresso D, Khayr W, Gluck EH; Antibiotic Usage in the Intensive Care Unit, Hosp Phys, April 1999: 4:(2)

31. Sarrigiannidis A, Gluck EH; Update on Mechanical Ventilation in the Intensive Care Unit, Hosp Phys, Aug 1999; 4(3)

32. Gluck EH, McLean M; Ethics in the Intensive Care Unit, Hosp Phys, Dec 1999, 4(4)

33. Snyder R, Gluck EH; Gastrointestinal Diseases in the Intensive Care Unit, Hosp Phys, Feb 2000, 5(1)

34. SnyderR, GluckEH; Bridging to Organ Transplantation: Lung, Heart and Liver, Hosp Phys, April 2000, 5(2)

35. Lutchman D, Gluck EH; Neurologic Emergencies in the Intensive Care Unit; Hosp Phys, July 2000, 5(3).

Chapters and Reviews and Editorial Boards:

1. Endotracheal Intubation and Mechanical Ventilation Quick reference to Internal Medicine: Igaku Shoin, Ltd, New York: Bone R, Rosen R, Editors, 1994.

2. Trauma Management for the Internist: Quick Reference Textbook of Internal Medicine; lgaku Shoin, Ltd, New York: Bone R, Rosen R, Editors 1994

3. Mechanical Ventilation: Principles and Management of Critical Care Medicine: CV Mosby, Chicago: Parillo J, Bone R, Editors: (1995).

4. Non-traditional Mechanical Ventilation: Principles and application of respiratory care equipment: Mosby CV, Eubanlts DH and Bone R, Editors, Chicago. May, 1994.

5. Year Book of Critical Care Medicine - Co- Editor 1992-1998

6. Year Book of Pulmonary Medicine - Co-Editor 1992-1995

7. Consulting Editor- Hospital Physician- Critical Care Medicine 1995-1998

8. High Frequency Ventilation - Acute Respiratory Distress in Adults; Evans T and Haslett C, Editors; Chapman and Hall Medical, London, UK. 1996

9. Series Editor- Hospital Physicians- Critical Care Medicine – Jan 1998 to present

10. Associate Editor - Audio Reviews - Chest Section. - Aug 1998 to present

DRUG RESEARCH

Eric Howard Gluck, M.D.

1982-1983	Co-investigator, Drug Study for Schering Corp., "A Multicenter Long Term Study Comparing the Safety and Efficacy of Albuterol Nebulizer solution 2.5 mg, with Isoproterenol Nebulizer Solution 2.5 mg Delivered by a Compressed-Air Powered Nebulizer to Reverse Bronchospasm".

1983-1984	Co-Investigator, Drug study for Schering Corp., “A Long-Term Study Comparing the Safety and Efficacy of Albuterol Nebulizer Solution, 2.5 mg Isoetharine Nebulizer Solution, 2.5 mg delivered by a compressor powered nebulizer to reverse bronchospasm".
1983-1984	Co-investigator, Drug study for Perdue-Frederick Co., Multi-Investigator open-label study of Uniphyl in patients with asthma, asthmatic-bronchitis or COPD.
1982	Co-Investigator, Drug study for Adria corp., "Early detection of Adriamycin toxicity".
1984	Co-investigator, Drug study for Warner-Lambert/ Parke-Davis Pharmaceutical Research Division, "The effect of sodium meclofenamate in premenstrual asthma".
1985-1986	Co-Investigator, Drug study for Schering Corp., “The effect of single doses of Labetalol and Atenolol on ventilatory function in patients with bronchial asthma”.
1985-1986	Co-Investigator, Drug study for Schering Corp., “Albuterol solution for inhalation in acute asthma”.
1985-1987	Co-Investigator, Drug study for Boehringer-Ingelheim Ltd., “Oxitropium bromide BA 253 90 day multicenter study”.
1985-1987	Co-Investigator, Drug study for Smith Kline and French Laboratories, “Comparative study of the safety and efficacy of monocid versus ceftriaxone for the treatment of community acquired lower respiratory tract infection in patients with chronic lung disease”.
1986-1987	Co-Investigator, Drug stuidy for Smith, Kline and French Laboratories, “Comparison of Tagamet and Placebo in the prophylaxis of Upper Gastrointestinal Bleeding due to Stress- Related Gastric Mucosal Damage”.
1987-1988	Investigator, Drug study for Cutter Biological “Intravenous Gamma Globulin in the treatment of steroid dependent asthma”.
1987-1988	Co-Investigator, Drug study for Schering Corp., “Study of the effects of adding proventil repetabs to theodur in patients with moderate to severe obstructive airway disease”.
1987-1988	Co-Investigator, Drug Study for Schering Corp., “Proventil solution for inhalation for home Use”.

1987	Co-Investigator, Drug Study for Carter-Wallace, Inc., Placebo controlled comparison of the effectiveness and safety of axelastine and controlled release theophylline in the management of theophylline dependent asthmatics".
1987-1989	Co-Investigator, Drug Study for Boehringer-Ingleheim Ltd., “Twelve-week, double blind, parallel study of atrovent solution in COPD patients who are on concurrent alupent therapy”.
1988	Co-Investigator, Drug Study for Schering Corp., “Comparison of theodur 300 mg BID to 450 mg TID”.
1988-1989	Co-Investigator, Drug Study for Nix-0-Tine Pharmaceuticals, “Efficacy and safety of repository corticotropin injection (NP0001) as an aid in smoking cessation”.
1988	Co-Investigator, Drug Study for Pfizer Central Research, “Azithromycin in the treatment of acute lower respiratory tract infections
1989	Co-Investigator, Drug Study for G.D. Searle and Co., “‘A Multicenter comparison of the safety and efficacy of Lomefloxacin and Cefeclor in the treatment of acute exacerbation of chronic bronchitis”.
1989	Co-Investigator, Drug Study for Boehringer-Ingleheim Ltd., “Multiple dose comparison of the combination of lpratropium and Albuterol with its components in a twelve-week parallel study in adults with chronic obstructive pulmonary disease (COPD)”.

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Divisions of Pulmonary and Critical Care Medicine
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Eric H Gluck, M D Division Chief
Cory Franklin, M D Sectioii Chief of Critical Care Medicine
Ashok Fulambarker, M D Section Chief of Pulmonary Medicine

Syed Akbarullah, M D
Raul Gazmuri, M D
Frank Maldonado. M D
Barry Mizock, M D
Karen O'Meara, M D
Joseph Rosman, M D
Jacob Samuels, M D
Domenick Sorresso, M D

Dear Atty Kolis

The following represents my initial impressions after reviewing the medical records of Lawrence Brown who had been hospitalized at University Hospitals of Cleveland.

The autopsy results clearly demonstrate that the cause of death was a massive pulmonary embolism. The cardiac arrest which preccdcd his death was in all medical probability caused by this pulmonary embolism.

The physicians who were caring for Mr. Bronn were an are of the presence of significant deep venous thrombosis. In addition they appeared to be aware that his pulmonary vascular bed was already compromised through the data they obtained from his pulmonary artery catheter. Prior to his surgery there was no pulmonary evaluation which is a deviation from standard of carc when one considers the fact that the patient had a significant smoking history. Subsequently a decision was made to place a filter in the inferior vena cava to protect the patient from a potential pulmonary embolism although a ventilation perfusion lung scan was not performed to determine if embolisation had already occurred. Unfortunately the procedure was delayed until the nest day. During this time period the patient was stricken by a massive pulmonary embolus and died. Had the patient received the filter in a expeditious manner he would not have died. Therefore this represents a deviation from standard of care and in all medical probability resulted in the patient's demise.

If you have any further questions regarding this issue please do not hesitate to call.

Sincerely.

Eric H. Cluck. M.D., FCCP. FCCM
Chief Department Medicine - NCVAMC
Associate Chief Department of Medicine- The Chicago Medical School
Division Chief- Pulmonary and Critical Care Medicine

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Barry Mizock, M.D.
Karen O'Meara, M.D.
Joseph Rosman, M.D.
Jacob Samuels, M.D.
Domenick Sorresso, M.D.

October 7, 1998

Supplemental Expert Report

Dear Attorney Kolis;

Nothing in the deposition of doctors has caused me to alter my opinion in the Lawrence Brown case. Specifically, although the physician states that the heparin was not sub therapeutic, he counter signed a note which suggested that it was and which called for an increase in the dose of heparin to compensate. Data in the literature *suggests* that a PT of 2.0 to 2.5 times control is considered appropriate to treat acute pulmonary embolization. Additionally, the physician claims that the risk *of* sedation exceeded the risk *of* pulmonary embolus and therefore the patient could not undergo the procedure. This could not be further from the accepted standard of care. It is most likely that the patient was receiving continuous alimentation which means that 50 to 90 cc of fluid is administered per hour on a continuous basis. This would be approximately 2 to 3 ounces of fluid. This amount of fluid would be eliminated from the stomach typically in <2 hours or could be suctioned from the stomach using the tube that was already in place. There was also ample time from the time that the diagnosis was made to allow the stomach to empty on its own if *the* decision to proceed with the procedure was *made.* Finally the physician also misunderstood your question about the protection granted by the administration of heparin in that, heparin will anticoagulate the blood immediately but unfortunately it does not protect from future embolization for at least 24hours and more reliably for 48 hours.

After reviewing the deposition of Dr. Lee I do not see anything that was said that would alter my opinion in this case. While the ventilation perfusion scan is difficult to interpret in a *patients* with an abnormal x-ray there is still useful information that can be obtained *from* it. Since the risk of doing the procedure is so minimal it is almost uniformly performed despite its limitations. Dr. Lee clearly states that the patient was a high-risk for surgery, it is still not clear why he then pursued such an aggressive approach to this patient's coronary artery disease when the lesion was only 50 percent (accepted literature is 75% obstruction). Dr. Lee demonstrates a poor understanding of pulmonary physiology and patients with chronic obstructive lung disease when he states that there are many people walking around with oxygen tension of 40 mm of mercury. There are very few patients in this circumstance and those who have these levels of oxygen in the blood require immediate attention. The placements of the fitter does not require an angiogram if there is prior documentation of the embolus or the likelihood is so high that there is no doubt in the clinicians mind that embolization has occurred, In this circumstance the physicians already felt that they had sufficient evidence of the embolus and therefore decided not to proceed with an angiogram. The study of the leg veins clearly demonstrated that the patient was at significant risk for an additional

pulmonary embolus. It is usually the subsequent pulmonary embolus that tips the patients over and has the potential to cause his or her demise.

I hope these additional statements clarify the remarks made by the physicians during the deposition and my opinion regarding them.

Sincerely,

uck, M.D., FCCP, FCCM
Pr e os of Medicine
Chief of Medicine - NCVAMC
Associate Chief of Medicine and Division Chief
of Pulmonary and Critical Care Medicine - CMS

STATE OF OHIO)
) SS:
COUNTY OF CUYAHOGA)

IN THE CIRCUIT COURT OF COMMON PLEAS

ESTATE OF LAWRENCE BROWN,)
Plaintiff,)
vs.) No. 346342
UNIVERSITY HOSPITALS OF CLEVELAND,)
et al.,)
Defendants.)

The deposition of ERIC H. GLUCK, M.D., called for examination, taken pursuant to the provisions of the Code of Civil Procedure and the Rules of the Supreme Court of the Ohio of Illinois pertaining to the taking of depositions for the purpose of discovery, taken before ANNETTE M. MONTALVO, a Notary Public within and for the County of Lake, State of Illinois, and a Certified Shorthand Reporter of said state, at Suite BA-126, North Chicago VA Medical Center, 3001 Green Bay Road, North Chicago, Illinois, on the 5th day of March, A.D. 1999, at 9:28 a.m.

AMM

PRESENT:

DONNA TAYLOR-KOLIS CO., L.P.A.,
(1370 Ontario Street, Suite 330,
Cleveland, Ohio 44113), by:
MS. DONNA TAYLOR-KOLIS,
appeared on behalf of the Plaintiff;

REMINGER & REMINGER,
(113 St. Clair Building,
Cleveland, Ohio 44114), by:
MR. MARC W. GROEDEL,
appeared on behalf of Defendants
Erin Furey, M.D., and Jai Lee, M.D.

REPORTED BY: ANNETTE M. MONTALVO, C.S.R.,
Certificate No. 84-3967.

(WHEREUPON, the witness was duly sworn.)

ERIC H. GLUCK, M.D.,

called as a witness herein, having been first duly sworn, was examined and testified as follows:

EXAMINATION

BY MR. GROEDEL:

Q. Please state your name.

A. Eric Howard Gluck.

Q. Dr. Gluck, my name is Mark Groedel and I represent Dr. Erin Furey. I am going to ask you some questions today about the opinions that you hold in this matter.

Have you ever been deposed?

A. Yes, I have.

Q. Okay. So I take it you understand that the purpose for my examination today is to obtain all of the opinions that you hold in this matter. Do you understand that?

A. Yes, I do.

Q. So when I leave here today, I would like to have a complete understanding of all of the criticisms that you hold as it relates to the care and treatment rendered to Mr. Brown. Okay?

A. Okay.

Q. Before the deposition, we had a few exhibits marked, and I would just like you to identify them for the record.

(WHEREUPON, certain documents were marked Gluck Deposition Exhibit Nos. 1, 2 and 3, for identification, as of 3/5/99.)

BY MR. GROEDEL:

Q. Exhibit No. 2 is a report that -- it is a copy of a report, obviously, because you didn't sign it, at least on this copy, but I just want to make sure that this is in fact one of the reports that you issued on this case?

A. Yes, it looks like it is, I would agree with that. It is not dated, either.

Q. Right.

Do you recall when you drafted that report, or when you typed it out?

A. It has been awhile. I couldn't tell you exactly the date. I mean, I could find out on my computer if you needed to know the precise date that the copy was made.

Q. Okay. Do you have some sort of file

for this case?

A. No, not really.

Q. Okay.

A. I just have some paperwork that we sent back and forth, and letters, and that is it.

Q. Okay. I am also going to hand you now what has been marked as Gluck Deposition Exhibit No, 3. Can you identify that?

A. That one is a letter that I sent to Attorney Kolis after having reviewed, I believe, these two depositions.

Q. It is the depositions of Dr. Lee and Furey?

A. Yes.

Q. Okay. Besides these two reports, Exhibits 2 and 3, have you written any other reports pertaining to this matter?

A. No other reports.

Q. Okay. Do you have any notes that you took while you were reviewing this case?

A. Any and all notes are scrolled in the copy of the record, in the margins of the places that I thought were important.

Q. Okay. So you do have some written

notes in the hospital chart there?

A. Not very many, but there are some, yes.

Q. Okay. And, lastly, I am going to show you what has been marked as Gluck Deposition Exhibit No. 1. Can you identify that for the record?

A. It is a copy of my curriculum vitae.

Q. And it is my understanding that it is slightly out of date?

A. Yes.

Q. In what respect?

A. I was promoted from associate professor to full professor, and there probably are a few more publications that I -- yes, a few more publications that are not included in this that I published recently.

Q. The additional publications that are not included in Exhibit No. 1, do any of them have any relevance to the issues of this case?

A. Not that I am specifically aware of.

Q. Of the articles and other materials that you have written over your career, are any of those relevant to the issues of this case?

A. Let me just review. I have not

specifically written or published anything with respect to pulmonary emboli. I have written some articles with respect to acute respiratory failure. So, indirectly, there might be some cross-references, but nothing directly related specifically that I have written.

Q. Where are we now? What is the location? What building is this?

A. This is building 133 at the North Chicago VA Medical Center. We are in the lower level in my office.

Q. Okay. And who is your employer?

A. The North -- the Veterans Administration, I guess.

Q. Okay. And what is your position here?

A. At this institution, I am chief of medicine and division chief of pulmonary and critical care medicine.

Q. Do you hold any academic positions?

A. Yes, I do.

Q. Tell me about those.

A. I am a professor of medicine at the Chicago Medical School and associate chair of the department of medicine at the Chicago Medical

School, and division chief of pulmonary and critical care medicine at Chicago Medical School.

Q. Is there a relationship between the VA and the Chicago Medical School?

A. The VA is one of the teaching campuses for the Chicago Medical School. It does not have its own university hospital. It is what is called a community based medical school.

Q. Is there a difference between the Chicago Medical School and the medical school that is at the University of Chicago? I mean, those are two separate institutions?

A. Yes, they are.

Q. Okay. Is there any relationship between the two?

A. None whatsoever.

Q. Okay. Do you have any teaching responsibilities?

A. Yes, I do.

Q. Tell me about those.

A. Well, as division chief for pulmonary and critical care, I am directly responsible for the education of the pulmonary and critical care fellows at the institution and at the medical

school.

As chief of medicine, I am directly responsible for a significant amount of the education for the interns and residents.

And as associate chair of the department of medicine at the school, I am responsible for the education of the medical students from first year all the way through the fourth year.

Q. So here at the VA, you do have critical care fellows?

A. Yes, we do.

Q. What percentage of your professional time, would you say, is involved in hands-on medical care?

A. I would say probably about 33 percent.

Q. And how is the rest of your professional time divided?

A. I would say probably about 15 to 20 percent research, about another 15 to 20 percent teaching, you know, nonrelated actual patient care, just run conference and things like that, and the rest is administration.

Q. Okay. Besides the VA, do you have

privileges at any other hospital?

A. No.

Q. Has your license to practice medicine ever been suspended, revoked or limited in any fashion?

A. No.

Q. Have your hospital privileges ever been suspended, revoked or limited in any fashion?

A. No.

Q. Have you ever had to practice under a consent decree or something along those lines?

A. No.

Q. Okay.

A. I don't even know what that is.

Q. Okay. Prior to your involvement in this case, did you ever review a medical malpractice case?

A. Yes.

Q. Can you give me some idea to approximately how often you do that sort of thing?

A. I would say I would review somewhere between eight and twelve cases a year.

Q. And for how long have you been doing that?

A. Maybe since I have been in Chicago, so I would guess about eight or nine years.

Q. And would you be able to give me an approximate breakdown as to whether those reviews are on behalf of the people bringing cases or doctor defendant cases?

A. I would say probably two to one, people bringing cases.

Q. Approximately how many depositions would you say you have given on a yearly basis in medical malpractice matters? I am not looking for an exact number.

A. Probably three or four.

Q. What do you charge for reviewing cases?

A. $350 an hour.

Q. What do you charge for a deposition?

A. Same thing.

Q. Have you ever testified in a courtroom in a medical malpractice case?

A. Three times.

Q. Okay. And were they on behalf of the defendant physician or the plaintiff bringing the case?

A. One for the defendant physician and two

for plaintiffs bringing a case.

Q. Okay. And how much do you charge for trial testimony?

A. The same thing, $350 an hour plus whatever time extra I have to spend if I have to go out of town.

Q. Okay. Prior to this case, have you ever reviewed any other cases for Ms. Kolis or her law firm?

A. Not prior to.

Q. Okay. Subsequent to this case, have you reviewed any other cases?

A. One other case.

Q. Okay. Do you know how it is that she came to find you to review this matter on her behalf?

A. Yes. Actually, it was by accident. She was looking to involve a different physician in Chicago, Dr. Franklin, I believe, who was at Cook County Hospital, and he was not interested in doing it, but he recommended me to her.

Q. Okay. Do you advertise your availability as a medical legal expert?

A. No.

Q. Is your name on the roster of any companies that are able to obtain medical experts for attorneys looking for them?

A. Not that I'm aware of.

Q. Tell me a little bit about your current practice. Just run through a normal week for me.

A. Well, actually, it is very variable. It just depends. Like, this week, I am on pulmonary service, so typically I will come in, do my administrative work, catch up on literature, and then I will do pulmonary rounds with the fellow who is on service, usually resident and medical students on service as well. And then I will eat lunch, and in the afternoon if we have any procedural things that have to be done, we do it then, and then I will just catch up on other work that needs to be done the rest of the day.

If I am on critical care service, then seven days a week I will round in the intensive care unit in the morning from, oh, about 8:30 until 10:30 or 11:00, then be available for any problems that arise the rest of the day and intermix those and I will try to catch up on my administrative stuff and my research and things

like that.

I usually spend about an hour with my research associate several times a week catching up on our research projects and protocols that are being submitted and processed. And then I go to a zillion meetings.

Q. Okay. These are administrative meetings?

A. Yes.

Q. Now, from your CV, it appears as though you have received formal training in internal medicine and pulmonary care, and is that it?

A. Yes.

Q. Okay. So you are board certified in internal medicine?

A. Yes.

Q. And in pulmonary medicine?

A. Yes.

Q. Any other board certifications?

A. Critical care medicine.

Q. Okay. Did you participate in any critical care fellowship?

A. No, actually, when I did my pulmonary training, there was no critical care fellowships

so I sort of got grandfathered in.

Q. So by virtue of the your critical -- your pulmonary training, you were able to become board certified in critical care?

A. Right.

Q. Did you pass all of those certification examinations on the first attempt?

A. Yes, I did.

Q. Have you reviewed any of the expert reports submitted on behalf of the defendants in this case?

A. Yes, I did.

Q. A report from Dr. John Hoyt?

A. If I can find it. I don't recall the people's individual names.

Q. Okay.

A. No, I don't have it here, so I have to rely on Donna Kolis to tell me what she sent me because whatever she sent me, I did review.

Q. Let me ask you this:

Do you know of a Dr. John Hoyt?

A. Personally?

Q. By reputation or personally.

A. I am not sure. There is a Dr. Hoyt who

is the chairman of the department of medicine, pulmonary medicine, at the University of Utah, but I don't know if it is the same person. I don't know him personally, but I know who he is because I trained at the University of Utah.

Q. Do you know of a Dr. Hoyt who has published anything in the field of critical care medicine?

A. If he did, I am not aware of his specific publications.

Q. Okay. How about Dr. John Downs, do you know of him?

A. Yes. Dr. Downs is fairly well known.

Q. And what do you know of his reputation as a critical care specialist?

A. He is pretty well respected.

Q. Have you read any of his articles or textbooks?

A. I don't think I have read his textbook word for word, but certainly I have seen some of his articles from time to time.

Q. Do you own any one of his textbooks?

A. No.

Q. Have you been in the audience whenever

he -- whenever he has lectured at any time on critical care issues?

A. I can't remember, per se, that I have actually been in the audience when he has lectured.

Q. Would you agree that he has a reputation as a critical care specialist of national repute?

A. Yes, probably, yes.

Q. Now, you have already told us the materials you have reviewed. That would be the two depositions, the one of Dr. Furey and one of Dr. Lee, you have reviewed the hospital records, the autopsy report, I take it?

A. Yes.

Q. And a pathology slide --

A. Yes.

Q. -- from the autopsy?

A. A single pathology slide.

Q. A slide. Okay.

Anything else?

A. Well, just those -- the reports that I forgot about, the expert report that Donna sent to me.

Q. Okay. So you think you did review the expert reports from Dr. Hoyt and Dr. Downs?

A. Yes.

Q. There was a report also from Dr. Mendelson, do you know if you reviewed that one, a pathologist from Cleveland?

A. If it was sent to me, I did, and so I don't recall exactly how many reports were sent, but I reviewed them all.

Q. Okay. Did you review any medical literature specifically for this case?

A. No, not really.

Q. As you sit here today, is there any literature out there relevant to the issues of this case that you would consider to be authoritative?

A. There is just so much that there is not just one specific thing that -- it is a very common problem, so there are tons of literature out there. I don't know that one would be more authoritative than any other.

Q. When you say it is a "common problem," what are you referring to?

A. Pulmonary emboli.

Q. Are you aware of any specific literature that deals with when or whether a Greenfield filter should be placed and what considerations go into whether it should be placed?

A. I would not be able to specifically cite an individual article or articles right now. There are articles that would support the use of those filters.

Q. Okay. Would you consider yourself an expert in the field of pathology?

A. No.

Q. You haven't had any formal training in pathology, have you?

A. No.

Q. Would you consider yourself an expert in the field of anesthesiology?

A. No.

Q. You haven't had any formal training in anesthesiology, have you?

A. No.

Q. Have you ever provided anesthesia for patients?

A. No.

Q. So I take then you have never provided anesthesia for a patient who is required the insertion of a Greenfield filter?

A. Well, I guess in that regard I would have to say that I provided similar anesthesia in other circumstances, but not in the circumstance of placing a Greenfield filter.

Q. Okay. So just so I am clear, you have never provided anesthesia for a patient who needed a Greenfield filter?

A. Correct.

Q. Okay. But you are saying you have provided some sort of anesthesia for other types of procedures, is that what you are saying?

A. Yes, the same kind of anesthesia that would be used in a Greenfield filter I have provided for other kinds of critical care and pulmonary procedures.

Q. What sort of anesthesia would that be?

A. Intravenous, what we now call conscious sedation.

Q. Okay. When you have provided that anesthesia, have you consulted with an anesthesiologist before doing so?

A. No, because I am credentialed to do that, as are all the critical care, pulmonary and gastroenterologists.

Q. Besides providing IV sedation, have you ever provided any other form of anesthesia for a patient?

A. Not for a patient.

Q. Have you ever intubated a patient for the purpose of providing anesthesia?

A. No.

Q. I take it you have intubated patients, though?

A. Yes.

Q. That is something you have done quite frequently?

A. Oh, yes.

Q. Okay. But when you have intubated a patient, it is to provide emergency ventilatory assistance?

A. Not always emergency, it is usually to provide access to the patient's airway, sometimes emergent and sometimes electively.

Q. Okay. In your institution, who is the individual who would provide anesthesia for

patients who require the insertion of a Greenfield filter?

A. At the VA? Here?

Q. Yes.

A, It would probably be the radiologist, one of the radiologists who do that would be credentialed to provide conscious sedation.

Q. In this institution, do anesthesiologists get involved in the providing of anesthesia for patients who require a Greenfield filter?

A. I would say that if they were available, they could -- we don't have full-time anesthesiologists here, we contract out the anesthesiology services, so if they are here, they would do it.

Q. So what sort of anesthesiology coverage do you have here at this institution?

A. We have a group of anesthesiologists from the community who are contracted to come into the hospital and do all our anesthesia --

Q. Okay.

A. -- in the operating room.

Q. And this group services other

institutions as well, I take it?

A. I don't know what institutions, but, yes, they have their own practice somewhere else.

Q. Okay. And is it because they are independent of the VA they may not be always available to provide anesthesia for a person that needs a Greenfield filter and is that why the radiologist might do it?

A. Well, that is part of the reason, but, certainly, the radiologists are credentialed and competent to provide conscious sedation for other procedures as well, so if in fact they wanted to do that, they could.

Q. Okay. Have you ever placed a Greenfield filter?

A. No.

Q. Have you ever ordered one to be placed in a patient?

A. Yes.

Q. About how many times would you say you have done that in your career?

A. Eight, ten times maybe.

Q. And that would be in your entire career?

A. Yes.

Q. Okay. Now, I note here that you are the division chief of the critical care department?

A. Where?

Q. At the VA here?

A. Yes.

Q. Okay. In this institution, is there an SICU and an MICU or is it altogether?

A. One unit.

Q. And what is it called?

A. Intensive care unit,

Q. So they make no distinction between a postsurgery intensive care patient and a medical intensive care patient?

A. Correct.

Q. In this institution, do they perform cardiac bypass surgery?

A. No.

Q. Have you ever been involved in the care of a post-bypass graft patient?

A. Oh, absolutely.

Q. When was the last time you were involved in the care of a post-CBAG patient?

A. About five or six years ago.
Q. And it was here or at another institution?
A. At another institution.
Q. Okay. How long have you been at this institution?
A. Five years.
Q. Okay. And where were you before?
A. Rush-Presbyterian St. Luke's Medical Center in Chicago, and before that Hartford Hospital in Connecticut.
Q. And while you were at Rush, did you take care of post-CBAG patients?
A. Yes.
Q. Can you identify for me the critical care textbooks that you own?
A. (indicating).
Q. Right behind me?
A. I only have one, I think. There are other textbooks up there that have some critical care in them, but the major one is that one by Parrillo and Bone. I got that for free.
Q. Oh, okay.
A. Just because I worked for Parrillo and

Bone at Rush-Presbyterian St. Luke's.

Q. Are there any other major critical care textbooks that you are aware of that you don't own?

A. Oh, yes, there are several.

Q. What are they?

A. There is another one by Dr. Bone, which I don't know the specific title of it, but there is also one by Dr. Wyle, and there is one on ventilation by Dr. Tobin, there is one on critical care by Dr. Hall. They are all very, very good. We have them all in the library and most of them are on the Internet so we have access to them without actually ordering them anymore.

Q. Okay. I guess the editors aren't too happy about that?

A. Oh, no, we pay for access to them.

Q. Okay.

A. I think they actually do better.

Q. I would like to ask you just a few general questions about pulmonary embolism.

Would you list for me the usual signs and symptoms or the hallmark signs and symptoms of pulmonary embolism?

A. Yes, pulmonary embolism, unfortunately, presents with very nonspecific signs. So most of the signs and symptoms are -- can be attributed to a pulmonary embolism or other disease. The most common presenting symptom is shortness of breath, an increased heart rate, a drop in oxygenation. There are some changes on EKG that are seen from time to time, but not all that often and not all that specific.

Q. Okay.

A. Chest pain, sorry.

Q. Pleuritic chest pain?

A. Yes, pleuritic chest pain.

Q. When you say that pulmonary embolism can often mimic other diseases, what other disease processes can look just like pulmonary embolism?

A. Pneumonia, congestive heart failure, pleurisy.

Q. Is the chest X-ray of a patient who has got pulmonary embolism frequently normal?

A. Yes. In fact, that is one of the ways that you actually -- that is one of the things that you actually use to help make the diagnosis.

Q. If you have got a symptomatic patient,

but a normal looking chest X-ray?

A. Yes.

Q. Are you familiar with the death rate in hospital -- the in-hospital death rate for patients who have PE, pulmonary embolism?

A. Well, I have never actually heard it placed like that. We usually look at the people who die prior to arriving at the hospital from pulmonary embolism, and then look at the people who inadvertently are found to have pulmonary embolism at autopsy, but for someone to actually arrive at the hospital and develop a pulmonary embolism and die in the hospital is very small.

Q. It is my understanding in this case that your essential criticism is that Dr. Furey did not place the Greenfield filter in a timely fashion or have it placed in a timely fashion?

A. That is one of the major aspects of the criticisms, yes.

Q. I would like to ask you some questions about the initial care that this patient received.

Would you agree that Mr. Brown had a heart attack prior to his arrival at University

Hospital?

A. Yes.

Q. And do you have an opinion as to the cause of his MI?

A. Just probably coronary artery disease.

Q. Okay. Do you have any criticisms of the decision to operate on Mr. Brown for his coronary artery disease?

A. Sort of. They center around the fact that it was a patient who presented with a very huge smoking history and low levels of oxygen in his blood and they sort of rushed right away to do the surgery on the patient without getting sufficient data, I thought, to assess the patient's risk for surgery and potential complications thereafter. And, in addition, the lesion that he presented with which was a 50 percent lesion, at least that is what is described in the chart, the LAD, is not a medical emergency. I mean, it is certainly a very important blood vessel in your body and certainly therapy is indicated; however, there is no need to immediately rush to do surgical intervention. It was not a medical emergency.

So what I was thinking is that the physicians had time to better assess the patient's risk for surgery and insure that he could have a smooth recovery.

Q. All right. Do you believe that it was a breach of the standard of care to actually perform bypass surgery based upon the extent of the lesion and where it was located?

A. I think that you would probably be able to find numerous physicians who would say that this patient could do just as well with medical therapy as with physical therapy, but as a breach of standard of care, that would probably not be true.

Q. Just so the record is clear, you don't believe it is a breach of the standard of care to have actually performed the surgery, correct?

A. For the specific procedure I would have to agree with you, but in the whole context of the patient, doing it as they did it, when they did it, they probably needed more data before they went to the OR for the patient.

Q. Okay.

MS. KOLIS: And I don't interject a lot at depositions, as Marc well knows, but for purposes of making the record clear, Marc, Dr. Gluck has no intentions of testifying on this issue at trial. He and I have discussed it.

MR. GROEDEL: Okay.

BY MR. GROEDEL:

Q. But just so I have a complete understanding of what your opinions are, and I appreciate your comment there, Donna, I thank you for it.

Is it your belief that the timing of the surgery was inappropriate?

A. Yes, I think that patient should have had further evaluation after the angiogram suggested that there was a lesion that could be treated either with medical care or surgical care prior to having the operation, yes.

Q. Okay. And what evaluations do you believe should have been performed?

A. At a minimum, pulmonary function tests, and probably more optimally a consultation with a pulmonologist.

Q. Do you have an opinion as to what a

pulmonary function study, if performed, would have shown?

A. It might have demonstrated severe obstructive lung disease, severe reduction in oxygenation, perhaps might have demonstrated some reversible components of this obstructive lung disease, which then would have been amenable to some treatment which would have improved the patient's lung function prior to surgery which would have made it less likely they would have problems postoperatively.

Q. Do you believe it is more probable than not that if a pulmonary function study had been performed, Mr. Brown would have been in more optimal pulmonary shape prior to surgery, or is that speculation?

A. Well, it would be difficult to say for sure, you would have to know what the results of the test were, but if it did show that the patient had a significant reduction in oxygen level prior to the surgery or reversible component, then the risk could have been reduced.

Q. But you don't know if those findings would have been made?

A. Correct.

Q. Based upon what you saw in the record, did you notice whether Mr. Brown had any untoward event during the operation itself from a pulmonary standpoint?

A. Nothing that I was able to discern from the medical records.

Q. Do you believe that it was a breach of the standard of care not to have Mr. Brown evaluated by a pulmonologist prior to his surgery?

A. Yes.

Q. Besides the ordering of a pulmonary function study, is there anything else that a pulmonologist in all likelihood would have ordered had that specialist evaluated Mr. Brown prior to surgery?

A. All depends on what the pulmonary function tests are, what the blood tests showed, what his evaluation of the patient's risk for surgery was. The pulmonologist could have made a very significant argument with the cardiographic surgeon that the appropriate thing to do would be to treat this guy medically and see how he responds to therapy and then bring him back in two

weeks, four weeks, a month or never for surgery, if he felt that the risk was too high to undergo that type of surgery.

Q. What you have just said, though, is pretty speculative though, right?

A. Well, yes, if you don't have the data, you can only assume what the -- I mean, if the pulmonary function tests were nice and normal or reasonable, he would have just said fine, I will just go ahead and do the surgery and I will just watch the patient postoperatively with you.

Q. And that could have been the case here, too?

A. Unlikely, but, yes, it could have been the case. The guy is a five pack a day smoker. It is unlikely he is going to have normal lung function.

Q. But his lung function could have been sufficiently acceptable that it would have been okay for him to go through with bypass surgery?

A. Yes. I would say the odds favored it would not -- it would have been abnormal, but okay for surgery.

Q. Now, postsurgery, we know that

Mr. Brown's level of pulmonary blood flow was measured -- I shouldn't say blood flow, but pulmonary hypertension was assessed?

A. Yes. He had elevated pulmonary artery pressures.

Q. And what would you attribute those elevated pulmonary artery pressures to postsurgery?

A. Based on the patient's presentation, the most likely explanation would be chronic obstructive lung disease. There are obviously other causes of pulmonary hypertension which could have been involved, but would not be discernible based on the history that is presented to us at this point in time.

Q. And when you say obstructive lung disease, is that the sort of problem that was caused by his long history of smoking?

A. Yes.

Q. And so this is a long-standing problem that he has got that, this lung disease?

A. Which part of it, the elevated pulmonary artery pressures or the COFD?

Q. The COFD.

A. I would say the COPD has been going on for a while, yes.

Q. Do you have any thoughts as to how long he was having pulmonary hypertension for?

A. Can't tell just by looking at a given pressure measurement. The higher they are, typically the longer the patient -- it has been going on. Most of the time though, you have to establish a temporary relationship between symptomatology or a change in symptomatology to establish when the pressures changed.

Q. Okay. Do you have any information to allow you to do that in this case?

A. No, I don't.

Q. Based upon the degree of pulmonary hypertension that we saw in this case, would you be able to characterize the extent of Mr. Brown's underlying lung disease?

A. I would have to say that it was very significant,

Q. And when you say very significant. You are talking about his COPD?

A. Yes, if in fact that was the sole cause of his elevated pulmonary artery pressure.

You need to have 50 percent or more of your blood vessels abnormal in your lungs before the blood pressure goes up like that. It is not a trivial reduction in lung function associated with pulmonary hypertension, it is a very significant one.

Q. But severe and chronic COPD can cause the type of pulmonary hypertension that we saw with Mr. Brown postoperatively?

A. Yes.

Q. We also know that after his surgery he was hypoxic?

A. Yes.

Q. And is hypoxia something that can also be seen with patients who have underlying lung disease like Mr. Brown had?

A. Mild hypoxia is fairly common with emphysema and it can be associated with a normal carbon dioxide level. In order to get oxygen levels as low as he had postoperatively without a increase in carbon dioxide levels, you would need to have an additional insult to the lung.

Q. And what additional insult are you talking about?

A. Volume overload, for example, pulmonary embolism for instance, pneumonia, atelectasis, something else in addition. We don't usually see patients with normal PCO2s and that level PO2 unless there is something else going on.

Q. In Mr. Brown's case, was he volume overloaded postsurgery?

A. I couldn't tell. I don't think so.

Q. Okay. Was he suffering from atelectasis postsurgery?

A. The X-rays that I read, the reports did not suggest that.

Q. Did you see any of the X-rays films, by the way?

A. No, I just read the reports.

Q. Okay. Actually, there is an X-ray report dated May 30, which talks about in the lower lobes there is evidence of combined segmental subsegmental atelectasis with airspace consolidation in the left lower lobe?

A. Yes.

Q. Would those be findings consistent with atelectasis?

A. Yes, mild atelectasis, not sufficient

to lower the PO2 to 40.

Q. Okay. Even in combination with a patient that has severe COPD?

A. Even in combination with a patient who has severe COPD.

Q. How about pulmonary edema?

A. If the pulmonary edema was significant, I mean, just mild pulmonary edema wouldn't do it, but significant pulmonary edema could lower the PO2 to 40.

Q. How would you characterize Mr. Brown's pulmonary edema?

A. Mild. The X-ray readings vary, actually, day-to-day, some a little bit more, some a little bit less, but nobody ever actually calls it. Most of the time, if I recall, there are adjectives of "congestion" and "mild" and "moderate," but no one actually says, "pulmonary edema". The assumption is there is lung water, but not a huge amount of lung water.

Q. So postoperatively, we know he has got COPD, which he has had for a long time, he has some degree of atelectasis, he has got some degree of pulmonary edema. What other conditions does

Mr. Brown have that could effect his degree of hypoxia?

A. Say the question once more.

Q. Besides his underlying COPD, his atelectasis and his pulmonary edema postsurgery, what other conditions did Mr. Brown have that would have led to his or contributed to his hypoxia?

A. Potentially, pulmonary embolism. That was undiagnosed.

Q. Okay. Anything else?

A. He had -- eventually he developed some mild renal failure and then elevated temperature, but other than that, nothing else that I recall.

Q. Would the renal failure contribute to his degree of hypoxia?

A. No, but it could be one of the reasons that he was having trouble with mild congestive failure.

Q. And how about an elevated temperature, would that contribute to his degree of hypoxia?

A. Typically not.

Q. Did Mr. Brown have many of the signs and symptoms consistent with a pneumonia or

infectious process in his lungs?

A. Initially, it did not appear that way. He was not producing purulent type sputum. He did have a low-grade fever, but there were no signs of lobar consolidation on his chest X-ray.

Q. Would you say that that situation changed?

A. Well, at times, the -- there are reports in the chart of being suctioned up for what appeared to be some purulent material. It is difficult to tell whether it -- the only way to distinguish when you suction up purulent material or you have pneumonia or just bronchitis is from the X-ray, and none of the X-rays clearly demonstrated what somebody would have called a lobar pneumonia that I recall off the top of my head. There may have been some instances where from day-to-day there was more or less consolidation, but nothing that stood out to be a massive type or a significant -- I wouldn't say massive, a significant type of pneumonia.

Q. Is interstitial and alveolar consolidation consistent with pneumonia?

A. That would be one of the things that

you could see with it. You could also see that with atelectasis and you could also see that with edema.

Q. How about bilateral pleural effusions?

A. That is not common with pneumonia. I mean, if you have massive pneumonia bilaterally involving lungs on both sides, you could get an effusion, but somebody with that kind of pneumonia would be devastatingly ill with sepsis.

Q. What about the presence of bullae in the upper lungs, what would that be consistent with?

A. That is part of his COPD. Probably has a significant component of emphysema and bullae degeneration of his lungs.

Q. What about ill-defined perivascular and peribronchial passages in the left mid and lower lung zones, are those findings that can be seen with pneumonia?

A. The way they are described there sounds more like edema --

Q. Than pneumonia?

A. _- than pneumonia.

Q. And why do you say that?

A. Typically, the perihilar region is one of the areas that you see edema show up initially in patients in the intensive care unit. Ill-defined, it usually is more associated with edema than pneumonia. Pneumonias are usually fairly well defined because they take away airspace and fill it with fluid and *so* it is usually pretty easy see.

Q. Okay. Now, we know from the records and from his testimony that Dr. Furey first saw Mr. Brown on June 2. Is that your understanding as well?

A. Yes. It wasn't quite obvious to me. I had a difficult time reading the signatures in the chart, but it appears that sometime on that -- either he went on service or he was asked to specifically monitor the patient. I think he just -- it was his turn to be on service and that is when he picked up the care of the patient.

Q. How would you characterize Mr. Brown's condition as of June 2?

A. June 2. He had been very, very, very sick prior to that. I believe by June 2, he had shown some signs of improvement, but still was

very sick.

Q. In what respect had he shown some signs of improvement?

A. If I have my dates correct, he was no longer a hundred percent oxygen. Let me just make sure that is correct.

Yes. He was on 80 percent, his oxygenation was slightly better. His creatinine, the creatinine had come down so his renal failure was a little bit better. He was still very hypernatremic at the time so he was volume depleted. He had a low-grade fever, but nothing terrible.

So he previously had very, very low oxygen levels on a hundred percent oxygen and now the oxygen levels were a bit better on 80 percent. There might have been just a slight improvement.

Q. Okay. So in other words, on June 2, his oxygen level was better and it was also better with less oxygen supplementation?

A. Well, I would say it was equal, but with less oxygen supplementation.

Q. Which I take is a good sign?

A. It is a sign of moving in the right direction, but still not a very good sign. He is on 80 percent oxygen and his oxygen level is still only as high as it is in my veins, and these are in his arteries. So I would say that he is in pretty big -- still very, very sick, but at least moving from not getting sicker, he is stabilized and maybe moving --

Q. In the right direction?

A. In the right direction. Too soon to tell perhaps, but certainly moving in that direction.

Q. Knowing what you know about Mr. Brown and his history, do you believe that he had a PO2 that was probably lower than most folks?

A. Yes.

Q. What would you say his normal PO2 would have been, what range?

A. Well, he doesn't have a normal PO2 and he has an abnormal PO2, and the question is, depending on how low it is, how much damage it is going to do to his body. The body is pretty tolerant of low levels of oxygen and as long as the PO2 is at least 55 or 60, closer to 60 than

55, there actually aren't very many problems that will develop. Once it gets below that though, the pulmonary artery pressures go up and the body becomes starved of oxygen and you develop cor pulmonale and right heart failure and that is problematic and that is when we usually place patients on supplemental oxygen all the time around the clock.

Q. So if Mr. Brown's PO2 from an ABG went up from 48 to 62, you would consider that to be a good sign?

A. Well, I would say that he is no longer going to be damaged specifically from the low level of PO2 in his arterial blood; however, on the other side, it all depends on what you had to do to get the PO2 up to 62. If you have to put him on 100 percent oxygen, then he can actually be damaged from the oxygen. After 24 or 48 hours of high levels of oxygen, it will do damage to the lungs. It is called oxygen toxicity.

So, yes, the initial concern is get the PO2 from a medically emergent level to something that the body will tolerate and then figure out why it was low and get the FiO2, the amount of

oxygen necessary to give you that PO2 down to a safe level, which is typically 60 percent or 50 percent.

Q. Okay. What degree of oxygen supplementation was Mr. Brown receiving on June 3?

A. Well, you know, that is interesting. On June 2, though, he is on, it says, 80 percent not only breathing mask, with a PO2 of either 49 or 48. I can't tell because the nomenclature of how you abbreviate the blood gases varies from hospital to hospital. Typically, it is usually easily discernible, but in this circumstance they are both almost the same number and I can't tell.

But then the note on 6-3 says he is on 40 percent, and that is really kind of interesting. I mean, I don't know whether in fact he really was on 40 percent or not, but that is what it says, but he still is very, very hypoxic. So if in fact it was on 40 percent with the same PO2, that would again have shown at least an improvement in oxygenation.

Q. And if he was on 40 percent oxygen and the PO2 went up from 48 to 62, that would also be a good sign, wouldn't it?

A. Yes.

Q. A few moments ago I had asked you to describe for me Mr. Brown's condition as of June 2 and you told me that he was very, very sick prior to that time, but it had shown some signs of improvement, but was still sick.

Is there anything else you wanted to add to that characterization?

A. No, I would still say as of 6-2 he is still extremely ill, continuous.

Q. Okay. And how would you characterize his respiratory status at that time?

A. Well, based on a PO2 of 48 or 49 on 80 percent, he is still in severe respiratory failure.

Q. And how would you characterize his respiratory status on June 3?

A. Well, based on the fact that he is on 40 percent with saturations that are more acceptable, I would say that he is only in mild to moderate respiratory failure at that point.

Q. After the ABG result of June 3, which I think was timed at about 4:30 in the afternoon or so, it shows the PO2 of 62, and up until

Mr. Brown's collapse, so to speak, is there any evidence that his respiratory status worsened?

A. Not in the chart, no.

Q. From that period of time, would it be fair to state that his respiratory status remained stable or even improved slightly?

A. I would just say that from that period of time, from the time where his PO2 got up to 62 until he died, I would say you can't say whether it got better or worse or stayed the same. There is just no information.

Q. Would a pulse oxymetry be helpful?

A. Yes, to some degree. I mean, the saturation stayed the same, so one would have to say that it didn't get worse.

Q. Okay. The chest X-ray report of June 3, do you want to take a quick look at that, just for a second? June 3, do you have it?

A. Yes.

Q. Based upon that report, would it be fair to say that from a radiographic standpoint, there was further improvement in the patient's respiratory status?

A. Yes.

Q. Now, I take it you are aware of the testimony about Dr. Furey initially saying that he felt that a Greenfield filter should be placed and that he then decided to hold off on the procedure until the following morning. You are familiar with that testimony --

A. Yes.

Q. -- and what is in the records about that?

A. Yes.

Q. Okay.

A. Mostly from his testimony because there is not much in the records about that. It says that he wants to do it, but then it doesn't say why he decided not to do it.

Q. Well, there is a nursing note, isn't there, which talks about the procedure being canceled that afternoon because the patient was an NPO?

A. Yes.

Q. Do you have any criticisms of Dr. Furey's judgment on that issue?

A. Yes.

Q. All right. Is that essentially your

criticism of Dr. Furey in this case, your standard of care criticism?

A. That is the major part.

Q. Well, do you have any other criticisms that you believe -- wherein you believe Dr. Furey breached the standard of care?

A. Well, I think that he should have recognized when he first came on service that this patient had pulmonary embolism and that there was risk for additional pulmonary embolism and should have made an attempt to diagnose whether in fact he did or did not have pulmonary emboli and then made an attempt to treat this patient aggressively with a filter at that point in time. So as soon as he came on service, that should have -- that problem should have been addressed.

Q. So you believe the filter should have been placed on June 2?

A. I think the filter should have been placed virtually as soon as they made a diagnosis of pulmonary embolism and established that he still had clot in his leg, yes.

Q. Well, when did they first establish that Mr. Brown had clot in his leg?

A. Unfortunately, they didn't establish it until they did a Doppler study on 6-3.

Q. Okay. Well, do you believe that the standard of care required that test to be performed earlier?

A. Absolutely.

Q. When do you believe it should have been performed?

A. As soon as they started evaluating this patient for hypoxia.

Q. Well, that actually started well before Dr. Furey got involved in the case, isn't that true?

A. That's correct.

Q. Based upon what you have seen in the records, would you agree that Mr. Brown started receiving treatment for pulmonary embolism well before Dr. Furey's involvement in the case?

A. Yes.

Q. And so the performance of an ultrasound would have simply confirmed the diagnosis?

A. No, not just simply confirmed the diagnosis, it assessed the risk for additional embolization. That is the key thing.

Q. And how is it that it does that?

A. If the patient has already flipped clots from his leg and he still has clot in his leg that could embolize to his lung, then that needs to be prevented if the patient is not going to be tolerant of even a small additional insult to his respiratory status.

So, in fact, even had they done a study which clearly had demonstrated that the patient had pulmonary embolism, they still would have been compelled by standard of care to document whether there are potential additional sources for emboli as best as they could to insure that the patient would not be at risk for getting additional clot since Heparin really doesn't protect anybody from additional clot for a long period of time.

Q. Are there patients who have documented deep vein thrombosis who received Heparin therapy?

A. Yes. Solely for that. Absolutely. That is the treatment for that initially, and then other forms of anticoagulation develop.

Q. Okay. And in Mr. Brown's case, wouldn't it have been acceptable to treat him with Heparin for a period of time even if deep vein

thrombosis had been documented?

A. Yes. The purpose -- yes. The answer to that question is yes. The purpose of documenting DVT is to assess the risk for secondary embolization. What we know about pulmonary embolism is that typically if the patient survives the initial embolus and they do not -- are not at risk for additional emboli, they usually do fairly well.

What usually kills the patient is either the first embolism is so huge that they die immediately, or prior to their ability to resolve the clots in the lung, they get a second insult which unfortunately does not have to be a very large insult because they have already damaged a significant percentage of their lung vasculature.

Q. Can Heparin successfully treat those patients who are at risk of developing a second embolism?

A. Yes, it can. And so what your assessment is, what is the risk of the patient if he were to get another embolization. If you start out with normal lung function and then the initial embolization is, let's say, a mild or moderate

one, then further intervention besides Heparin is unnecessary because even if he were to throw another clot in the next few days before the clots in his leg start dissolving, he would be able to withstand it. And so the risk of that is less than the risk of putting in a filter.

But in a patient who has severe compromised lung status, when even a small clot could cause him to go into heart failure and respiratory failure, then even though the Heparin will eventually take care of it, you just don't -- you cannot risk it. You need to put the filter in to protect the patient immediately, and in which case you still use the Heparin to treat both DVT and the pulmonary embolism.

Q. How is it that Heparin works for patients who have pulmonary embolism?

A. Well, it is an interesting thing that we have been using Heparin for a long, long time and yet specifically no one can actually document exactly what it does.

We know what it doesn't do. It probably doesn't dissolve very much of the clot inside the lung. The lung does that itself. It

has its own innate Heparin.

It probably doesn't protect the patient for a minimum of 24 hours to potentially at least 72 hours and even longer from recurrent clot. We know that from numerous studies.

Q. When you say recurrent clot, what are you talking about?

A. Suppose there is a clot in the leg, part of which goes to the lung, but part of which still is in the leg. The Heparin does not protect you from more of the clot in the leg going up to the lung at least for several days until that clot starts dissolving, at which point in time once the clot is dissolved and absorbed, then the risk goes down. And so Heparin probably has its major effect in preventing additional clot forming and helping in the resolution of the clot that is there, but doesn't protect initially from subsequent reembolization from clot in the leg.

Q. For 24 to 72 hours?

A. A minimum of 24, at least 72 hours, but any clot in the leg that is there can still break off and Heparin can't protect you from that breaking off.

Q. Okay. So you are saying that whenever a patient has a documented deep vein thrombosis, even if they start that person immediately on Heparin, that person is still at risk for the next one to three days from developing further clots that can break off and go into the lungs?

A. Absolutely.

Q. And kill them?

A. Absolutely.

Q. How many patients over the years have you treated for pulmonary embolism?

A. Dozens.

Q. Over a hundred?

A. I don't think over a hundred, but dozens. Pretty close to a hundred.

Q. Okay. And of those patients, how many have required the use of a Greenfield filter?

A. I think I said somewhere maybe between eight and twelve.

Q. Okay. So the remainder of those patients you just treated with Heparin?

A. Absolutely.

Q. And they would have been still at a risk for developing a fatal clot?

A. No, actually, they are not at risk for developing a fatal clot because, typically, the clots get smaller and smaller and smaller as time goes by. If their lung function is sufficient, they can absorb any small fragments or even moderate size clots going up to the lung without causing death, so that is how you make the differentiation.

If I had a patient who, let's say, had a moderate pulmonary embolism and still had significant amount of clot in the leg and was a COPD, I would put in a filter. If I had a patient with the same moderate clot and had absolutely normal lung function, I probably wouldn't put in the filter because even if another clot were to be released, it wouldn't do the patient in.

And, certainly, if a patient just has DVT and no emboli to start with, then just the Heparin is usually all that is indicated.

Q. In this case, do you have an opinion as to when Mr. Brown first started developing or having clots in his lungs?

A. Well, it is very difficult to say. I think that he's probably had some embolization

early -- very early in his course. And, in fact, there may have even been clots that he had prior to his myocardial infarction. I don't know. Some of the clots that were seen in his lungs were pretty old. Other clots, obviously, could not have been old because he had an 80 or 90 percent occlusion of his pulmonary artery based on the autopsy results, and to most persons, that would be fatal. It would be extraordinarily unlikely for even a gifted athlete to be able to withstand an 80 or 90 percent occlusion of the pulmonary artery without going to permanent right heart failure and potentially dying. That is why people die from pulmonary embolism.

So some of the clots are pretty old. I am not sufficiently versed in dating clots to say whether they are months old or weeks old or just a few days old. But, obviously, some of the clots that show up in the lung that are old came from the venous blood vessels in his lung, they didn't start up here. So the fact that some of them are old up here doesn't necessarily mean that they didn't arrive in the lung recently. And that is why the pathologist would be much better to speak

about the pathological changes.

But in my opinion, I think he had a pulmonary embolism postoperatively and then additionally had a massive pulmonary embolism -- well, I shouldn't say that. He had a terminating pulmonary embolism event when he died, which may not have necessarily been even a large pulmonary embolism because he was so compromised already. Whether in fact he had a pulmonary emboli predating even his myocardial infarction, I couldn't tell you, but it is not impossible.

Q. Okay. Do you believe that he had pulmonary emboli between the time he was admitted to the hospital and his bypass surgery?

A. If he did, it would have been very, very subtle because there is no significant clinical change that would have alerted us to the fact that he did have an embolism. So it is not impossible he didn't, but if he did, it would have to be a real small one.

Q. Okay.

A. The major critical change is immediately postoperatively when not only does the PO2 fall to 40, but it doesn't respond to very,

very high levels of oxygen to try improve it, which means that some very significant change occurred there.

Q. So you would believe then that he probably had a pulmonary embolism postoperatively?

A. Yes.

Q. When in the postoperative period?

A. It is hard to say, but early on, because the initial notes postoperatively are the ones that talk about that -- he did have on the 5-28 note, the nurse talks about trying to take him off his oxygen and his saturations falling very abruptly, so that is a possibility. It is very difficult to know, but somewhere over there, I mean, he is still -- at that point in time he was on nasal cannula and still maintaining reasonable oxygen levels so it probably didn't happen as of that note, but somewhere after that when his PO2 is at 42 and does not respond to very, very large increases in oxygen. So as best as I can, somewhere between that note.

Q. Which is dated May 28?

A. And it probably was timed, but I can't see the time because there is a hole punch right

in it.

Q. Okay. I am sure Donna did that on purpose.

A. And then 5-29, just like -- which is also not timed, unfortunately. People do not understand how important it is to time your notes.

Q. Okay. By the time Dr. Furey saw this patient on June 2, obviously, Mr. Brown had pulmonary emboli already in his lungs?

A. I believe so, yes.

Q. Would you be able to characterize for me the extent of pulmonary emboli he probably had in his lungs by that point?

A. It is difficult to actually tell you what -- how large a pulmonary embolism it was because I truly believe that he had abnormal lung function to start with. The more abnormal his lung function was to start, the smaller the embolism that he needed to create this kind of scenario. And that is why knowing what his pulmonary function test was preoperatively would have been very, very important to everybody. Because if they had seen that he had a relatively normal pulmonary function test, then they would

have assumed that this must have been a massive clot then. Even if he had a very abnormal one, the end result was that he was so tenuous there that they would need to be very aggressive with the therapy because he is at very major risk for a second clot and that would do him in.

Q. So what is the answer to the question as to what degree of clot does he already have in his lungs by the time Dr. Furey first saw him?

A. By the time Furey --

Q. On June 2?

A. Now, see, that adds a second part to the equation.

Depending on his lung function to start with, he would predict how big the clot needed to be to get him that sick initially. And then during this period of time, hopefully, the body is trying to dissolve the clot to some degree, so the clot may actually be getting smaller in the lung.

And, again, if this guy has mild lung function abnormalities, then this guy had a massive pulmonary embolism. If he had severe lung disfunction, then he only had a mild to moderate pulmonary embolism. But the bottom line is,

either way, his tolerance of an additional clot is severely diminished and that is what the problem is in the case.

Q. Based upon the autopsy, does that give you any guidance as to telling us how big the actual clots were in Mr. Brown's lungs when he was first seen by Dr. Furey?

A. No. Because the data has shown -- I mean, we know what they were at the end, obviously, 80 to 90 percent occlusion, and that is what did the patient in. But clots sometimes dissolve very rapidly, sometimes dissolve very slowly, so it would be really difficult to know exactly what the size of the clot was then.

Q. Could Mr. Brown have had a clot in his pulmonary arteries occluding 70 to 80 percent of them at the time he was first seen by Dr. Furey?

A. Unlike1y.

Q. But possible?

A. It is possible, but real unlikely, the reason being that he was not showing any major, major signs of right heart failure. And this is a gentleman who has got right heart disease as evidenced already by an elevated pulmonary artery

pressure. He could not be very tolerant of a 70 to 80 percent occlusion of his pulmonary artery.

Even a very healthy person would show significant signs of reduction in cardiac output and liver failure and peripheral edema and signs of cor pulmonale associated with that kind of an event. So at that point in time, it couldn't have been 70 or 80 percent. It would have to be less than that.

Q. You just don't know how much less?

A. Right. Like I said, if it was -- if he had severely abnormal lungs, he could have only had a 10 or 15 percent occlusion of his pulmonary arteries and still be severely ill.

Q. On June 3, was there some indication of improvement in the patient's PTT times?

A. They were getting to the therapeutic range.

Q. What would you consider to be the therapeutic range?

A. Well, you know, judging from the literature, PTT between 55 and 88 seconds is what is considered optimally. One and a half to 2.3 times is what is quoted in the literature as the

optimal level for that. Initially it was a bit on the low side and they were having a difficult time titrating his Heparin appropriately.

Q. Well, if the normal range at the hospital was between 22 to 31, which is what the lab says it is, would a PTT level of 44 be at a therapeutic range?

A. Probably not. 44 would -- you know, that would have to assume that everybody had a level of about 20 and 23, and that is typically not what we see in the laboratory. Most people would say that even with the problems associated with the laboratory, you need a minimum of 55 seconds, and probably a little bit higher than that to get optimal. I saw a couple of nice articles about that that have been published.

Q. What articles? What are you reading from?

A. It is just a little handbook. I am just trying to find a reference.

Q. Okay. Who gives this out?

MS. KOLIS: That is the one the residents carry around at UH.

THE WITNESS: Is it?

MS. KOLIS: Yes.

THE WITNESS: Everyone gets this.

BY THE WITNESS:

A. This was 1995, by Dr. Hyers and Dr. Weg from the University of Michigan. That is a where the data for the appropriate level came from.

BY MR. GROEDEL:

Q. Is there a volume and page cite?

A. Volume 108, page 335.

Q. Okay. And can I see what you are looking at?

A. Yes.

Q. I wanted to look at the page you were looking at.

A. Oh, okay. There are probably other places that similar things are written down, but I know Dr. Weg and I know the University of Michigan group's work in this area and I trust them.

Q. Thank you.

A. Top page, bottom line.

Q. So it says here that the therapeutic range of 55 to 85 is roughly equivalent to a plasma Heparin concentration range of .2 to .4 by Protamine titration.

What does that mean?

A. See, the actual -- you are using the PTT indirectly to assess how much Heparin is in the blood. That is the thing that you want at a certain level is Heparin in the blood.

So in order to measure the level of Heparin in the blood, what you do is you add Heparin to it and then you titrate it against something that counteracts the Heparin effect. And if you know exactly how much of that substance you have added, you can figure out what the level of Heparin was in the blood. And the anti-Heparin medication is called Protamine. It is the mean of assessing the level.

Q. And then this statement goes on to say in this book, Handbook of Antithrombotic Theory, by Dr. Hyers, it goes on to say: Therapeutic range will vary with different APTT reagents and coagulation machines.

What does that mean?

A. Well, there is some variability and that is why the level is not 55 or 65 or 85. It is a range of 55 to 85. Basically, what they are telling you is that the vast majority of labs,

irrespective of how they run the test, if you are between 55 to 85, you will be fine.

Q. Thank you.

So would you consider a PTT level of 51 to be close to therapeutic?

A. Yes, I wouldn't quibble very much over that.

Q. So on June 3, if he has got a close to the therapeutic level -- he has a close to therapeutic level of Heparin in him?

A. Correct.

Q. And he is stable from a respiratory standpoint?

A. Relatively speaking.

Q. Is the risk of recurrent PE quite low when the PTT is greater than 1.5 times the control value?

A. As far as the literature suggests, there is no relationship, the risk of recurrent pulmonary embolism, that the risk goes down once you become therapeutic. The risk basically -- well, let me see how to best phrase it.

If you don't give Heparin, then the patient will continue to make more clot and that

increases the risk for embolization. If you give Heparin, the patient will stop making more clot and dissolve the clot that is there and that is what reduces the risk for embolization.

Q. And if you have reached a PTT level of 1.5 over control, doesn't that indicate that the risk of recurrent pulmonary embolism is quite low?

A. No, it just means that the risk of making more clot is low. It doesn't mean that the person could not release the clot that he already has in his leg.

Q. Even if that patient has been on Heparin for a number of days already?

A. Even if the patient has been on Heparin for a number of days already.

Q. Does the fact that a patient has been on Heparin for a number of days provide some additive value to the Heparin that he is receiving on that day?

A. Yes. Basically, what it is saying is that if you look at this as a building up of clot in the leg, once the patient is on Heparin, that stops. And then the normal body response would be for reabsorption. So, obviously, the longer the

person has been on anticoagulation, the less likely he is to have more clot build and the more likely he is to have less total clot in his body, and, therefore, that is what reduces his risk. And, eventually, if you have no clot, the risk would be zero.

Q. Is it your opinion then that the standard of care required that the filter be placed on June 2 as opposed to June 3?

A. Based on optimal treatment of the patient or based on the way the patient was treated at this point in time?

Q. Well, I want to know --

A. I don't know if it is a hypothetical question or -- because the only way I can answer that question is that they strongly suspected that the patient had pulmonary embolism the day after operation, which was, I think, the 31st or the 1st.

Q. Surgery was May 28.

A. Okay. The 29th, or whenever they first thought about pulmonary embolism, then what should have happened is he should have been evaluated for pulmonary embolism and DVT. And then there is no

doubt in my mind that had they done that then and there, then they would have reached the same conclusion that they reached on 6-3, which was to place a filter, but they didn't,

The point of the matter is that on 6-3 they did reach that conclusion and they opted to wait until 6-4 to put the filter in.

Q. And you believe that that was a breach of the standard?

A. Yes.

Q. If Dr. Furey saw the patient on June 2, do you believe it was a breach of the standard of care not to have the filter placed that day?

A. Yes.

Q. Would there be any -- strike that.

Is there any literature that you could cite me to which would support your contention that the standard of care required the filter to be placed on June 2?

A. I think that it is -- there is no specific literature. If you look in all the textbooks, it is pretty much one of the indications for filter placement would be pulmonary embolism with continued clot and

somebody who could not tolerate further embolization.

Q. And what textbooks are you referring to?

A. I think that virtually any pulmonary textbook or critical care textbook would have something like that. I don't think it would matter which one you are looking at.

Q. Okay.

A. It is pretty standard.

Q. If Mr. Brown needed to be sedated for the insertion of the filter, would that carry with it the risk of aspiration?

A. If he was still being fed. But if they stopped feeding him and allowed the stomach to empty, then he could be sedated very nicely.

Q. And about how long would you say it would take for the stomach to empty on its own?

A. Well, based on the way they are feeding him, which is continuous infusion, probably an hour, hour and a half, because, typically, you are only feeding -- I didn't look specifically, but I think I remember something about 80 ccs an hour, which is basically, you know, three ounces, three

and a half ounces of food. It wouldn't take very long for that to empty out of the stomach.

It is elemental, too, you know, it is not like you have to -- it is not very hard to digest, so an hour or so of stopping the feeding. And if they needed to, they could even suck the stuff out of the stomach if they needed to do a procedure right away.

Q. But sucking out the stuff from the stomach via a NG tube doesn't guarantee you have got the stomach empty, does it?

A. No, but if you wait an hour, hour and a half, it would. We know that the residuals after feeding, because that is one of the measures we make, we look at the residuals when we are trying to increase a patient's feeding, and we know that they are usually 20 MLs, 10 MLs, and that would not be a very significant risk for aspiration.

Q. Does a patient's medical status have an impact upon the degree of speed in which his stomach will empty?

A. Yes.

Q. So will it -- will gastric emptying slow down if the patient is sick?

A. Yes.

Q. How long would it have taken for Mr. Brown's stomach then to empty?

A. Well, my assumption is that if they are giving him the fluid at the rate they are giving him, then it is something every hour because that is -- otherwise if it wasn't, he would end up getting gastric distension and he would have to stop the feeding. So whatever they were giving him was emptying in an hour so that they could continue the infusion at the same rate.

Q. So what is the answer?

A. Even if it was emptying slower, it was still emptying sufficiently that there wouldn't be very much food left in an hour.

Q. Are there any risks inherent with the insertion of a filter?

A. Yes.

Q. What are they?

A. Well, obviously, you are going to have to place any large blood vessel -- you have to find a large blood vessel and cut down, so, obviously, you can always make some errors there and nick something that you don't want to nick.

Q. So the risk of bleeding?

A. Risk of bleeding would be increased. In most institutions, the catheter is actually passed from the top to the bottom so there is a small risk for arrhythmias as you go through the heart. Obviously, there is a risk of having the malpositioning of the filter.

Q. What problems can that cause?

A. It can cause problems with the kidneys if they position it more proximal to the renal vein than they are supposed to, then they can impair the emptying of blood from the kidneys and cause renal failure. But, typically, those are done in the radiologist suite and they pretty much can figure out exactly where to place it. And then the last risk is perforation of the blood vessel once it is put in place.

Q. Are any of these potential complications life threatening?

A. Typically not.

Q. Besides your criticism with respect to the timing of the placement of the filter, do you have any other standard of care criticisms of Dr. Furey in this case?

A. No.

Q. Can emboli pass through a Greenfield filter?

A. Only very, very tiny ones. The mesh work is such that -- obviously, blood flow has to go through it. So what they have done is they have optimized it so that it is very, very small and only small clots can pass through.

Q. If enough small clots pass through, can they be potentially life threatening to somebody like Mr. Brown?

A. It would be extremely unlikely because they are really tiny clots.

Q. Okay. Even with the insertion of a Greenfield filter, can clots also pass through collateral vessels and go to the lungs?

A. Yes, but it takes a while, though. It takes typically weeks before the other collateral veins will develop in sufficient size because, again, that is the whole idea behind the filter is that all the other veins coming up from the lower extremity using the collaterals are very small so if a clot is going to go through one of those, it would have to be a very small clot as well. But

over a period of time, weeks, maybe a couple of months, those bloods vessels would dilate to some degree and then they could actually allow the passage of a big clot, but that is pretty uncommon because the patient is usually on anticoagulation for a very long period of time, if not forever, after they have the filter placed.

Q Can new thrombi develop above the filter or on the prong or strut of the filter?

A They can develop on the underside of the filter. Occasionally they can get little tiny clots along the insertion points because what will happen is the blood will flow up and create eddy currents and get little tiny clots there. Those are usually not life threatening. It is real unlikely for clots to go more proximal because that is a big blood vessel with a large flow and, typically, if you are going to get clots, you need to have a reduced flow, and so that is very, very uncommon, except in patients with cancer.

Q. In your experience as a critical care specialist, have you ever delayed the placing of the filter because of comorbid medical conditions?

A. No.

Q. When you review a case with respect to standard of care issues, would you agree that it is important for you to review the case prospectively?

A. 1 don't know what you mean. Everything that I get is all retrospective.

Q. Well, when you got this case, you obviously knew that Mr. Brown died?

A. Yes, I did.

Q. You knew there was a bad result?

A. Yes.

Q. Okay. Would you agree that it is not fair to let the final outcome of the case effect how you evaluate the care rendered?

A. Oh, absolutely.

Q. Okay. Would you agree, generally speaking, that doctors constantly exercise their medical judgment when it comes to treating patients?

A. Absolutely.

Q. And sometimes doctors when exercising that medical judgment make decisions that turn out to be incorrect, true?

A. Absolutely.

Q. That has happened to you?
A. Yes.
Q. And simply because a doctor exercises judgment that in retrospect turns out to be wrong, that doesn't necessarily mean that the doctor was negligent; is that true?
A. That is correct.
Q. So, generally speaking, incorrect judgment does not necessarily mean negligence or poor care?
A. That is correct. The majority of the cases that I review, I don't find anything wrong.
Q. Was Mr. Brown at risk for cardiac arrhythmias?
A. Anybody who has had coronary bypass surgery would be at risk for cardiac arrhythmias and patients with significant chronic obstructive lung disease are at risk for cardiac arrhythmias.
Q. Would he be at risk for an arhythmia that could be fatal?
A. Sure.
Q. Is it possible that the terminal event that sent Mr. Brown over the edge, so to speak, was an arrhythmia?

A. It is unlikely. In the intensive care unit, they would be able to document that. The monitors in the unit typically record for several hours or even in our unit, 24 hours, and maintain that before they refresh it. So we have patients with them for 24 hours before a major event so they would be able to determine if that was the cause of his death.

Q. In this case, would he have been on a monitor which would have been able to depict an arrhythmia?

A. If he is in the intensive care unit, that is standard of care. In fact, that is a JCHO requirement.

Q. Okay.

A. Unless somebody specifically writes an order to DC the monitor.

Q. What is the purposes of a TED hose?

A. Excuse me?

Q. What is the purpose of TED hose for a patient in this setting?

A. Typically, we are trying to protect or prevent the formation or the development, I guess, of phlebitis in a patient who is bed ridden.

Q. Does the use of TED hose in a patient like Mr. Brown have any beneficial effect as it relates to his deep vein thrombosis and his risk of pulmonary embolism?

A. Once the clots are there, the TED hose doesn't count. It is only if before they get there, before the phlebitis, that the TED hose helps. I don't think it would help, but it certainly wouldn't help.

Q. So once his deep vein thrombosis was documented, if he had TED hose on and then they were removed, that wouldn't have any impact upon him?

A. No, I don't think that it would have any effect.

Q. You mentioned that you reviewed the pathology -- well, a pathology slide?

A. Yes.

Q. Did you review it yourself or with somebody else?

A. No, I reviewed it with a friend of mine.

Q. And who is that friend?

A. He doesn't want to be -- when I

testify, he didn't want to get involved. He has asked not to.

Q. Is he a pathologist?

A. Yes.

Q. All right. Are you going to be rendering any opinions at trial with regard to what that slide shows?

A. No.

Q. Okay.

A. I just wanted to make sure it was lung.

Q. Okay. Was it?

A. Yes.

Q. All right. The pathology report talks about a massive organizing pulmonary embolism found in the right and the left main pulmonary arteries.

Do you know what the pathologist meant when he used the term, "organizing"?

A. Yes. It means that it didn't form there recently. It either was formed somewhere else where it had been organizing, so it is not fresh like within hours, or if it formed where it was, it has been there for several days. I don't know how many days several has to be.

Q. Okay. So, to you, the term, "organizing," doesn't give you any indication as to where the clot existed for the several days that it was around?

A. Right. It could have been organizing in the leg and then broke off and then showed up in the lung and it looked the same.

Q. Okay. I know you are not a pathologist, but do you have any knowledge as to whether or not a pathologist can then look at that slide and determine whether the clot was organizing in the lungs as opposed to organizing in the deep veins?

A. Only late findings that he could do, if it was organizing in the place where it was. There are some late findings that I remember would be helpful to the pathologist,

Q. And what are those late findings?

A. There is what is called the process of epithelialization where actually the clot is sitting up against the wall, the new cells grow over it, and, actually, those are cells that are similar to the blood vessel cells and they form a smooth surface over that and that is called

epithelialization.

The other thing that happens is sometimes if the clot obstructs the whole blood vessel completely, then you will see recannulization. Obviously, that has to take place in situ. It couldn't have been somewhere else and then recannulized there. Those are the only two processes I'm aware of. There may be other ones that the pathologist knows.

Q. I take it, you would defer to a pathologist when it comes to assessing whether the clot in this case developed in the deep vein as opposed to the lung?

A. It is so unlikely that the clot developed in a lung in these kind of cases that I don't know that I would even have to question, but, yes, I would defer, if the question arose, yes, I would ask the pathologist. It is not to say I would always believe what he said because clinically, obviously, you know, we see at the bedside in real time what is happening and he only sees what has happened.

Q. Okay. The autopsy also made reference to the term, "organized remote pulmonary

embolisms". Do you have an understanding as to what is meant by the term, "organized"?

A. I think that is just the same thing, it just happened to conjugate the verb in the past tense rather than, "organizing". I think you are basically saying that some of the clots were of different ages, meaning that probably they all didn't come on the same day, they may have come on separate days.

Q. So what is your opinion then as to what caused Mr. Brown's death?

A. I think he had at least two episodes of pulmonary embolization. And the second one obstructed a sufficient amount of additional pulmonary vasculature that that caused his death.

Q. Okay. Now, the autopsy talks about this greater than 80 percent occlusion of the right and left main pulmonary arteries, and it describes it as a massive organizing pulmonary embolism.

Is it then your testimony then that this embolism, because it was identified as organizing, was at least several days old?

A. It was several days old, but not

necessarily several days old in the lung. It may have been several days old somewhere else.

Q. But it could have been several days old in the lung?

A. Unlikely, because you can't have an 80 or 90 percent occlusion of the pulmonary arteries and not have formative heart failure from that. It is just impossible. Most patients would die instantaneously with that kind of occlusion.

Q. Do you have an opinion as to when the second clot first got into Mr. Brown's lungs?

A. Well, I actually didn't say second, I said he had two episodes. I am not saying that he didn't have more than two episodes. So I am just saying that there was at least one episode postoperative and one episode that killed him. It is not impossible even that he had a small episode in between those two, but at least those two events are pretty obvious.

Q. Okay. And when you say, "episode," what is the second episode?

A. Death.

Q. At the very end?

A. Yes.

Q. And so then it is your opinion that a clot traveled from his deep veins to his lungs at that time as well?

A. Yes.

Q. Are you able to say how large the clot was at that time, the second -- that traveled from his deep veins to his lungs?

A. I would say that it didn't have to be very large based on the amount of clot that he probably had before. It could have been a small clot, still one that would have been preventable by a filter, but it did not have to be a big clot.

Q. Now, according to the autopsy, Mr. Brown had what is known as severe pulmonary congestion. What is that?

A. Well, you know, again, "congestion," is not a scientific term, it is more a descriptive term. The pathologists that I have worked with, when they say, "congestion," typically mean that the lungs are full of blood or water, and that is -- the problem is that you don't know what it is congested with. Sometimes they will even say it is congested with sputum, mucus. But most of the time, I think the majority of them will say

when they talk about congestion, they are talking about water, but occasionally they will be talking about blood.

Q. In this case, do you know what was the cause of his severe pulmonary congestion?

A. No, I don't.

Q. Is that something that would be related to his long-standing emphysema?

A. Not typically.

Q. The autopsy also noted the presence of severe pulmonary hypertension. What would have been the cause of that condition?

A. I think at least a good percentage of the pulmonary hypertension I have seen was from the COPD. We knew that, right, because a pulmonary artery catheter was inplaced and they had made measurements. So we knew that.

Q. The autopsy also made reference to a remote MI that had occurred in his right ventricle. Did you see that?

A. Yes.

Q. Does that mean he had a heart attack that effected his right ventricle before he came into the hospitalization that is at issue?

A. Again, I don't know what the pathologist means by, "remote," but my assumption was that that predated even the myocardium infarction that he had had at the beginning of this hospitalization.

Q. And it is my understanding then that he had a second MI in about the same location and that is the MI that brought him to University Hospital; is that correct?

A. Yes.

Q. And I think the autopsy also makes reference to a remote infarct in the left ventricle, too, did you see that?

A. Yes.

Q. And so that would have involved him having a heart attack in his left ventricle prior to this admission, correct?

A. Yes.

Q. So prior to the admission in issue, Mr. Brown had had two heart attacks?

A. Yes. Typically the right heart attack is not usually -- patients don't usually present with it, unless it is pretty significant, so a lot of people can have minor right heart attacks and

not know about it. The left heart attack, you know, is typically what most people will present with. So, yes, he had two remote ones.

But, again, the pathologist, you know, the pathologist could actually tell you whether it is days, weeks, and probably could actually even tell you if it is years. So they should have been able to actually give a better timing on it, but maybe they just didn't think it was important at this point in time.

Q. But based upon your reading of the report, it would be fair to assume that he had two myocardial infarctions before the third one that brought him to the hospital?

A. Yes.

Q. And then the third one was the one that brought him to UH?

A. Right.

Q. The autopsy also made reference to bullous emphysema, a right greater than left. Is that his underlying COPD?

A. Yes.

Q. And, I take it, you would agree that he probably had severe emphysema?

A. It would be extraordinary unlikely for him not to have at least moderate disease with the amount of cigarette smoking he did. Now, some people are lucky and they smoke a lot and don't get as much disease as other people, but it would be unlikely.

Q. But based on the autopsy, it looks like in this case Mr. Brown did have severe respiratory disease?

A. Yes.

Q. I saw that the lungs were described as being modeled with black pigment. What does that mean?

A. The black pigment is typically what you get from the cigarettes. It is carbonaceous residual that ends up in your lungs, associated with the tar and nicotine. I don't know, again, what they mean by -- I mean, "mild," just means that there is not a single color, so it is almost freckled with different colors.

Normally, if you see a healthy lung, they are actually quite pretty, sort of this pink, mauve-ish color, very smooth, and this obviously is not that kind of lung.

Q. Okay. The autopsy made reference to Mr. Brown having cardiomegaly. I think they said that his heart weighed 700 grams.,

How would you characterize that in terms of degree?

A. Well, a normal heart weighs somewhere between 350 and 400 grams, so that is a pretty big increase. But, again, you know, what you need to know is the specific, which chambers were enlarged and how much was hypertrophy and how much was actually dilatation, to make an assessment about what is going on.

You would expect this gentleman to have a big right heart because of his severe COPD and pulmonary hypertension. He probably had a big right atrium. If he had a myocardial infarct, then he might even have an enlarged left ventricle. It is difficult to assess how big the left atrium would be based on that. You would expect him to have significant chamber enlargement and a big heart.

Q. Okay. So suffice it to say, he had a big heart as a result of his long-standing problems even before he hit the doors at

University Hospital?

A. Yes.

Q. Do you know the difference between a transmural infarct and a subendocardial infarct?

A. Yes.

Q. What is the difference?

A. The left ventricle wall has a thickness and the blood supply goes from the outside to the inside, which means that the blood -- the wall that is furthest from the outside is at more risk because the blood supply is smaller and smaller and smaller.

The transmural infarct is one that destroys the heart tissue from wall to wall. The subendocardial is one that just destroys the tissue in this area of the heart, the furthest away from the outside.

Q. Okay. So a transmural infarction is worse than a subendocardial one?

A. Yes and no. Typically -- you know, it all depends on the scenario. It would seem logical that it is because you destroy a lot more tissue. The only thing is, is that the fact that you destroyed all that first tissue at once

doesn't necessarily mean that you aren't at risk to getting another one. And oftentimes the second one that you get associated with a subendocardial or myocardial infarction can be associated with more complication.

But on the whole, transmural infarctions tend to be the ones that are more significant, more likely to result in sudden acute patient death.

Q. Okay. How would you characterize the degree of scarring noted in Mr. Brown's heart on autopsy?

A. I wouldn't be able to comment on that. It is not something that I --

Q. Out of your field?

A. Yes. I mean, I listen to people talk about it, but I don't have enough personal experience to sort of pigeonhole it into saying that that is significant, insignificant or anything like that.

Q. Fine, fine.

What is the biventricular hypertrophy?

A. That is the two ventricles that I suggested might be enlarged. The right is one and

the left is the other.

Q. Okay. If his right ventricle was .9 centimeters thick, would that be an enlarged right ventricle?

A. Yes. Usually the right ventricle is very thin.

Q. How thin?

A. Just a couple of millimeters. And the left ventricle is usually thicker than that, .9 sonometers. I would say it is probably 50 percent larger, 50 percent more thick than it should be.

Q. Okay. How large or how thick is the left ventricle usually?

A. See, now, again, I don't -- that is not my chamber that I spend a lot of time in so I would have to look that up and go by the pathologist telling me it is enlarged.

Q. Okay.

A. And a lot of times the cardiologist will tell us this with an echo. So they will tell us if it is hypertrophy or just enlarged.

Q. Okay. How about the septum, does that have a degree of thickness?

A. Yes.

Q. Do you know what it is?
A. No, not off the top of my head.
Q. Rased upon all of the findings that we saw in his autopsy report as well as the information that you gleaned from reviewing the medical records with regard to the smoking history, do you have an opinion as to what Mr. Brown's probable life expectancy was if he had not died during the admission at University Hospital?
A. I would say, this is a 60-year-old guy --
Q. I think he was 64 at the time of admission.
MS. KOLIS: He was. He was 64 at the date of death. Actually, it was his 64th birthday. Sorry.
MR. GROEDEL: Okay.
BY THE WITNESS:
A. If his PO2 was really 40 on room air at the time that he was admitted, I would say that a five-year survival rate was probably 40 or 50 percent.
BY MR. GROEDEL:

Q. Do you have any indication that it was?

A. Well, you know, I just thought that somebody had mentioned that he had a very low saturation in the 70s when it first -- when he first showed up and they put a nasal cannula on him and it went up into the 80s.

So extrapolating from what a normal oxygen lung association is, his PO2 should have been around 45 or 46. So based on that, yes, he had very severe -- that would put him in a classification of severe COPD, which under those circumstances probably would give him a five-year survival rate of about 40, 50 percent.

Q. So it is more likely than not he wouldn't have lived five years?

A. It probably just, you know, if you want to err on the side of being slightly optimistic, it is probably equally -- equal chance that he would have made it five years.

Q. Okay.

A. But that, you know, in that, I also take into account his coronary arteries.

Q. Sure.

A. Those two will interact with one

another.

Q. Right.

A. So it is not just based on his COPD.

Q. Sure, right.

But I take it, his COPD would have been the most significant factor in the reduction of his life expectancy?

A. Yes, because, basically, you know, he only had that single left anterior descending artery region and his myocardial function was not terrible postoperatively. So I think his COPD would have been the most compelling, assuming that he didn't have any other medical diseases that were lurking behind that we didn't know about.

Q. Are you aware of any other?

A. No, but this guy has a zillion packs smoking history. Lung cancer or some other possibility, that is something that you would always be afraid of.

Q. Do you have any other opinions that you plan on discussing at trial that we have yet to cover?

A. No, I think we have covered them all. I don't think that I have discussed anything else

that I -- the case with Attorney Kolis at all, except the ones that you have pointed out here.

Q. Do you plan on coming to Cleveland for trial?

A. If so invited.

Q. Okay. And I assume if you develop any new opinions beyond those that we have discussed today, you will let Donna know and I am sure she will apprise me of them as well?

A. Yes.

Q. Thank you.

Can I take a look at your records there just to see what sort of notes you have?

A. The only markings that I think I have put are in the progress notes.

Q. Thank you.

A. And they are in blue so they stand out.

Q. Good. Thank you.

A. And you won't be able to read some of them.

Q. Okay. I will ask you to help me then.

A. Okay.

Q. Can you read for me your writing next to this RN note of 5-28-97?

A. Questioned whether there was a nuance of chest pain. At first I couldn't tell from what she was saying, whether she was using the abbreviation with the "S" over it, as patient without complaints of chest pain, which is just funny she would write it that way. So just questioning whether it was a nuance of chest pain.

And then I said that there is a femoral line, and I was wondering whether the femoral line potentially could have been a source of developing phlebitis in his leg.

Q. Did you ever come to any conclusions about that one way or the other?

A. Just, you know, it is coincidence so, you know, so I don't know. Certainly, that is where he developed his clot and the line was in the groin in that area, and the right side was clean so I thought that there was potentially a relationship between them. It is hard to say for sure.

Q. Can you read for me your handwriting for the next progress note dated May 29, 1997?

A. Okay.

Q. Just read it verbatim and then you can explain it.

A. I am trying to.

Q. Oh, okay.

A. Only discuss -- oh, let's see.

I think that looks like: Only discussed patient's sudden falling sat., without chest ray findings more likely PE. PC02 is normal. PO2 does not increase with increase in the (inaudible).

Q. So what is the significance of what you are writing there?

A. Well, basically, I'm assessing this patient's fallen PO2 and trying to go through my mind at that point in time what the differential diagnosis would be.

If this were an exacerbation of COPD, I would have expected the carbon dioxide to go up with the fall in PO2.

Q. Hyperventilating?

A. Hypoventilating.

Q. Hypoventilating.

A. Right. If this were massive pulmonary edema or ARDS or pneumonia, I would have expected

to see significant chest X-ray findings. So the fact I saw a significant reduction in PO2 without a combinatory increase in CO2 and with relatively normal chest X-ray, it wasn't normal, but it was relatively normal, I thought that pulmonary embolism needed to be a very strong consideration at that point in time.

Q. On 5-29, for 9:00 SIC unit, you have a note that says: Why accept false ox in 70s?

A. Yes. Meaning that the saturation in the 70s, that is a dangerously low saturation to maintain a patient at.

Q. And then you have a handwritten note next to the 5-29, SIC nursing note that says: Saturation falling further, and then you have an arrow FiO2 --

A. Increased FiO2.

Q. Is that "with"?

A. Yes, if it is a "C" with a line over it.

Q. Reasonable cardiac output?

A. Yes.

Q. And not in full pulmonary edema?

A. Right. So the point of the matter is,

is that this guy could not have been in severe congestive heart failure because the cardiac output was pretty reasonable at that point in time. Again, just looking for more and more bits of evidence that should have led these physicians to conclude that this guy had a significant pulmonary embolism and needed a very vigorous diagnostic workup.

Q. And then you have a note written next to the cardiology note of May 31, which says stable from heart standpoint, no pulmonary edema?

A. I figured if the cardiology guys were looking at this patient and saying that this is not -- that this is not a bad left heart -- because that is the heart that they look at. Pulmonologists look at the right side, they look at the left side.

And if they were saying that they weren't concerned, in fact, I think if I am not mistaken, in that note they said they were really going to, you know, just call us if you need us more of a type of note. And so then I thought, obviously, this guy couldn't be in full pulmonary edema at that point in time, otherwise the

cardiologist would be very excited and agitated at that point in time.

Q. And then on the 5-31, there is an SICU note where you circle COPD versus PE, and you write, finally considered diagnosis, hypoxic prior to surgery. Right?

A. Right.

Q. And then on 6-1, 6-1 SICU note, you write, doesn't notice?

A. I have to look at it.

Q. Why don't you just read it.

A. Doesn't make sense. Once the creatinine goes down, embolism may not be detectable.

The logic here was, well, I am going to wait until the creatinine gets better and then I am going to do the angiogram. Well, if he waits until the creatinine gets better, several days go by. If this is a very significant clot, the clot is not going to be diagnosable any more by any test. So that logic didn't make any sense.

I mean, either you are going to do the test now with the elevated creatinine or waiting three or four days is just putting the patient at

risk for a test that may not be useful diagnostically.

Q. Is there a risk to a patient who undergoes a test like this who has got elevated kidney values, renal values?

A. Well, if the creatinine is very high, there is risk, and even when it is not very high there is always some risk, but if the radiologist knows that you are concerned about elevated creatinine, they will use less dye and, therefore, they can really make the risk almost insignificant.

Q. And what do you have written above the note dated June 3, it is a nursing note I believe?

A. Pulmonary emboli changing here, sats are better.

Q. What does that mean?

A. Well, I was thinking that at this point in time the initial episode of pulmonary emboli were in some way, shape or form improving because his sats were getting better at lower FiO2s or the sats were staying the same at lower FiO2s, whatever it was. The oxygenation was better so, therefore, the pulmonary emboli must have been

resolving. That is another bit of information, also. The fact that you follow the progress of a disease sometimes, that also gives you a handle on what the disease was.

Q. Why don't you read for me your two notes next to the progress note of June 3, there is a radiologist note and then there is a progress note below it.

A. I don't know what I meant exactly. It says: See the clot, which was there since pre-op. And I don't know what that means.

Q. so you --

A. Oh, actually, it is not see the clot. See the note, which was see if something was there from pre-op.

Oh, yes. Talking about the catheter in his groin and I wanted to see when they put in the line there, and that was, I think -- I can't remember now if it was pre-op or inter-op.

Consider filter here, and then there is -- I am just making a note that the guy finally says consider the filter here because of consistent hypoxia.

Q. Okay.

A. So it is just an annotation.

Q. So your handwritten note next to the June 3 note --

A. Refers to the bottom note.

Q. Okay. And then you have got a handwritten notation where it says intubated here, and this is an anesthesia note at 5:00 a.m., close to when Mr. Brown died.

And then you have another note written next to the cardiothoracic surgery note timed at 5:30 a.m. And it says arrests and dies, is that what that says?

A. Yes, uh-huh.

Q. Is that it from your handwritten notes?

A. Yes, I don't think I put any notes in the nurse's section or anything like that.

MR. GROEDEL: Okay. I have no further questions. Thank you, Doctor.

THE WITNESS: You're welcome.

THE COURT REPORTER: Signature?

MS. KOLIS: He should probably read it.

FURTHER DEPONENT SAITH NOT.

AMM

STATE OF OHIO)
) SS:
COUNTY OF CUYAHOGA)

IN THE CIRCUIT COURT OF COMMON PLEAS

ESTATE OF LAWRENCE BROWN,)
Plaintiff,)
vs.) No. 346342
UNIVERSITY HOSPITALS OF CLEVELAND,)
et al.,)
Defendants.)

I hereby certify that I have read the foregoing transcript of my deposition given at the time and place aforesaid, consisting of Pages 1 to 108, inclusive, and I do again subscribe and make oath that the same is a true, correct and complete transcript of my deposition so given as aforesaid, and includes changes, if any, so made by me.

ERIC H. GLUCK, M.D.

SUBSCRIBED AND SWORN TO
before me this day
of , A.D. 199 .

Notary Public

AMM

STATE OF ILLINOIS)
)
COUNTY OF L A K E)

I, ANNETTE M. MONTALVO, a Notary Public within and for the County of Lake, State of Illinois, and a Certified Shorthand Reporter of said state, do hereby certify:

That previous to the commencement of the examination of the witness, the witness was duly sworn to testify the whole truth concerning the matters herein;

That the foregoing deposition transcript was reported stenographically by me, was thereafter reduced to typewriting under my personal direction and constitutes a true record of the testimony given and the proceedings had;

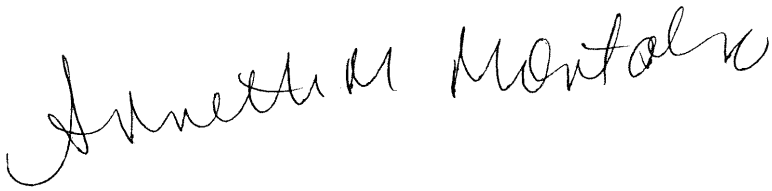
That the said deposition was taken before me at the time and place specified;

That the reading and signing by the witness of the deposition transcript was agreed upon as stated herein;

That I am not a relative or employee or attorney or counsel, nor a relative or employee of such attorney or counsel for any of the parties

hereto, nor interested directly or indirectly in the outcome of this action.

IN WITNESS WHEREOF, I do hereunto set my hand and affix my seal of office at Chicago, Illinois, this 31st day of March, 1999.



Notary Public, Lake County, Illinois.

My commission expires 1/14/03.

C.S.R. Certificate No. 84-3967.

AMM

I N D E X

'WITNESS	EXAMINATION
ERIC H. GLUCK, M.D.	
By Mr. Groedel	3

E X H I B I T S

NUMBER	MARKED FOR ID
Gluck Deposition Exhibit Nos. 1, 2 and 3	4

AMM