



# State Medical Board of Ohio

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July 12, 2000

Saravana Sivashanker, M.D.  
216 Lake Bluff Drive  
Columbus, OH 43235

Dear Doctor Sivashanker:

Please find enclosed certified copies of the Entry of Order; the Report and Recommendation of Sharon W. Murphy, Attorney Hearing Examiner, State Medical Board of Ohio; and an excerpt of draft Minutes of the State Medical Board, meeting in regular session on July 12, 2000, including motions approving and confirming the Report and Recommendation as the Findings and Order of the State Medical Board of Ohio.

Section 119.12, Ohio Revised Code, may authorize an appeal from this Order. Such an appeal may be taken to the Franklin County Court of Common Pleas only.

Such an appeal setting forth the Order appealed from and the grounds of the appeal must be commenced by the filing of a Notice of Appeal with the State Medical Board of Ohio and the Franklin County Court of Common Pleas within fifteen (15) days after the mailing of this notice and in accordance with the requirements of Section 119.12, Ohio Revised Code.

THE STATE MEDICAL BOARD OF OHIO

Anand G. Garg, M.D.  
Secretary

AGG:jam  
Enclosures

CERTIFIED MAIL RECEIPT NO. Z 281 981 395  
RETURN RECEIPT REQUESTED

cc: James H. Banks, Esq.  
CERTIFIED MAIL RECEIPT NO. Z 281 981 396  
RETURN RECEIPT REQUESTED

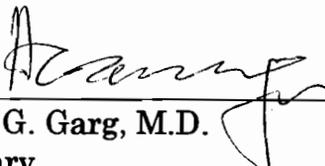
*Mailed: 7/17/00*

CERTIFICATION

I hereby certify that the attached copy of the Entry of Order of the State Medical Board of Ohio; Report and Recommendation of Sharon W. Murphy, State Medical Board Attorney Hearing Examiner; and excerpt of draft Minutes of the State Medical Board, meeting in regular session on July 12, 2000, including motions approving and confirming the Findings of Fact, Conclusions and Proposed Order of the Hearing Examiner as the Findings and Order of the State Medical Board of Ohio; constitute a true and complete copy of the Findings and Order of the State Medical Board in the Matter of Saravana Sivashanker, M.D., as it appears in the Journal of the State Medical Board of Ohio.

This certification is made by authority of the State Medical Board of Ohio and in its behalf.

(SEAL)

  
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Anand G. Garg, M.D.  
Secretary

JULY 12, 2000  
Date

**BEFORE THE STATE MEDICAL BOARD OF OHIO**

IN THE MATTER OF

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SARAVANA SIVASHANKER, M.D.

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**ENTRY OF ORDER**

This matter came on for consideration before the State Medical Board of Ohio on July 12, 2000.

Upon the Report and Recommendation of Sharon W. Murphy, State Medical Board Attorney Hearing Examiner, designated in this Matter pursuant to R.C. 4731.23, a true copy of which Report and Recommendation is attached hereto and incorporated herein, and upon the approval and confirmation by vote of the Board on the above date, the following Order is hereby entered on the Journal of the State Medical Board of Ohio for the above date.

It is hereby ORDERED that:

1. The certificate of Saravana E. Sivashanker, M.D., to practice medicine and surgery in the State of Ohio shall be **SUSPENDED** for an indefinite period of time, but not less than six months.
  
2. Within thirty days of the effective date of this Order, Dr. Sivashanker shall provide a copy of this Order by certified mail, return receipt requested, to the proper licensing authority of any state or jurisdiction in which he currently holds any professional license. Dr. Sivashanker shall also provide a copy of this Order by certified mail, return receipt requested, at the time of application to the proper licensing authority of any state in which he applies for any professional license or reinstatement of any professional license. Further, Dr. Sivashanker shall provide this Board with a copy of the return receipt as proof of notification within thirty days of receiving that return receipt.

3. Within thirty days of the effective date of this Order, Dr. Sivashanker shall provide a copy of this Order to all employers or entities with which he is under contract to provide physician services or is receiving training, and the Chief of Staff at each hospital where Dr. Sivashanker has privileges or appointments. Further, Dr. Sivashanker shall provide a copy of this Order to all employers or entities with which he contracts to provide physician services, or applies for or receives training, and the Chief of Staff at each hospital where Dr. Sivashanker applies for or obtains privileges or appointments.
4. The Board shall not consider reinstatement of Dr. Sivashanker's certificate to practice unless all of the following minimum requirements have been met:
  - a. Dr. Sivashanker shall submit an application for reinstatement, accompanied by appropriate fees.
  - b. Upon submission of his application for reinstatement, Dr. Sivashanker shall provide acceptable documentation of satisfactory completion of a course on maintaining adequate and appropriate medical records, such course to be approved in advance by the Board or its designee. Any courses taken in compliance with this provision shall be in addition to the CME requirements for relicensure for the CME acquisition period(s) in which they are completed.
  - c. Upon submission of his application for reinstatement, Dr. Sivashanker shall provide acceptable documentation of Dr. Sivashanker's enrollment in a post-graduate training program in the area of internal medicine of at least six-months duration. Such program shall be approved in advance by the Board.
  - d. In the event that Dr. Sivashanker has not been engaged in the active practice of medicine and surgery for a period in excess of two years prior to application for reinstatement, the Board may exercise its discretion under Section 4731.222, Ohio Revised Code, to require additional evidence of his fitness to resume practice.
5. Upon reinstatement, Dr. Sivashanker's certificate shall be subject to the following PROBATIONARY terms, conditions, and limitations for a period of at least five years:

- a. Dr. Sivashanker shall not request modification of the terms, conditions, or limitations of probation for at least one year after imposition of these probationary terms, conditions, and limitations.
- b. Dr. Sivashanker shall obey all federal, state, and local laws, and all rules governing the practice of medicine and surgery in Ohio.
- c. Dr. Sivashanker's certificate shall be LIMITED to participation in the post-graduate training program which was approved by the Board prior to reinstatement. The limitation shall not be terminated until Dr. Sivashanker provides the Board with acceptable documentation verifying successful completion of such program.
- d. Prior to the termination of the limitation set forth in paragraph 5.c, above, Dr. Sivashanker shall submit to the Board and receive its approval for a plan of practice in Ohio which, until otherwise determined by the Board, shall be limited to a supervised structured environment in which Dr. Sivashanker's activities will be directly supervised and overseen by a monitoring physician approved in advance by the Board.

The monitoring physician shall monitor Dr. Sivashanker and his patient charts. The chart review may be done on a random basis, with the number of charts reviewed to be determined by the Board. The monitoring physician shall provide the Board with reports on Dr. Sivashanker's progress and status and on the status of his patient charts on a quarterly basis, or as otherwise directed by the Board. All monitoring physician reports required under this paragraph must be received in the Board's offices no later than the due date for Dr. Sivashanker's quarterly declaration. It is Dr. Sivashanker's responsibility to ensure that the reports are timely submitted.

Dr. Sivashanker shall obtain the Board's prior approval for any alteration to the practice plan approved pursuant to this Order.

In the event that the approved monitoring physician becomes unable or unwilling to serve, Dr. Sivashanker shall immediately notify the Board in writing and shall make arrangements for another monitoring physician as soon as practicable.

Dr. Sivashanker shall refrain from practicing until such supervision is in place, unless otherwise determined by the Board. Dr. Sivashanker shall ensure that the previously designated monitoring physician also notifies the Board directly

of his or her inability to continue to serve and the reasons therefor.

- e. Dr. Sivashanker shall appear in person for interviews before the full Board or its designated representative within three months of the date in which probation becomes effective, at three month intervals thereafter, and upon his request for termination of the probationary period, or as otherwise requested by the Board.

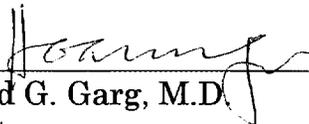
If an appearance is missed or is rescheduled for any reason, ensuing appearances shall be scheduled based on the appearance date as originally scheduled. Although the Board will normally give him written notification of scheduled appearances, it is Dr. Sivashanker's responsibility to know when personal appearances will occur. If he does not receive written notification from the Board by the end of the month in which the appearance should have occurred, Dr. Sivashanker shall immediately submit to the Board a written request to be notified of his next scheduled appearance.

- f. Dr. Sivashanker shall submit quarterly declarations under penalty of Board disciplinary action or criminal prosecution, stating whether there has been compliance with all the conditions of probation. The first quarterly declaration must be received in the Board's offices on the first day of the third month following the month in which probation becomes effective, provided that if the effective date is on or after the 16th day of the month, the first quarterly declaration must be received in the Board's offices on the first day of the fourth month following. Subsequent quarterly declarations must be received in the Board's offices on or before the first day of every third month.
- g. In the event that Dr. Sivashanker should leave Ohio for three consecutive months, or reside or practice outside the State, Dr. Sivashanker must notify the Board in writing of the dates of departure and return. Periods of time spent outside Ohio will not apply to the reduction of this probationary period, unless otherwise determined by motion of the Board in instances where the Board can be assured that the purposes of the probationary monitoring are being fulfilled.
- h. If Dr. Sivashanker violates probation in any respect, the Board, after giving him notice and the opportunity to be heard, may institute whatever disciplinary action it deems appropriate, up to and including the permanent revocation of his certificate.

6. Upon successful completion of probation, as evidenced by a written release from the Board, Dr. Sivashanker's certificate will be fully restored.

This Order shall become effective thirty days from the date of mailing of notification of approval by the Board

(SEAL)

  
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Anand G. Garg, M.D.  
Secretary

JULY 12, 2000  
Date

**REPORT AND RECOMMENDATION  
IN THE MATTER OF SARAVANA SIVASHANKER, M.D.**

The Matter of Saravana E. Sivashanker, M.D., was heard by Sharon W. Murphy, Attorney Hearing Examiner for the State Medical Board of Ohio, on September 21, 22, and 23, 1999; December 1, 2, 6, and 9, 1999; February 29, 2000; and March 1, 2000.

**INTRODUCTION**

I. Basis for Hearing

A. By letter dated March 10, 1999, the State Medical Board of Ohio [Board] notified Saravana E. Sivashanker, M.D., that it had proposed to take disciplinary action against his certificate to practice medicine and surgery in Ohio. The Board based its proposed action on Dr. Sivashanker's care and treatment of four patients. The Board set forth the following allegations:

1. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 1, a 59-year-old male with a history of mental retardation, hypogonadism, osteoporosis, diabetes, and chronic obstructive pulmonary disease [COPD].

a. On May 19, 1988, Patient 1 was admitted to the hospital for urological surgery. Throughout the hospitalization, Dr. Sivashanker ordered Lasix and nitroglycerin for Patient 1. However, chest x-rays did not show congestive heart failure [CHF]. In addition, Dr. Sivashanker noted in his discharge summary that Patient 1's myocardial band (MB) isoenzyme levels were positive; however, the laboratory reports in Patient 1's chart indicate that the MB levels were normal.

Dr. Sivashanker's final diagnosis was acute lung edema, but the medical records do not support this diagnosis. Instead, the medical records support a diagnosis of COPD decompensation, which Dr. Sivashanker failed to recognize and treat appropriately.

b. On January 5, 1990, Dr. Sivashanker admitted Patient 1 to the hospital for exacerbation of COPD and for CHF. Dr. Sivashanker treated Patient 1 for CHF, when there was no clinical evidence of CHF. Moreover, it was inappropriate for Dr. Sivashanker to direct the nurses to use intravenous Lasix for chest congestion on an as-needed basis. In addition, Dr. Sivashanker inappropriately continued to prescribe intravenous Lasix

concurrently with intravenous saline in order to correct iatrogenic hyponatremia, hypokalemia, and hypotension. Dr. Sivashanker interpreted and treated this clinical syndrome as early cardiogenic shock, when Patient 1 actually had volume depletion, pre-renal azotemia, hyponatremia, and hypokalemia from excessive use of diuretics.

2. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 2, a 75-year-old woman with a history of stroke, hypertension, and atrial fibrillation.
  - a. Patient 2 was admitted to the hospital on January 27, 1990, due to a left hemispheric stroke. An upper gastrointestinal endoscopy showed that Patient 2 had an “active” duodenal ulcer. Dr. Sivashanker prescribed Coumadin therapy for Patient 2. However, Dr. Sivashanker inappropriately failed to document in the medical records his rationale for treating Patient 2 with Coumadin, in light of the active duodenal ulcer.
  - b. On May 21, 1991, Dr. Sivashanker admitted Patient 2 to the hospital for syncope. Patient 2 had chronic atrial fibrillation. A previous out-patient Holter monitor had shown multifocal premature ventricular contractions and bradycardia. At admission, Patient 2 was taking Coumadin and baby aspirin for stroke prevention. The Coumadin and aspirin were discontinued after a digital rectal examination showed “streaks of blood on glove.” To evaluate this finding, Dr. Sivashanker performed a flexible sigmoidoscopy and a barium enema, which showed only diverticuli.

When discharged on May 31, 1991, Patient 2 was still in atrial fibrillation, but Dr. Sivashanker did not prescribe Coumadin. Dr. Sivashanker should have reinstated Coumadin therapy for Patient 2 at discharge or in the out-patient setting if follow-up stool guiacs were negative. Dr. Sivashanker’s medical records indicate that Dr. Sivashanker did not perform stool guiac testing following discharge and that Patient 2 did not resume taking Coumadin until May 1995, when Coumadin was reinstated at a neurologist’s recommendation. Patient 2 remained in atrial fibrillation and continued to be at risk of recurring stroke throughout this time period, in part due to Dr. Sivashanker’s failure to continue Coumadin therapy.
  - c. On September 11, 1996, Patient 2 presented to Dr. Sivashanker’s office with complaints of hemoptysis and vaginal bleeding. Dr. Sivashanker failed to document how he addressed these symptoms.

3. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 3, an 82-year-old woman with a history of CHF, arteriosclerotic cardiovascular disease, and organic brain syndrome.

Patient 3 was admitted to the hospital on March 15, 1990, with the diagnosis of rapid atrial fibrillation and secondary mild CHF. It was inappropriate for Dr. Sivashanker to direct the nurses to use intravenous Lasix for chest congestion on an as-needed basis.

On March 23, 1990, Dr. Sivashanker noted the presence of chest congestion and ordered 80 mg of intravenous Lasix, but continued Patient 3 on intravenous normal saline. A chest x-ray at that time showed no evidence of significant CHF. Since there was no evidence of significant CHF, the use of diuretics was inappropriate.

Dr. Sivashanker's order of March 28, 1990, to give sodium chloride intravenously until Patient 3's serum sodium level rose above 135 was inappropriate, as it subjected Patient 3 to the risk of CHF and neurological complications.

4. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 4, a 68-year-old male. From 1984 to 1992, Dr. Sivashanker treated Patient 4 for hypertension, diabetes, COPD, and heart disease.

- a. Patient 4 was admitted to the hospital on February 27, 1990, with a history of chest pain, cough, progressive shortness of breath, and an exacerbation of COPD.

On February 27, 1990, Patient 4's creatine kinase (CK) level was elevated and the myocardial band (MB) fraction was 3.6. On February 28, 1990, Patient 4's CK level remained elevated and the MB had risen to 11.3. On March 1, 1990, Patient 4's CK level remained elevated and the MB was 8.6. Dr. Sivashanker performed three electrocardiograph tests during this time period which showed no Q waves or ST segment elevation. Patient 4 complained of chest pain during this time period. Despite Patient 4's symptoms and laboratory reports, Dr. Sivashanker failed to diagnose and treat appropriately Patient 4's acute myocardial infarction.

- b. On December 12, 1990, Patient 4 was admitted to the hospital with shortness of breath and chest pain. On admission, Patient 4's CK level was twice the normal level, and the MB was 13. Patient 4 was admitted to the Intensive Care Unit and treated with a nitroglycerin patch and Cardene. Dr. Sivashanker did not request a cardiology consultation. Patient 4

## Report and Recommendation

In the Matter of Saravana E. Sivashanker, M.D.

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exhibited symptoms of an acute myocardial event. However, Dr. Sivashanker failed to treat Patient 4 appropriately, or Dr. Sivashanker failed to document his rationale for conservative treatment of Patient 4.

- c. Dr. Sivashanker's office records indicate that Dr. Sivashanker notified Patient 4 in a letter of April 9, 1992, that, until Patient 4's outstanding bill of \$1,004.96 was paid, Dr. Sivashanker could no longer see him. Dr. Sivashanker inappropriately terminated his patient-physician relationship with Patient 4.

Finally, the Board alleged that Dr. Sivashanker's treatment of Patients 1 through 4 constitutes "[a] departure from, or the failure to conform to, minimal standards of care of similar practitioners under the same or similar circumstances, whether or not actual injury to a patient is established," as that clause is used in Section 4731.22(B)(6), Ohio Revised Code." Accordingly, the Board advised Dr. Sivashanker of his right to request a hearing in this matter. (State's Exhibit 5A).

- B. On April 5, 1999, James H. Banks, Esq., submitted a written hearing request on behalf of Dr. Sivashanker. (State's Exhibit 5C).

## II. Appearances

- A. On behalf of the State of Ohio: Betty D. Montgomery, Attorney General, by Anne B. Strait, Assistant Attorney General.
- B. On behalf of the Respondent: James H. Banks, Esq.

## **EVIDENCE EXAMINED**

### I. Testimony Heard

- A. Presented by the State
  1. Saravana E. Sivashanker, M.D., as if on cross-examination
  2. William C. Miller, M.D.
- B. Presented by the Respondent
  1. Saravana E. Sivashanker, M.D.
  2. Palamalai Mahizhnan, M.D.
  3. Wayne L. Beaver, M.D.
  4. William M. Chinn, M.D.

II. Exhibits Examined

A. Presented by the State

- \* 1. State's Exhibits 1, 1A-1G: Medical records for Patient 1.
- \* 2. State's Exhibits 2, 2A-2B: Medical records for Patient 2.
- \* 3. State's Exhibits 3, 3A: Medical records for Patient 3.
- \* 4. State's Exhibits 4, 4A-4D: Medical records for Patient 4.
- 5. State's Exhibits 5A-5R: Procedural exhibits. [Note: State's Exhibit 5B is a patient key which is sealed to protect patient confidentiality.]
- 6. State's Exhibit 6: Curriculum vitae of Dr. Miller.
- 7. State's Exhibit 7: Curriculum vitae of Dr. Sivashanker.
- \* 8. State's Exhibits 8-11: Reports, summaries, and questions sent to Dr. Mahizhnan by Dr. Sivashanker while Dr. Mahizhnan was preparing to testify at hearing.

B. Presented by the Respondent

- \* 1. Respondent's Exhibits A-D: Expert reports regarding Patients 1 through 4 prepared by Dr. Sivashanker prior to hearing.
- \* 2. Respondent's Exhibits E-K: Expert reports regarding Patients 1 through 4 prepared by Dr. Miller prior to hearing.
- \* 3. Respondent's Exhibits L-R: Expert reports regarding Patients 1 through 4 prepared by Dr. Mahizhnan prior to hearing.
- \* 4. Respondent's Exhibit S: Expert report regarding Patients 1 through 3 prepared by Dr. Beaver prior to hearing.
- \* 5. Respondent's Exhibit T: Expert report regarding Patients 1 through 4 prepared by Dr. Chinn prior to hearing.
- \* 6. Respondent's Exhibits U-V: Documents created by Miller in preparation for hearing.

7. Respondent's Exhibit X: A list, compiled by Dr. Sivashanker, of continuing medical education obtained by Dr. Sivashanker.
8. Respondent's Exhibit Y: Dr. Mahizhnan's curriculum vitae.
9. Respondent's Exhibit Z: Withdrawn.
10. Respondent's Exhibit AA: Dr. Beaver's curriculum vitae.
- \* 11. Respondent's Exhibit BB: February 8, 1991, consultation report by Dr. Chinn regarding Patient 1, with cover letter to Dr. Sivashanker.

C. Post-hearing admission to the record

Board Exhibit A: Joint Notice to Hold the Hearing Record Open for Purposes of Negotiation.

Note: All exhibits marked with an asterisk [\*] have been sealed to protect patient confidentiality.

### **PROCEDURAL MATTERS**

On March 10, 2000, the parties submitted a Joint Notice to Hold the Hearing Record Open for Purposes of Negotiation. The notice advised that, pursuant to Rule 4731-13-17, Ohio Administrative Code, the parties requested that the hearing record in this matter be held open for an additional thirty days, until and including April 9, 2000, for the purposes of continued settlement negotiations. The record was held open. As of April 10, 2000, neither party had submitted any additional documentation. Accordingly, the hearing record closed at that time.

### **SUMMARY OF THE EVIDENCE**

All exhibits and transcripts of testimony, even if not specifically mentioned, were thoroughly reviewed and considered by the Attorney Hearing Examiner prior to preparing this Report and Recommendation.

#### **SARAVANA SIVASHANKER, M.D.**

Saravana E. Sivashanker, M.D., testified that he had received an MBBS degree from the Madras Medical College in Madras, India. After graduating from medical school, Dr. Sivashanker studied neurosurgery for five to six months and cardiology for an additional five to six months. Thereafter, Dr. Sivashanker completed a three year

residency in internal medicine in India. Dr. Sivashanker relocated to the United States in 1976. (Hearing Transcript [Tr.] at 22-23; State's Exhibit [St. Ex.] 7).

In 1978, Dr. Sivashanker completed one year of a general medicine residency in Yonkers, New York. Subsequently, he completed two years of an internal medicine program at the V.A. Medical Center, in Dayton, Ohio, and his final year at the Mount Carmel Medical Center, in Columbus, Ohio. Dr. Sivashanker testified that he has passed his board certification examination in internal medicine. (Tr. at 23-24; St. Ex. 7).

In 1981, Dr. Sivashanker opened a solo practice in Ostrander, Ohio. Dr. Sivashanker maintained hospital privileges at Grady Memorial Hospital in Delaware and at Union Memorial Hospital in Marysville, Ohio. Dr. Sivashanker testified that he closed his private practice in 1996 due to problems with managed care and his inability to maintain sufficient income. Dr. Sivashanker further testified that he had "given up" all of his hospital privileges. On cross-examination, however, Dr. Sivashanker admitted that his privileges at two hospitals had been revoked. (Tr. at 23-25, 27-28, 1051).

After closing his private practice, Dr. Sivashanker practiced at the Corrections Medical Center for the State Department of Rehabilitation and Corrections. Dr. Sivashanker testified that the Department of Rehabilitation and Corrections had decided not to renew his contract after the issuance of the Board's notice of opportunity for hearing. For some time thereafter, Dr. Sivashanker worked at an urgent care center in Elyria, Ohio. In December 1999, Dr. Sivashanker reported that he was not currently practicing medicine. (Tr. at 25-27, 1049-1050).

## **EXPERT WITNESSES**

### **WILLIAM C. MILLER, M.D.**

William C. Miller, M.D., testified at hearing on behalf of the State. Dr. Miller testified that he had received a medical degree from the University of Cincinnati Medical School, Cincinnati, Ohio, in 1972. He completed an internship and residency at the Good Samaritan Hospital in Cincinnati, Ohio, in 1975. Thereafter, Dr. Miller completed a two year fellowship in nephrology at the University of Cincinnati. Dr. Miller is certified by the American Board of Internal Medicine. Dr. Miller has been practicing general internal medicine in a suburb of Cincinnati since 1977. He is affiliated with Anderson Mercy Hospital. (Tr. at 191-195; St. Ex. 6).

### **PALAMALAI MAHIZHNNAN, M.D.**

Palamalai Mahizhnan, M.D., testified at hearing on behalf of Dr. Sivashanker. Dr. Mahizhnan testified that he had received a MBBS degree from the University of

Madras in Madras, India, in 1972. Thereafter, he completed a one year internship at the University of Massachusetts in Worcester, Massachusetts. In 1980, Dr. Mahizhnan completed a residency in internal medicine at Wright State University, in Dayton, Ohio. In 1982, he completed a two year hematology/oncology fellowship at the Medical College of Georgia in Augusta, Georgia. Dr. Mahizhnan testified that he is board certified in internal medicine and in hematology/oncology. He has been practicing hematology and oncology in Georgia since 1984. In a teaching capacity, Dr. Mahizhnan also treats general internal medicine patients. He is affiliated with the Medical College of Georgia and a number of hospitals in that area. (Tr. at 858-864, 1060, 1296-1297; Respondent's Exhibit [Resp. Ex.] Y).

Dr. Mahizhnan testified that he first met Dr. Sivashanker while completing his residency at Wright State University in Dayton, Ohio. They have maintained a personal friendship over the years. (Tr. at 1060-1061).

**WAYNE L. BEAVER, M.D.**

Wayne L. Beaver, M.D., testified at hearing on behalf of Dr. Sivashanker. Dr. Beaver testified that he had obtained his medical degree at Indiana University School of Medicine in 1968. He completed an internship and residency in internal medicine at the Indiana University Hospital Complex in 1973. Thereafter, Dr. Beaver completed a cardiology fellowship at the same institution. Dr. Beaver testified that, after a few years in Colorado and in the military, he completed a fellowship in invasive cardiology at Mount Carmel Medical Center in Columbus, Ohio, in 1978. Dr. Beaver has remained in private practice affiliated with Mount Carmel Medical Center since that time. Dr. Beaver is board certified in internal medicine and cardiology. He further testified that he has recently completed the newly created interventional cardiology boards. (Tr. at 1154-1155; Resp. Ex. AA).

Dr. Beaver testified that he has known Dr. Sivashanker since Dr. Sivashanker was a resident at Mount Carmel Medical Center. Dr. Beaver stated that he has testified on behalf of Dr. Sivashanker during proceedings with regard to the revocation of Dr. Sivashanker's privileges at three area hospitals. (Tr. at 1156, 1187).

**WILLIAM M. CHINN, M.D.**

William M. Chinn, M.D., testified at hearing on behalf of Dr. Sivashanker. Dr. Chinn testified that he had received a medical degree from the Ohio State University Medical School in 1969. Dr. Chinn completed an internal medicine internship at the State University of Iowa Hospitals and one year of an internal medicine residency at the Ohio State University Hospitals. Dr. Chinn served two years in the military before completing his residency in internal medicine at Mount Carmel Medical Center in 1971. Thereafter, Dr. Chinn completed two years of additional training in pulmonary diseases at the Ohio

State University Hospitals and at the State University of Iowa Hospitals. Dr. Chinn has maintained a private practice in pulmonary medicine in Columbus, primarily at Mount Carmel Medical Center, since 1976. (Tr. at 1375-1376).

Dr. Chinn testified that his relationship with Dr. Sivashanker is strictly professional. Dr. Chinn further stated that he has testified on Dr. Sivashanker's behalf at informal hearings regarding the revocation of Dr. Sivashanker's privileges at two local hospitals. (Tr. at 1376-1377, 1440-1441).

### **PATIENT 1**

Patient 1 first saw Dr. Sivashanker in October 1986. Patient 1, a 59 year old male, presented with a history of mental retardation, juvenile diabetes, congestive heart failure [CHF] with low cardiac output, left bundle branch block, mitral regurgitation with stenosis, rheumatic fever, scarlet fever, chronic obstructive pulmonary disease [COPD], hiatal hernia, esophagitis, seizure disorder, hypogonadism, osteoporosis, and dysarthria. In addition, Patient 1 had smoked more than a pack of cigarettes a day for the past thirty years. (St. Ex. 1 at 2).

Patient 1 had an echocardiogram and Doppler studies performed by C.G. Reddy, M.D., a cardiologist, in November 1986. The tests revealed a calcified mitral valve with mitral regurgitation, diminished cardiac wall motion, an enlarged left atrium, aortic sclerosis with thickening, and probable aortic stenosis. On March 18, 1987, Dr. Sivashanker performed an electrocardiogram [EKG]. In hand-written notes, Dr. Sivashanker interpreted the EKG as revealing "subendocardial infarct, inferolateral wall; bi-atrial enlargement; and intraventricular conduction delay." (St. Ex. 1 at 4a, 31-32, 59).

On March 24, 1987, Dr. Reddy advised Dr. Sivashanker that Patient 1 had had a cardiac catheterization which had revealed no significant coronary artery disease nor any significant mitral or aortic valve blood flow abnormalities. Dr. Reddy further reported that Patient 1's overall left ventricular function appeared to be normal. Finally, Dr. Reddy reported that Patient 1's "extreme incapacitation [was] due to far advanced COPD and its attendant consequences." The cardiac catheterization also demonstrated chronic hypoxemia with a pO<sub>2</sub> [partial pressure of oxygen] of 60 and a pCO<sub>2</sub> [partial pressure of carbon dioxide] of 47. (St. Ex. 1 at 10-12).

Patient 1 suffered four anterior wall infarctions between June and August 1987. On one occasion, there was additional evidence of inferolateral wall ischemia. Patient 1 was hospitalized on all four occasions under the care of Dr. Sivashanker. There is no indication that Dr. Sivashanker obtained a cardiology consultation during any of the hospitalizations. (St. Ex. 1 at 68, 169-174).

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In April 1988, Patient 1 was hospitalized with complaints of vague chest discomfort and shortness of breath. An EKG showed T wave inversion in the inferolateral leads “suggestive of acute ischemia.” The CK was normal. (St. Ex. 1 at 176).

### **PATIENT 1’S MAY 19, 1988, HOSPITAL ADMISSION**

#### *Basis for the Admission; Hospital Course*

1. On May 19, 1988, Patient 1 was admitted to Grady Memorial Hospital for urological surgery, a TURP. Patient 1 was admitted by the surgeon, Antonio Mortera, M.D., and the TURP was performed that day. Dr. Sivashanker testified that he had been on vacation when Patient 1 was admitted. Nevertheless, Dr. Sivashanker countersigned Dr. Mortera’s admission orders. (Tr. at 29-30; St. Ex. 1B at 4-7, 11-12, 72).

Upon admission, Dr. Mortera documented that Patient 1’s lungs were clear without rales. Patient 1’s cardiac rhythm was regular, with a pan-systolic murmur. A chest x-ray revealed no evidence of acute cardiopulmonary disease. (St. Ex. 1B at 5, 58).

Surgery was performed at approximately 8:00 a.m. Later that afternoon, Patient 1 suddenly became restless, diaphoretic, and short of breath. Patient 1 denied chest pain. Dr. Mortera obtained a consultation with an internist, Dr. French. Dr. French noted that a chest x-ray had demonstrated acute pulmonary edema. Arterial blood gases revealed a pH of 7.39, a pCO<sub>2</sub> of 48.2, a pO<sub>2</sub> of 59.5, and an oxygen saturation of 90.4. An EKG revealed no acute changes. Finally, Patient 1’s CK was within normal limits, but his LDH was 610 [normal range 94-172]. The laboratory noted, however, that the specimen had been moderately hemolyzed. Dr. French listed his impressions as acute pulmonary edema and rule out myocardial infarction. (St. Ex. 1B at 10, 53, 55).

Patient 1 was admitted to a telemetry unit, based on pre-printed standing orders. The orders are dated by a nurse on May 19, 1988, and Dr. Sivashanker’s is the only physician signature on the orders. Dr. French recorded hand written orders, which included an order for Lasix 40 mg intravenously [IV] now, and 40 mg IV every eight hours. Dr. French’s orders also included an order for Transderm Nitro and “No Code Blue.” Patient 1’s total intake that day, both during and after surgery, was 1575 c.c., plus 800 c.c. of Sorbitol. Patient 1’s output was 3100 c.c. (St. Ex. 1B at 74-76, 83, 100, 101).

On May 20, 1988, the progress notes indicate that Patient 1 was less short of breath and that his lungs were clear. Lasix was decreased to 40 mg IV daily. Later that afternoon, Patient 1’s temperature was 102.4°. Blood cultures were obtained, and his white cell count was 19.5 [normal range 7.8 ± 3]. His cardiac enzymes were

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“essentially normal.” Patient 1’s total intake was 1890 c.c. His output was 4025 c.c. (St. Ex. 1B at 9, 32, 55, 77, 78, 83).

On May 21, Dr. French changed the Lasix from IV to PO [by mouth]. (St. Ex. 1B at 78).

Dr. Sivashanker wrote his first order and progress note on May 21, 1988, at approximately 5:30 p.m. Dr. Sivashanker wrote that Patient 1 was short of breath, and that examination of his chest had revealed bilateral rhonchi and scattered rales. Patient 1’s cardiac enzymes were “essentially normal.” His total intake was 2850 c.c. and his output was 2000 c.c. (St. Ex. 1B at 20, 56, 83).

On May 22, 1988, Dr. Sivashanker wrote that Patient 1’s blood pressure had dropped to 70/60, and that Patient 1 had bilateral rales and rhonchi. Dr. Sivashanker further noted that Patient 1 was lethargic and that his temperature had dropped from 104° to 97°. Dr. Sivashanker acknowledged the possibility of septic shock, and ordered 0.9% sodium chloride IV at 100 c.c. per hour if Patient 1’s systolic pressure should fall to less than ninety. Moreover, Dr. Sivashanker ordered that, if Patient 1’s lungs were to fill up with rales and/or rhonchi, the nurse should give Lasix 20 mg IVP every eight hours. (St. Ex. 1B at 21, 79). Less than two hours later, however, Dr. Sivashanker wrote that Patient 1 had taken a shower, his blood pressure had returned to 110/60, and he was sitting in a wheel chair. A chest x-ray revealed no evidence of acute cardiopulmonary disease. Dr. Sivashanker concluded that Patient 1’s condition had returned to normal. (St. Ex. 1B at 21, 59).

Patient 1’s total intake on May 22 was 1740 c.c. His output was 1875 c.c. After May 22, the hospital staff no longer documented Patient 1’s total intake and output. (St. Ex. 1B at 83).

On May 23, 1988, the care of Patient 1 was transferred to Dr. Sivashanker’s service. In his progress notes, Dr. Sivashanker stated that Patient 1 had had a positive MB suggesting a possible subendocardial myocardial infarction. The laboratory reports, however, do not show an MB as being performed. Moreover, the CK was below normal at 51 [normal range 61-224]. An EKG performed that day revealed a possible lateral wall infarct, old, non-specific ST-T changes, and bi-atrial overload (St. Ex. 1B at 22, 68).

On May 24, 1988, Dr. Sivashanker noted that Patient 1 was doing better, that he had no complaints of chest pain, and that his lungs were clear. Patient 1 was discharged that day. Dr. Sivashanker’s discharge summary included the following diagnoses: acute lung edema, conduction disorder, mixed acid-base balance, and coronary atherosclerosis. Moreover, although Dr. Sivashanker did not list a diagnosis for a cardiac event, the discharge summary noted that Patient 1’s “MB was positive,

suggesting he [had] developed an acute subendocardial MI.” (St. Ex. 1B at 6-7, 22). [Note, however, the hospital record does not contain any indication that Patient 1 had a positive MB at any time during this hospitalization, other than Dr. Sivashanker’s notation in the progress notes and in the discharge summary.]

*Regarding the Board’s allegations that: (1) throughout the hospitalization, Dr. Sivashanker had ordered Lasix and nitroglycerin for Patient 1, despite the fact that chest x-rays did not show CHF; (2) Dr. Sivashanker’s final diagnosis had been acute lung edema, despite the fact that the medical records do not support this diagnosis; and (3) the medical records support a diagnosis of COPD decompensation, which Dr. Sivashanker failed to recognize and treat appropriately.*

***Dr. Miller’s Testimony***

2. Dr. Miller criticized Dr. Sivashanker’s care and treatment of Patient 1 in that Dr. Sivashanker had diagnosed Patient 1 as having CHF and/or acute lung edema despite the fact that the record revealed no evidence of heart failure. Dr. Miller testified that the correct diagnosis should have been decompensation of chronic lung disease or COPD. (Tr. at 206, 317). Dr. Miller defined CHF as follows:

a condition which exists when, because the heart is weakened, it cannot pump the blood \* \* \* away from the lungs and into the rest of the body. That fluid, blood, backs up in the lung. This fluid oozes into the air sacs or alveoli in the lung, and creates a shortness of breath condition.

(Tr. at 210). Dr. Miller defined COPD as follows:

a condition where the lung tissue is scarred from, in this situation, smoking. There may be concomitant bronchitis or mucous production in the lung, which can accumulate and also spasm the bronchial tubes, which results in shortness of breath as well.

(Tr. at 210). Dr. Miller testified that CHF and COPD can co-exist, but that they are separate entities. He further testified that symptoms of the two conditions can be similar, and cited rales as an example. Dr. Miller specified that rales are not necessarily indicative of acute CHF. (Tr. at 210-211).

3. Dr. Miller testified that, in forming his conclusion that Patient 1 had had COPD decompensation rather than CHF, he had relied on the chest x-ray taken post-operatively. Dr. Miller testified that the x-ray had not revealed an enlarged heart or any other evidence of CHF. Dr. Miller testified that some of the changes you would expect to see on a chest x-ray with a patient in acute CHF would include either interstitial

edema or evidence of increased vascular prominence. Dr. Miller testified that these were also not present in Patient 1's chest x-ray. (Tr. at 207-212, 374-376, 396).

Dr. Miller acknowledged that Patient 1 had demonstrated signs of shortness of breath postoperatively, and that a cardiologist had detected chest rales and acute pulmonary edema. Dr. Miller further acknowledged that if a person has CHF which is compensated and controlled, the chest x-ray may appear normal. Nevertheless, Dr. Miller stated that, in Patient 1's case, the alleged changes were acute and should have been reflected in the chest x-ray. (Tr. at 207-212, 374-376, 396).

4. Dr. Miller further testified that, in forming his conclusion that Patient 1 had had COPD decompensation rather than CHF, he been influenced by his belief that Patient 1 had had no history of cardiac problems. In forming that belief, Dr. Miller testified that he had relied on the 1987 heart catheterization which had been normal. Dr. Miller testified that the cardiac catheterization is the "gold standard" for evaluating CHF. (Tr. at 206-209, 212-214).

On cross examination, however, Dr. Miller acknowledged that Dr. Sivashanker's office records contain substantial evidence of previous cardiac problems suffered by Patient 1. This evidence includes a November 1986 hospital discharge summary indicating that Patient 1 had been hospitalized with diagnoses including decompensation of CHF. Moreover, discharge summaries from 1987 indicated that Patient 1 had suffered multiple acute inferior myocardial infarctions. (Tr. at 206, 209, 410, 413).

Nevertheless, Dr. Miller testified that he had not considered any of the information contained in these discharge summaries when he had determined that Patient 1 had not had a history of CHF. Dr. Miller explained that he had not considered the information because he had not had the complete hospital records. Furthermore, Dr. Miller testified that he had discounted the information contained in the discharge summaries because it was not consistent with the cardiac catheterization performed in 1987. Finally, Dr. Miller testified that those records were not really relevant to the patient's presentation in 1990. (Tr. at 414, 417).

5. Dr. Miller acknowledged that the Lasix and Nitroglycerin given to Patient 1 prior to May 22 had been ordered by Dr. French and Dr. Mortera. (Tr. at 400).
6. Dr. Miller agreed that Dr. Sivashanker could not be held responsible for care provided to Patient 1 prior to Dr. Sivashanker's return on May 21 or 22. Nevertheless, once he returned, Dr. Sivashanker had been responsible to evaluate what had happened to Patient 1 and to form an independent conclusion. Dr. Miller concluded that Dr. Sivashanker had erred in agreeing that Patient 1 had had an exacerbation of CHF rather than decompensated COPD, despite the fact that the diagnosis had been made in

Dr. Sivashanker's absence. Dr. Miller testified that Dr. Sivashanker should have corrected the diagnosis made by other physicians. (Tr. at 222, 317).

7. Dr. Miller testified that the only complaint he had regarding Dr. Sivashanker's care was the fact that Dr. Sivashanker had written in his discharge summary that Patient 1 had had acute lung edema, which was a misdiagnosis. (Tr. at 386, 391, 393).

Nevertheless, Dr. Miller later testified that Dr. Sivashanker's care of Patient 1 on May 22, 23, and 24, 1988, had been inappropriate because Dr. Sivashanker had ordered Lasix on those days, when Patient 1 had had no evidence of acute CHF and the lung findings had been related to lung disease rather than cardiac disease. (Tr. at 399-400).

***Dr. Sivashanker's Testimony***

8. Dr. Sivashanker testified that any error in diagnosing CHF and COPD should have been addressed to the physicians taking care of Patient 1 at that time, not to Dr. Sivashanker. Dr. Sivashanker stated that the CHF had been diagnosed by various physicians prior to Dr. Sivashanker's taking over care of Patient 1 on May 21, 1988. (Tr. at 37).
9. Dr. Sivashanker testified that, when Patient 1 had undergone the urological procedure, the surgeon had been unaware of Patient 1's history of CHF. Dr. Sivashanker further testified that, during surgery, the surgeon and the anesthesiologist had administered excessive amounts of IV fluids, which had resulted in acute respiratory difficulty for Patient 1. Because Dr. Sivashanker was out of town at that time, the surgeon had consulted an internist, Dr. French. Dr. French diagnosed acute pulmonary edema. Dr. Sivashanker testified that Dr. French had ordered Lasix in "perhaps excessive amounts," which had resulted in subsequent marked hypovolemia and acute hypotension. (Tr. at 35-36, 778-779).

Nevertheless, Dr. Sivashanker stated that Lasix had been the appropriate treatment because Patient 1 had been in pulmonary edema. Dr. Sivashanker testified that he agreed with the conclusions of the various physicians who had diagnosed CHF and/or pulmonary edema based on a combination of factors. These factors included Patient 1's rhonchi and rales and his history of mitral valve disease, myocardial infarction and CHF. Dr. Sivashanker further testified that symptoms consistent with CHF could be found documented throughout the hospital record. Dr. Sivashanker cited references to dyspnea, tachypnea, wheezes, and congestion. (Tr. at 41-42, 48-50, 778, 825-832).

10. Dr. Sivashanker testified that he agreed with Dr. French's diagnosis of pulmonary edema, despite a chest x-ray report which showed "no evidence of acute

cardiopulmonary disease.” Dr. Sivashanker testified that the “[c]hest x-ray is not the way to diagnose acute decompensation of a chronic heart failure [because] it takes between 12 to 24 hours of time lag before chest x-ray would show classic pulmonary edema findings.” Dr. Sivashanker further stated that the radiologist reviewing the x-ray has not seen the patient and, thus, errors can be made. Dr. Sivashanker stated that he tends to trust the clinician who sees the patient. (Tr. at 46-47, 779-781, 814-818).

11. Dr. Sivashanker testified that he had not ordered nitroglycerin and Lasix for Patient 1. He stated that those medications had been ordered by Dr. French. (Tr. at 809-811, 814).
12. Dr. Sivashanker testified that Patient 1 had remained in CHF despite the massive doses of Lasix given by Dr. French. Dr. Sivashanker testified that he, himself, had ordered Lasix 20 mg IV on May 22, because, in spite of low blood pressure, Patient 1 was going into heart failure on and off. Dr. Sivashanker stated that he had made the diagnosis based on rales heard in Patient 1’s lungs. (Tr. at 42-43, 50).
13. Dr. Sivashanker further testified that it was possible to conclude that Patient 1 had not had an exacerbation of COPD because Patient 1’s pCO<sub>2</sub> was not elevated, which is a classic finding in COPD. (Tr. at 835).

***Dr. Mahizhnan’s Testimony***

14. Dr. Mahizhnan testified that the surgeon and the consulting cardiologist had been correct in diagnosing Patient 1 with CHF. Moreover, Dr. Mahizhnan testified that the blood gases and other information indicate that Patient 1 had not suffered a decompensation of COPD at any time during the hospitalization. (Tr. at 1971-1972).
15. Dr. Mahizhnan agreed with Dr. Sivashanker’s testimony that CHF or pulmonary edema may be present although it is not indicated on chest x-ray. (Tr. at 1086-1090).

***Dr. Beaver’s Testimony***

16. Dr. Beaver testified that Patient 1’s case had been a very complicated case. Dr. Beaver testified that Patient 1 had had a history of CHF, and the excessive fluids he received during surgery would have led to an exacerbation of the CHF. Moreover, Patient 1 demonstrated symptoms consistent with CHF. Therefore, CHF was the most likely diagnosis. Dr. Beaver concluded that Dr. Sivashanker’s care and treatment of Patient 1 had been appropriate. (Tr. at 1157-1158, 1160, 1200-1208, 1265-1271).
17. Dr. Beaver testified that early CHF is diagnosed based on dyspnea, orthopnea, paroxysmal nocturnal dyspnea, or waking up from sleep because the patient can not breathe. Dr. Beaver stated that the chest ray is helpful, but not always reliable, because a chest x-ray may not reveal changes in the lungs for many hours. Finally, Dr. Beaver

stated that rhonchi and rales are not necessarily symptoms of CHF. (Tr. at 1158-1159, 1166-1167, 1208-1211).

18. Dr. Beaver testified that a patient may demonstrate clinical evidence of CHF despite a negative chest x-ray. He stated that it is more likely in persons who have COPD. Dr. Beaver explained that persons who have chronic lung diseases may not manifest all of the findings of CHF, particularly the fullness of the lungs, the venous redistribution findings, pleural effusions, and an enlarged heart. Dr. Beaver stated that all of these findings may be modified by lung disease. (Tr. at 1161-1162).

*Dr. Chinn's Testimony*

19. Dr. Chinn testified that he had consulted in the care of Patient 1 in February 1991, and that Patient 1's history at that time was similar to that documented in the February 1988 hospital records. Dr. Chinn had diagnosed Patient 1 as having COPD with chronic emphysema secondary to cigarette smoking, and multiple other problems. Dr. Chinn testified that he had also noted an S4 gallop, which is consistent with CHF. (Tr. at 1380-1382; Resp. Ex. BB).
20. Dr. Chinn testified that, during the February 1988 hospitalization, Patient 1 had demonstrated CHF. Nevertheless, if he had had any pulmonary edema, it was only mild pulmonary edema. Moreover, Dr. Chinn testified that Patient 1 had presented with chronic compensated COPD. (Tr. at 1387-1388).

Dr. Chinn explained that Patient 1's chest x-ray did not support a conclusion that Patient 1 had been suffering CHF. When asked if a chest x-ray would demonstrate pulmonary edema if in fact the patient was experiencing pulmonary edema, Dr. Chinn testified that pulmonary edema could not be diagnosed unless findings were confirmed by x-ray. He stated that if it is not evident on x-ray, it must be very mild pulmonary edema. Dr. Chinn did state, however, that with a patient who has COPD, the chest x-ray is much more difficult to interpret than in a person with normal lungs. (Tr. at 1385-1387).

21. Dr. Chinn testified that, in concluding that Patient 1 had been suffering CHF, he had considered Dr. Reddy's 1987 cardiac catheterization report and the 1986 echocardiogram. Dr. Chinn testified that, in the cardiac catheterization report, Dr. Reddy had documented pulmonary hypertension which would indicate that there was increased pressure in the artery conducting blood from the right side of the heart to the lungs. Dr. Chinn testified that the pulmonary hypertension would have resulted from Patient 1's chronic emphysema. Nevertheless, the effect would be to cause increased stress to the right side of the heart and eventual heart failure.

Dr. Chinn further noted that Dr. Reddy had documented a diminished cardiac output which may have been an indication of left ventricular failure. (Tr. at 1470-1474).

Finally, Dr. Chinn further noted that Dr. Reddy had documented diminished cardiac wall motion. Dr. Chinn testified that such a finding is most likely a reflection of CHF. Dr. Chinn testified that if a patient is diagnosed with CHF, the condition will likely worsen over time, and a physician should always consider the possibility of worsening CHF. (Tr. at 1484-1487).

22. Dr. Chinn concluded that Dr. Sivashanker's had treated Patient 1 appropriately. He stated that the postoperative respiratory insufficiency had been addressed. Moreover the patient had responded to treatment and had left the hospital with virtually clear lungs. (Tr. at 1387-1388).

*Regarding the Board's allegation that Dr. Sivashanker had noted in the discharge summary that Patient 1's myocardial band (MB) isoenzyme levels were positive; however, the laboratory reports in the patient's chart indicated that the MB levels were normal.*

#### ***Dr. Miller's Testimony***

23. Dr. Miller testified that Dr. Sivashanker's care and treatment of Patient 1 had fallen below the minimal standard of care because Dr. Sivashanker had misdiagnosed Patient 1's clinical condition by claiming that the MB had been elevated. Dr. Miller testified that the MB is a measurement used to determine whether a patient has had a heart attack. Dr. Miller noted that one lab report had indicated that Patient 1's MB had been elevated. Nevertheless, the laboratory staff had concluded that the elevation had resulted from hemolysis of the blood specimen rather than from an accurate reflection of Patient 1's MB. Moreover, Dr. Miller noted that the absolute CK had been within normal limits. Therefore, Dr. Miller concluded that the MB had been normal. (Tr. at 224-225).

#### ***Dr. Sivashanker's Testimony***

24. Dr. Sivashanker acknowledged that, in dictating the discharge summary for the May 19, 1988, hospitalization, he had stated that Patient 1's MB had been positive and suggested that Patient 1 had had an acute subendocardial myocardial infarction. Dr. Sivashanker stated that, because Patient 1 had had a previous myocardial infarction, Dr. Sivashanker had not known if the MB truly had been elevated. Dr. Sivashanker explained that, because there had been "a blip" on the first test, he had been obligated to consider the possibility of cardiac damage. Dr. Sivashanker further testified that the normal result upon repeat of the test did not "matter." (Tr. at 56-60, 822-823).

Dr. Sivashanker concluded that Patient 1 may have had a myocardial infarction, but Dr. Sivashanker had not felt “strongly” that he had. Therefore, Dr. Sivashanker had not added myocardial infarction to the diagnoses. Nevertheless, Dr. Sivashanker testified that he had mentioned the elevated MB in the discharge summary simply as “an observation.” Dr. Sivashanker testified that he had not documented his reasoning because Patient 1 had been such a complicated patient. (Tr. at 56-60, 822-823).

***Dr. Mahizhnan’s Testimony***

- 25 Dr. Mahizhnan testified that Patient 1 had not had a myocardial infarction during this admission. (Tr. at 1305-1308).

**PATIENT 1’S JANUARY 5, 1990, HOSPITAL ADMISSION**

*Basis for the Admission; Hospital Course*

26. On January 5, 1990, Patient 1 was admitted to Grady Memorial Hospital with a diagnosis of “Acute Exacerbation of COPD/CHF.” In the emergency department [ER], the examination revealed gross inspiratory rhonchi in all lung fields and mild shortness of breath. Patient 1 also demonstrated jugular vein distention and non-pitting pedal edema. The chest x-ray revealed no acute changes. Arterial blood gases on room air demonstrated a pH of 7.42, a pO<sub>2</sub> of 62.4, and a pCO<sub>2</sub> of 41.6. Oxygen saturation was 92.2. Dr. Sivashanker wrote an admission note, and indicated that Patient 1’s sister had requested “No Code Blue.” (St. Ex. 1E at 19, 25, 88, 92).

Upon admission, Dr. Sivashanker ordered Lasix 40 mg IV, twice daily; Capoten 12.5 mg, twice daily; a NitroDur patch 5 mg, daily; and numerous other medications. He also ordered a fluid restriction of 1200 c.c. per day and a consultation with a cardiologist, Ronald Frazier, M.D. (St. Ex. 1E at 16, 101).

Upon consultation, Dr. Frazier noted that “despite aggressive and appropriate medical treatment, [Patient 1] continued to have recurrent [chest pain] and CHF.” Dr. Frazier noted his impressions, which included the following:

- ASHD [arteriosclerotic heart disease], status post myocardial infarction;
- Left bundle branch block;
- CHF, secondary to ASHD and dilated cardiomyopathy of unknown etiology; and
- Diabetes mellitus.

(St. Ex. 1E at 16). Dr. Frazier noted that Patient 1's prognosis was poor due to his "end stage CHF." He recommended the use of Capoten, Lanoxin, and Lasix. He also recommended the use of nitrates if tolerated by Patient 1. (St. Ex. 1E at 16).

On January 6, 1990, Dr. Sivashanker wrote that Patient 1 had been coughing up a lot of sputum and that he was short of breath at rest. On chest examination, Dr. Sivashanker documented scattered rhonchi and rales. Dr. Sivashanker wrote that he agreed with Dr. Frazier's consult. At that time, Patient 1's sodium was 137 [normal range 137-150] and his potassium was 3.8 [normal range 3.5-5.3]. His BUN was 19 [normal range 7-22]; no creatinine level was calculated. (St. Ex. 1E at 24, 67, 68).

Dr. Sivashanker changed the Lasix to 20 mg IV four times a day PRN [as needed]. He also changed the Capoten to 25 mg twice daily so long as Patient 1's systolic pressure was greater than 90. Patient 1's total fluid intake that day was 1350 c.c., and his output was 3540 c.c. (St. Ex. 1E at 102, 114). Thereafter, intake and output was not consistently recorded.

On January 8, 1990, Dr. Sivashanker discontinued the fluid restriction. On January 9, Dr. Sivashanker documented that the nurses had recorded increased chest rales. Dr. Sivashanker noted that Patient 1 had coughed up a lot of sputum and that his lungs had cleared. On January 10, Dr. Sivashanker documented bilateral chest rales, anteriorly. He also noted that Patient 1 was short of breath. Dr. Sivashanker diagnosed decompensated CHF. (St. Ex. 1E at 25, 26, 103).

On January 11, 1990, Dr. Sivashanker wrote rales that cleared after coughing, shortness of breath at rest, and a large amount of sputum production. Dr. Sivashanker ordered Lasix 20 mg IV now and every four hours PRN. On January 12, Dr. Sivashanker discontinued the Capoten and ordered Capozide 25/15 three times daily. (St. Ex. 1E at 26, 104, 105).

On January 14, 1990, Dr. Sivashanker wrote that Patient 1 had been experiencing acute shortness of breath, bilateral chest rales, a blood pressure of 86/50, and an S3 gallop. Dr. Sivashanker diagnosed acute pulmonary edema, and ordered Lasix 20 mg IV NOW and twice daily so long as Patient 1's systolic pressure remained at or above 85. Patient 1's total fluid intake that day was 1650 and his output was 2200. (St. Ex. 1E at 27, 106, 115).

On January 15, 1990, Dr. Sivashanker noted that Patient remained short of breath at rest and was "terminally ill." Dr. Sivashanker wrote that Patient 1's "physical status, including advanced COPD and CHF with mitral regurgitation, makes it almost very high risk to do any cardiac surgery." Dr. Sivashanker noted that Patient 1's lungs revealed "scattered rhonchi." A chest x-ray demonstrated "no evidence of overt cardiac decompensation." At that time, Patient 1's sodium level was 127, his

potassium level was 3.3, and his chloride level was 80 [normal range 99-113]. In addition, Patient 1's BUN was 29, and no creatinine level was calculated. Patient 1's total fluid intake that day was 1380; his output was 1405. There is no indication that Patient 1 was experiencing diarrhea. (St. Ex. 1E at 28, 71, 93, 116, 170-171).

On January 16, 1990, Dr. Sivashanker wrote that Patient 1's chest was clear. The nurses' notes indicate that Patient 1 had had one solid stool. (St. Ex. 1E at 28, 172).

On January 17, 1990, Dr. Sivashanker noted that Patient 1's chest revealed bilateral rhonchi. A chest x-ray revealed "no free fluid or evidence of overt decompensation." At that time, Patient 1's sodium level was 120, his potassium level was 2.7, and his chloride level was 71. In addition, Patient 1's BUN was 41, and his creatinine level was 1.9 [normal range 0.5-1.2]. Dr. Sivashanker ordered potassium chloride 40 mEq, in 250 c.c. normal saline for a total of 500 c.c., to be followed by additional electrolyte studies. (St. Ex. 1E at 29, 72, 95, 108).

Later that day, Dr. Sivashanker noted that Patient 1 had developed acute hypotension. The chest examination revealed rales and rhonchi. Patient 1's serum sodium was 124 and his serum potassium was 2.7. Dr. Sivashanker diagnosed acute pre-renal azotemia. Dr. Sivashanker ordered normal saline to run "wide open" until Patient 1's systolic pressure reached 100. Thereafter, he ordered IV fluids of normal saline with 40 mEq of potassium chloride per liter to run at 125 c.c. per hour. He also ordered Lasix 20-40 mg IV every four hours PRN for congestion. Patient 1's total fluid intake that day was 3422 c.c.; his output was 2035 c.c. The nurses' notes also indicated that Patient 1 had had one large soft stool late in the day. (St. Ex. 1E at 29, 73, 109, 116, 178).

On January 17, 1990, Patient 1 demonstrated possible seizure activity. A CT scan of the brain revealed a possible small infarction of the occipital horn of the left lateral ventricle. No hemorrhage was identified. (St. Ex. 1E at 94).

On January 18, 1990, Dr. Sivashanker noted that Patient 1 was looking "a little bit better." The chest examination revealed bilateral rhonchi. Patient 1's serum sodium was 129, his serum potassium was 4.0, and his serum chloride was 89. In addition, Patient 1's BUN was 31 and his creatinine level was 1.6. Patient 1's total fluid intake that day was 4626; his output was 4025. The nurses' notes indicate that Patient 1 "evacuated a small amount of brown, loose BM" on one occasion. (St. Ex. 1E at 30, 175, 116, 180).

On January 19, 1990, Dr. Sivashanker noted that examination of Patient 1's chest had revealed bilateral rales. Dr. Sivashanker also noted "congested" and "slight fluid overload / see orders." Later in the day, Patient 1's BUN was 49 and his creatinine level was 1.9. Patient 1's total fluid intake that day was 4260; his output was 4555. The

nurses' notes indicate that Patient 1 was incontinent of loose stool on one occasion. Dr. Sivashanker documented "running diarrhea." (St. Ex. 1E at 31, 78, 116, 183).

On January 20, 1990, Dr. Sivashanker noted that Patient 1's blood pressure had dropped to 50/0 and that his chest was clear. Patient 1's serum sodium was 121, his serum potassium was 3.0, and his serum chloride was 79. In addition, Patient 1's BUN was 42 and his creatinine was 1.5. Dr. Sivashanker ordered IV fluids of normal saline with 40 mEq potassium chloride per liter to run "wide open" until the systolic pressure was greater than 90 and, thereafter, at 100 c.c. per hour thereafter. He also ordered that the Capozide be held until the systolic pressure was greater than 120. (St. Ex. 1E at 31, 79, 111).

Later that day, Dr. Sivashanker wrote that Patient 1 was "doing well with Capozide and Cardene but seems to be hypovolemic and dropping BP now, so they were held. Hypokalemia could be secondary to the Capozide, but other causes could be hypo-[illegible] or hypo-[illegible] syndrome." Dr. Sivashanker noted that Patient 1's lungs were "mostly clear. He wrote that Patient 1 was critically ill and remained "No Code Blue." Dr. Sivashanker noted that he would "continue conservative medical therapy as [Patient 1 was] still alert." (St. Ex. 1E at 32).

On January 21, 1990, Dr. Sivashanker noted that Patient 1's systolic pressure was 90 to 100, and his sodium was 121. The chest examination revealed "bilateral rhonchi and few scattered rales - chronic." Patient 1's serum sodium was 134, his serum potassium was 4.1, and his serum chloride was 93. In addition, Patient 1's BUN was 26 and his creatinine level was 1.4. (St. Ex. 1E at 32, 81).

Patient 1 was discharged from the hospital on January 23, 1990. Dr. Sivashanker dictated a discharge summary. The discharge diagnoses include the following: CHF with decompensation; early cardiogenic shock; and acute renal insufficiency secondary to hypovolemia and hypotension. (St. Ex. 1E at 9-12).

*Regarding the Board's allegation that Dr. Sivashanker had treated Patient 1 for CHF, when there was no clinical evidence of CHF.*

#### ***Dr. Miller's Testimony***

27. Dr. Miller testified that Dr. Sivashanker's care and treatment of Patient 1 during the January 5, 1990, hospital admission had fallen below the minimal standard of care in that Dr. Sivashanker had treated Patient 1 for CHF, although the chest x-ray had not revealed any acute changes that would indicate CHF. Dr. Miller testified that the chest x-ray taken January 5 had revealed mild interstitial prominence with minimal blunting which, Dr. Miller testified, are changes seen with chronic lung disease rather than CHF. Dr. Miller testified that, if a person is experiencing acute CHF or pulmonary

edema, the chest x-ray will reveal it in some fashion. Moreover, Dr. Miller further testified that, even with compensated congestive failure, the chest x-ray would likely reveal an enlarged heart. (Tr. at 226-227, 232, 234, 370-371).

Dr. Miller acknowledged that Dr. Frazier had listed his impression of CHF secondary to ASHD and had recommended the use of Lasix for diuresis. Dr. Miller stated that he was “somewhat at a loss to explain” Dr. Frazier’s recommendation. Dr. Miller stated that, if Patient 1 had had dilated cardiomyopathy, it would have been demonstrated on the chest x-ray. Dr. Miller concluded that he disagreed with Dr. Frazier’s opinion. (Tr. at 235).

Dr. Miller also testified that he disagreed with Dr. Frazier’s impression of ASHD because the clinical evidence was not consistent with that diagnosis. Dr. Miller testified that factors that would lead to a diagnosis of ASHD include a medical history of cardiac problems, family history, smoking history, and abnormal cholesterol values, in addition to physical findings and laboratory studies. Dr. Miller further stated that Patient 1’s record revealed no evidence of previous heart disease. (Tr. at 318-340).

Dr. Miller acknowledged that Patient 1 had also demonstrated additional symptoms that were consistent with a diagnosis of CHF, including shortness of breath, an S3 gallop, tachycardia, frothy white sputum, bilateral rhonchi, scattered rales, jugular vein distention, non-pitting edema. Moreover, Dr. Miller acknowledged that the ER physician had diagnosed acute exacerbation of CHF and chronic obstructive pulmonary disease. Nevertheless, Dr. Miller testified that he had concluded that these findings were related to COPD rather than CHF based on the admission chest x-ray and the heart catheterization performed three years earlier. (Tr. at 354-368).

28. Finally, however, Dr. Miller admitted that, in light of the information pointed out to him during cross-examination, his opinion had changed since preparing his expert report and since testifying on direct examination. At that time, Dr. Miller admitted that Patient 1 may have had some “mild congestive heart failure.” (Tr. at 366-368).

Moreover, on cross-examination, Dr. Miller was referred to the series of EKGs performed between January 5 and 7, 1990, which demonstrated an anterior myocardial infarction, age undetermined, and intraventricular conduction delay. Dr. Miller testified that, based on these EKGs, he would have to change his opinion regarding Dr. Frazier’s report. Dr. Miller concluded that the medical record does contain evidence of previous heart disease. (Tr. at 339-344).

#### ***Dr. Sivashanker’s Testimony***

29. Dr. Sivashanker testified that, throughout this hospitalization, he had appropriately treated Patient 1 for CHF. (Tr. at 60-61).

Dr. Sivashanker testified that the question presented is whether Patient 1 had been suffering an exacerbation of CHF or COPD. Dr. Sivashanker testified that Patient 1 had

had chronic COPD with some symptoms of bronchitis and emphysema. He never completely resolved these symptoms because he was a heavy smoker, and continued to smoke. \* \* \* It would be difficult for a reviewer, without examining this patient, nearly nine years after the fact, to say that the patient's dyspnea was due to emphysema or heart failure.

(Tr. at 783-784).

Dr. Sivashanker noted that symptoms consistent with CHF had been documented throughout the hospital record, including rales, frothy sputum, tachypnea, orthopnea, jugular vein distention, S3 gallop, hypotension, systolic murmur, pedal edema and pleural edema. Moreover, Dr. Sivashanker testified that Patient 1 had had an extensive cardiac history, including myocardial infarctions, EKG changes, complaints of chest pain, chronic hypotension related to CHF, and decreased cardiac output as documented by the cardiac catheterization. (Tr. at 785-798, 841-851).

30. Dr. Sivashanker testified that Patient 1 had not presented with an exacerbation of COPD which could be demonstrated by Patient 1's pCO<sub>2</sub> of 41 on admission. Dr. Sivashanker explained that patients with an exacerbation of COPD normally present with a pCO<sub>2</sub> of 55 or greater, as a reflection of marked carbon dioxide retention. Moreover, Dr. Sivashanker testified that Patient 1's sputum was clear, frothy, and/or white, which is more consistent with pulmonary edema than COPD. (Tr. at 806).

***Dr. Mahizhnan's Testimony***

31. Dr. Mahizhnan testified that he disagreed with the Board's allegation that Dr. Sivashanker had inappropriately diagnosed CHF. As basis for his disagreement, Dr. Mahizhnan testified that there had been notes from three physicians supporting the diagnosis of CHF. Moreover, Patient 1 had had symptoms consistent with the diagnosis, including dyspnea, an S3 gallop, increased jugular venous pressure, rales and rhonchi, tachycardia, and edema. (Tr. at 1072-1075).

***Dr. Beaver's Testimony***

32. Dr. Beaver testified that Patient 1 had demonstrated symptoms of CHF, including an S3 gallop, dyspnea, frothy white sputum, rales and rhonchi. Dr. Beaver testified that

Patient 1 may also have had an exacerbation of COPD, as the two commonly occur simultaneously. (Tr. at 1212-1213, 1223-1225).

***Dr. Chinn's Testimony***

33. Dr. Chinn testified that Patient 1 had demonstrated signs of CHF on admission to the hospital on January 5, 1990. Dr. Chinn listed the signs and symptoms as shortness of breath at rest, rales, rhonchi, an S3 gallop, tachycardia, jugular venous distention, pedal edema, and dilated cardiomyopathy. (Tr. at 1389).

*Regarding the Board's allegations that: (1) Dr. Sivashanker had inappropriately continued to prescribe intravenous Lasix concurrently with intravenous saline in order to correct iatrogenic hyponatremia, hypokalemia, and hypotension; and (2) Dr. Sivashanker had interpreted and treated this clinical syndrome as early cardiogenic shock when Patient 1 had actually had volume depletion, pre-renal azotemia, hyponatremia, and hypokalemia from excessive use of diuretics.*

***Dr. Miller's Testimony***

34. Dr. Miller testified that Dr. Sivashanker's care and treatment of Patient 1 during the January 5, 1990, hospital admission had fallen below the minimal standard of care in that Dr. Sivashanker had used diuretics in an inappropriate manner which had resulted in hyponatremia, hypokalemia and hypotension. (Tr. at 226-227, 232).

Dr. Miller testified that the emergency room physician who saw Patient 1 upon admission had diagnosed CHF. Dr. Miller stated that, in a person with chronic lung disease, the physician tries to maintain the patient "a little bit on the dry side." Therefore, some dose of diuretics in the early course of the hospitalization had been appropriate. Dr. Miller stated that the inappropriate prescribing of diuretics had started on approximately January 13 or 14, when the use of diuretics had become excessive. (Tr. at 236-237, 349-350).

35. Dr. Miller testified that on January 13, 1990, Dr. Sivashanker had prescribed Capozide, which is a combination of an ACE inhibitor and a diuretic—hydrochlorothiazide—three times daily for approximately one week. Patient 1 had also received additional doses of Lasix. Dr. Miller testified that, by January 17, Patient 1's serum sodium had dropped to 120, which is a fairly serious situation. In addition, Patient 1's serum potassium had dropped to 2.7. Dr. Miller also testified that the BUN, which measures kidney function and which rises in conditions of dehydration, was also elevated. Finally, Dr. Miller testified that Patient 1's blood pressure had dropped and necessitated the administration of intravenous saline. Dr. Miller concluded that these conditions had resulted from excessive use of diuretics. (Tr. at 228-230).

36. Finally, Dr. Miller noted that Dr. Sivashanker had stated that Patient 1's dehydration, hypokalemia, and hypotension had been caused by Patient 1's not eating or drinking adequate fluids and severe diarrhea. Dr. Miller testified that, even if that was true, giving diuretics to such a patient makes no medical sense in the absence of overt heart failure. (Tr. at 239-240).

***Dr. Sivashanker's Testimony***

37. Dr. Sivashanker testified that he had ordered diuretics only when Patient 1 had demonstrated "acute pulmonary congestion and chest rales" and that diuretics had not been "a significant cause of hyponatremia in this case." Dr. Sivashanker further stated that he had closely monitored the use of diuretics and Patient 1's electrolyte levels. Dr. Sivashanker testified that, although he had ordered the Lasix to be administered four times a day, if needed, Patient 1 had only received Lasix on January 5, January 6 and January 11, 1990. Moreover, Patient 1 had received only 195 mg of thiazide between January 7 and January 10, 1990. Dr. Sivashanker testified that a normal dose of thiazide is between 50 and 100 mg daily. (Tr. at 805-807, 886).
38. Dr. Sivashanker testified that Patient 1 had been on diuretics in the past, and had not had problems with hyponatremia. Therefore, diuretics were probably not the cause of Patient 1's problems during this hospitalization. (Tr. at 807, 884-885).
39. Moreover, Dr. Sivashanker testified that during the January 1990 hospitalization, Patient 1 had had some diarrhea, and when Patient 1's serum sodium levels dropped, his serum potassium also dropped. Accordingly, Dr. Sivashanker argued, the hyponatremia had been caused by incessant diarrhea. (Tr. at 807, 884-885).

***Dr. Mahizhnan's Testimony***

40. Dr. Mahizhnan testified that Patient 1's hypotension had resulted from volume depletion, based on the use of diuretics, diarrhea, and Patient 1's not eating and drinking. (Tr. at 1321-1324).
41. Initially, Dr. Mahizhnan testified that that, in light of Patient 1's severe hypotension, hypovolemia, syncope when standing, and cerebral hypoperfusion, Dr. Sivashanker's management of the diuretic therapy should have been better. (Tr. at 1324-1330).

Moreover, Dr. Mahizhnan testified that the treatment of choice for a patient who has a low blood pressure but who may be in CHF is to monitor via a Swan Ganz catheter. Dr. Mahizhnan further testified that, in a situation like that presented here, where a Swan Ganz catheter is not available, it is appropriate to administer diuretics and IV saline to prevent congestion and maintain blood pressure. On cross-examination, Dr. Mahizhnan

acknowledged that he had assumed that a Swan-Ganz catheter had not been available at that hospital, but he did not know for certain. (Tr. at 1078-1086, 1317-1321).

Later, Dr. Mahizhnan testified that if Patient 1 had been terminally ill and the family desired that Dr. Sivashanker not be very aggressive, then there would have been no need for a Swan-Ganz catheter. Dr. Mahizhnan acknowledged that the family had requested “no further intervention except medical.” Dr. Mahizhnan testified that such a request would rule out any invasive treatment and allow only supportive therapy. Therefore, based on that understanding for Patient 1’s family’s request, Dr. Mahizhnan concluded that Dr. Sivashanker’s treatment of Patient 1 had been appropriate. (Tr. at 1356-1359).

### ***Dr. Beaver’s Testimony***

42. Dr. Beaver acknowledged that Patient 1 may have been experiencing pre-renal azotemia or volume depletion in light of the elevated BUN. Dr. Beaver further stated, however, that when a patient with CHF receives diuretics, the BUN and creatinine will rise slightly. Dr. Beaver acknowledged that the amount of Capozide and Lasix prescribed by Dr. Sivashanker could have caused the electrolyte imbalances in Patient 1. (Tr. at 1216-1219).

Dr. Beaver further testified that it is sometimes difficult to determine the cause of a low blood pressure. Causes can range from volume depletion to cardiogenic shock. Diagnosis often requires special studies like a Swan-Ganz catheter, used to measure the pressures within the heart. Nevertheless, Dr. Beaver stated that Patient 1 may not have been a candidate for such measures. (Tr. at 1163-1164).

Finally, Dr. Beaver testified that no one can know for certain whether Patient 1 had an exacerbation of CHF at that time, without Swan Ganz studies to review. Nevertheless, Dr. Beaver testified that Dr. Sivashanker had made reasonable decisions based on the information available to him at that time. (Tr. at 1219-1223).

### ***Dr. Chinn’s Testimony***

43. Dr. Chinn testified that “[h]ow much Lasix to use for a patient who is in acute heart failure is always a clinical question and it’s a judgment that the doctor who is most immediately at the bedside has to make.” Dr. Chinn testified that there were many factor which led to Patient 1’s hypovolemia, and diuretics probably contributed to it. (Tr. at 1394).

Dr. Chinn further testified that it is probably not appropriate to provide diuretics and intravenous saline for an extended period of time. Nevertheless, in an acutely ill patient who has a number of medical problems, fluids can shift from one body compartment to another. Therefore, it may be appropriate to utilize Lasix to get water out of the lung,

and to give saline to increase the fluid volume in the intravascular space. Dr. Chinn concluded that Dr. Sivashanker had treated Patient 1 appropriately. (Tr. at 1394-1396).

*Regarding the Board's allegation that it had been inappropriate for Dr. Sivashanker to direct the nurses to use intravenous Lasix for chest congestion on an as-needed basis*

***Dr. Miller's Testimony***

44. Dr. Miller testified that writing an order for nurses to give Lasix PRN is not acceptable. Dr. Miller stated that the decision to administer Lasix is a medical decision that the physician makes based on information which may be supplied by the nurses. Dr. Miller concluded that it is below the standard of care for an internist to delegate the decision to a nurse as to when to administer Lasix. (Tr. at 218-221).

***Dr. Sivashanker's Testimony***

45. Dr. Sivashanker admitted that his order, as written, could have caused some confusion. Therefore, Dr. Sivashanker concluded that the order had been inappropriate. Dr. Sivashanker admitted that he had not written any parameters for the nurses to use when determining if Lasix should be administered. Dr. Sivashanker testified that he had intended the nurses to give Lasix if Patient 1 developed a "pulmonary condition," but had failed to document his intention. Nevertheless, Dr. Sivashanker testified that he had discussed with the nurses when the nurses were to give Lasix. In addition, Dr. Sivashanker reasoned that the nurses had not had any problems with the order because they had not called him with questions. (Tr. at 61-62, 853-856).

***Dr. Mahizhnan's Testimony***

46. Dr. Mahizhnan testified that it is not unusual for physicians to rely on nurses when determining the need for PRN medications, especially when the physician is familiar with the nurses and the nurses are competent. Nevertheless, Dr. Mahizhnan acknowledged that, with a medication such as Lasix, it would be preferable to have the nurse call the physician before administering the medication. (Tr. at 1075-1078).

***Dr. Beaver's Testimony***

47. Dr. Beaver testified that an order for nurses to give Lasix on a PRN basis is merely a variation of what physicians do on a routine basis. Dr. Beaver stated that all physicians rely on nurses' judgment. Nevertheless, on cross-examination, Dr. Beaver testified that he has never written an order for Lasix PRN and that he would instruct resident under his tutelage not to write such an order. Dr. Beaver explained that "the argument about the order is that it allows the nurses to have too much leeway; in other words, too much judgment. They are not trained to do that and therefore, they may

administer inappropriate treatment.” Nevertheless, Dr. Beaver maintained his position that writing an order in this manner is “not a huge variation from standard practice.” (Tr. at 1196-1200).

***Dr. Chinn’s Testimony***

48. Dr. Chinn testified that prescribing Lasix PRN “at the discretion of the nurse without parameters is probably not appropriate.” (Tr. at 1391-1393, 1477-1483).

**PATIENT 2**

Patient 2, a 74 year old female, was admitted to St. Ann’s Hospital in Westerville, Ohio, on January 27, 1990, after suffering a left hemispheric cerebral vascular accident [CVA]. Dr. Sivashanker first met Patient 2 upon her admission to the hospital. (Tr. at 70; St. Ex. 2A at 2-4).

**PATIENT 2’S JANUARY 27, 1990, HOSPITAL ADMISSION**

*Basis for the Admission; Hospital Course*

49. Patient 2, a 74 year old female, was admitted to St. Ann’s Hospital in Westerville, Ohio, on January 27, 1990, after suffering a left hemispheric stroke. Patient 2’s family reported that Patient 2 had been having blackout spells over the past month, with right sided weakness and speech difficulties. Her past medical history included transient ischemic attacks; atrial fibrillation; severe phlebitis of the right leg with thromboembolism; and hypertension. (St. Ex. 2A at 3, 9).

Upon admission, Dr. Sivashanker evaluated Patient 2 and diagnosed the following:

- Right hemiplegia, possible middle cerebral artery territory;
- Most likely embolic stroke;
- Chronic atrial fibrillation with frequent premature ventricular contractions;
- Exogenous obesity;
- Chronic bilateral varicose veins with chronic edema of the feet due to chronic venous insufficiency; and
- History of pulmonary embolism in the past.

(St. Ex. 2A at 9). In his admission plan, Dr. Sivashanker advised that he would employ IV steroids to reduce any cerebral edema. He further noted that he would

obtain a CT of the brain and, if the CT of the brain was negative, he would start IV Heparin. (St. Ex. 2A at 10, 17).

On January 27, 1990, Dr. Sivashanker obtained a neurological consultation with Leslie A. Friedman, M.D. In his consultation report, Dr. Friedman recommended the following:

As we discussed, the issue of heparin with acute cardiac embolic stroke is controversial when deficit is large (which indicates considerable ischemic brain is present). A significant number of these can become hemorrhagic - so most of the stroke 'experts' say to hold off on heparin for a few days, repeat CT, and, if no signs of bleed, then OK to start Coumadin.

In the meantime, Duplex, Echo, EEG, etc. Start PT-OT. Look at Dodd [Hall] vs. Mt. Carmel for rehab. I spoke with daughters and explained things.

(St. Ex. 2A at 11).

On January 28, 1990, Dr. Sivashanker ordered a cardiology consultation with Bruce W. Graham, M.D., or H. Joel Gorfinkle, M.D. Patient 2 was seen by Dr. Graham later that day. Dr. Graham noted a history of rheumatic fever and atrial fibrillation, but stated that Patient 2's cardiac status appeared to be stable at that time. (St. Ex. 2A at 12).

On January 31, 1990, Dr. Friedman advised that the CT should be repeated. Moreover, Dr. Friedman wrote that, if no problem was indicated on the CT, he would recommend low dose Coumadin in light of the continuing atrial fibrillation. On February 1, 1990, Dr. Sivashanker wrote that he had discussed anticoagulation with Dr. Friedman. (St. Ex. 1A at 19, 20).

On February 1, 1990, Dr. Friedman noted that the CT had shown a new infarct at the left basal ganglia without signs of bleeding. Dr. Friedman wrote "it is OK to gently start Coumadin and aim for a ProTime of  $\approx 17$  as we discussed." Dr. Sivashanker started Coumadin and ordered daily ProTime levels. (St. Ex. 2A at 20, 31, 45).

Social services made frequent notes regarding planned placement in a rehabilitation facility after discharge. On February 3, 1990, Dr. Sivashanker noted that Patient 2 was stable and that her coagulation studies were adequate. On February 4, 1990, Dr. Gorfinkle signed off Patient 2's case, noting that the cardiac status was stable and that cardioversion would not be considered. (St. Ex. 2A at 19, 21, 22).

On February 6, 1990, Patient 2 was seen by Richard A. Edgin, M.D., a gastroenterologist, for complaints of nausea and epigastric distress. Dr. Edgin performed an endoscopy which revealed two duodenal ulcers “without stigmata of hemorrhage.” Dr. Edgin recommended the use of Zantac indefinitely, so long as Patient 2 continued to take Coumadin. He further recommended that, if Patient 2 were to develop a bleeding problem, she should be given antibiotic prophylaxis and be treated with a heater probe. (St. Ex. 2A at 13, 14).

On February 7, 1990, Dr. Sivashanker noted that Patient 2 had had an endoscopy, but made no additional comments regarding Patient 2’s gastrointestinal status. Patient 2 was discharged later that day. Dr. Sivashanker discharged Patient 2 to Dodd Hall on daily Coumadin along with one baby aspirin per day. He continued to follow Patient 2 in his office. (St. Ex. 2A at 3-4, 24).

*Regarding the Board’s allegation that Dr. Sivashanker inappropriately failed to document in the medical records his rationale for treating Patient 2 with Coumadin, in light of the active duodenal ulcer.*

***Dr. Miller’s Testimony***

50. Dr. Miller testified that Dr. Sivashanker’s care and treatment of Patient 2 during the January 27, 1990, hospitalization had fallen below the minimal standards of care because Dr. Sivashanker had failed to document his consideration of the risks and benefits of anti-coagulant use. Moreover, Dr. Miller testified that Dr. Sivashanker had not documented his plans to monitor Patient 2 for signs of bleeding after hospitalization. (Tr. at 245-247).

Dr. Miller testified that his concern was that Dr. Sivashanker had failed to document his rationale for using Coumadin in light of the risks of using Coumadin. Dr. Miller testified that, on one hand, Patient 2 had suffered a stroke while maintaining a cardiac rhythm of atrial fibrillation, which strongly supports the use of an anti-coagulant. On the other hand, Patient 2 had demonstrated signs of gastric bleeding, for which the use of anti-coagulants is risky. (Tr. at 245-247, 431-442, 466).

Dr. Miller acknowledged that the medical record contained the report of Dr. Edgin, who recommended that Patient 2 be treated with antibiotic prophylaxis and a heater probe if Patient 2 started to bleed. Dr. Miller testified that this was not sufficient documentation as it addressed only treatment during hospitalization rather than after discharge. (Tr. at 248).

Dr. Miller further acknowledged Dr. Friedman’s report discussing the proper use and monitoring of anti-coagulation therapy. Dr. Miller testified that it is very important to obtain specialist consultation, but it is necessary that the attending physician, who is

“the captain of the ship” to document everything that is happening to the patient, including his instructions to the patient. (Tr. at 249).

51. Dr. Miller testified that, other than the failure to document, Dr. Sivashanker’s treatment of Patient 2 had been proper and that Dr. Sivashanker had appropriately used Coumadin to treat Patient 2’s condition. (Tr. at 244-245, 431-442).

***Dr. Sivashanker’s Testimony***

52. When asked where in the record he had documented his thinking on the use of Coumadin therapy, Dr. Sivashanker referred to a number of different consultant’s reports, including neurology, gastroenterology, and cardiology. Dr. Sivashanker also referred to his own documentation referring to opinions of consultants. Dr. Sivashanker could not locate any place in the hospital record where he documented his own thinking apart from an analysis of the consultants’ reports. (Tr. at 69-77).

***Dr. Mahizhnan’s Testimony***

53. Dr. Mahizhnan testified Dr. Sivashanker’s notes indicate that he had agreed with the consultants; therefore, there had been enough documentation. (Tr. at 1102-1103).

***Dr. Beaver’s Testimony***

54. Dr. Beaver testified that the medical record had contained insufficient documentation of Dr. Sivashanker’s reasons for initiating Coumadin therapy. (Tr. at 1227).

***Dr. Chinn’s Testimony***

55. Dr. Chinn testified that Dr. Sivashanker had not specifically documented his rationale for treating Patient 2 with Coumadin in light of the active duodenal ulcer. Nevertheless, the record demonstrates that Dr. Sivashanker had considered the risks and benefits of Coumadin therapy. Dr. Beaver noted that Dr. Sivashanker had consulted with a cardiologist, a gastroenterologist and a neurologist, all of whom acknowledged the risks and benefits and recommended the use of Coumadin. Dr. Chinn concluded that Dr. Sivashanker had provided sufficient documentation. (Tr. at 1401-1404).

**PATIENT 2’S MAY 21, 1991, HOSPITAL ADMISSION**

*Dr. Sivashanker’s Office Records, Post January 27, 1990, Hospital Admission*

56. A discharge summary from Dodd Hall at the Ohio State University Hospital revealed that, on March 27, 1990, an echocardiogram had been performed on Patient 2. The

echocardiogram had revealed “a dilated left ventricle with diffuse hypocontractility. She had a mild to moderate degree of aortic insufficiency and the possibility of a left ventricular thrombus was noted.” The echocardiogram was repeated and confirmed the presence of a left ventricular thrombosis. The report indicated that full anticoagulation was initiated with IV Heparin which was changed over to oral Coumadin prior to Patient 2’s discharge from Dodd Hall. Patient 2 was discharged home on Coumadin 5 mg daily. (St. Ex. 2 at 66, 68).

*Basis for the May 21, 1991, Hospital Admission; Hospital Course*

57. On May 21, 1991, Dr. Sivashanker admitted Patient 2 to Mount Carmel Medical Center for complaints of frequent “blackout spells” over the past week. Family members represented that the spells had occurred more often when Patient 2 was in a supine position. In addition, a Holter monitor had revealed atrial fibrillation with long pauses and frequent premature ventricular contractions. Upon admission to the hospital, Dr. Sivashanker ordered Coumadin 2.5 mg and baby aspirin ½ tablet daily. Patient 2’s hemoglobin was 13.3 [normal range 12.0-16.0]; her hematocrit was 40.7 [normal range 37-47]; her ProTime was 12.5 [control 1.4-12.7]; and her stool guiac was negative. Dr. Sivashanker obtained cardiac consultation with Dr. Beaver. (St. Ex. 2B at 6-7, 21, 31, 81).

On May 22, 1991, a CT of the head revealed a small focal lacunar infarct, most likely old. (St. Ex. 2B at 122).

On May 23, 1991, Dr. Sivashanker performed a rectal examination. He found hard stool with “streaks of blood” on his glove. The stool tested guiac positive. Dr. Sivashanker questioned hemorrhoids and colon cancer. He discontinued the Coumadin and baby aspirin, and consulted Robert Ludwig, M.D., a gastroenterologist. Dr. Ludwig recommended a sigmoidoscopy. (St. Ex. 2B at 18, 24, 32).

Dr. Sivashanker performed a sigmoidoscopy on May 24, 1991, with Dr. Ludwig observing. Dr. Sivashanker noted multiple diverticuli and internal hemorrhoids. Nevertheless, Dr. Sivashanker noted that visualization had been precluded by a large amount of stool in the colon. Dr. Sivashanker planned a colonoscopy to rule out colon cancer. (St. Ex. 2B at 9).

On May 25, 1991, Dr. Sivashanker ordered serial stool guiac for three days. Patient 2’s stool tested positive that day. (St. Ex. 2B at 25, 33).

On May 28, 1991, Patient 2 refused the colonoscopy. Thereafter, Dr. Sivashanker discussed the test with Patient 2, and Patient 2 agreed to have it performed. Nevertheless, the record contains only the report of a barium enema, which revealed

multiple diverticuli. Patient 2's hemoglobin was 13.8 and her hematocrit was 42.8. (St. Ex. 2B at 35, 81, 124).

On May 29, 1991, Dr. Sivashanker wrote that he would not pursue the gastrointestinal bleeding any further, other than to continue guiac testing. (St. Ex. 2B at 35). On May 30, 1991, Patient 2's stool tested positive for blood. (St. Ex. 2B at 36).

On May 31, 1991, Patient 2 was discharged to her daughter's home. Patient 2's hemoglobin was 14.6 and her hematocrit was 45.6. Her ProTime was 12.5. Dr. Sivashanker noted that he would continue to monitor her stool guiacs and blood work after discharge. Dr. Sivashanker did not reinstitute the Coumadin or baby aspirin upon discharge. Patient 2's cardiac rhythm remained atrial fibrillation. (St. Ex. 2B at 5, 20, 29, 37, 81).

58. Dr. Sivashanker saw Patient 2 in his office twice in June 1991. He ordered complete bloodwork at that time. He did not order stool guiac. In October 1991, during a pelvic examination, Dr. Sivashanker found blood in Patient 2's cervix. A CT of the pelvis was within normal limits for the patient's age. (St. Ex. 2 at 8-9, 103).

In July 1992, Dr. Sivashanker noted that Patient 2 was being evaluated for nursing home placement. There is no indication, however, that Patient 2 was placed in a nursing home at that time. (St. Ex. 2B at 11).

In September 1993, Dr. Sivashanker noted bleeding in Patient 2's underwear, but he could not tell whether the source was rectal or vaginal. Patient 2 also complained of right lower quadrant abdominal pain. Dr. Sivashanker scheduled a pap and pelvic examination and a sigmoidoscopy. He also ordered a complete blood count. (St. Ex. 2 at 13).

In April 1995, Dr. Sivashanker noted that Patient 2 was residing in a nursing home. He also noted that her stool guiac had been negative. (St. Ex. 2 at 14).

59. Dr. Sivashanker's office notes contain a May 22, 1995, consultation written by Brian P. Fahey, D.O., a neurologist, to William P. Maher, D.O. Dr. Fahey advised that Patient 2 had presented with possible symptoms of TIA, including numbness on the left side and difficulty standing. In his plans, Dr. Fahey recommended an MRI of the brain, continued daily aspirin, and consideration of the use of Coumadin. Dr. Fahey wrote: "With the history of atrial fibrillation - I naturally raise the possibility of use of Coumadin, although we have to consider the risks of medication." (St. Ex. 2B at 128-129).

On May 31, 1995, an MRI revealed "Findings of old deep lacunar infarcts on the left, one of which appears hemorrhagic as indicated by the presence of hemosiderin. There

do not appear to be findings of a new cerebral infarct based on these studies.” (St. Ex. 2B at 122-123).

On June 12, 1995, Dr. Fahey again wrote to Dr. Maher. At that time, Dr. Fahey advised that it should be assumed that Patient 2 was suffering “cerebrovascular events (TIA or reversible neurologic deficit).” He further advised as follows:

With the history of atrial fibrillation, we must constantly rule out the need to use Coumadin on a long-term basis; therefore, I have taken the liberty of scheduling an appointment with Dr. Beaver to evaluate for recommendation on the long-term use of Coumadin. We must consider the risks of this medication, especially with her increased risk of falling due to previous stroke. However, she is wheelchair dependent, decreasing the risks from falling and subsequent complications from Coumadin.

(St. Ex. 2 at 130-131). On June 23, 1991, Dr. Sivashanker further noted that Dr. Fahey had “agreed on low dose Coumadin. Dr. Sivashanker prescribed Coumadin, 1 mg daily. (St. Ex. 2B at 15).

*Regarding the Board's allegations that:*

- (1) *Prior to admission, Patient 2 had been taking Coumadin and baby aspirin for stroke prevention. The Coumadin and aspirin were discontinued after a digital rectal examination showed “streaks of blood on glove.” To evaluate this finding, Dr. Sivashanker performed a flexible sigmoidoscopy and a barium enema, which showed only diverticuli.*
- (2) *Nevertheless, when discharged on May 31, 1991, Patient 2 was still in atrial fibrillation, but Dr. Sivashanker did not prescribe Coumadin. Dr. Sivashanker should have reinstated Coumadin therapy for Patient 2 at discharge or in the out-patient setting if follow-up stool guiacs were negative.*
- (3) *Moreover, Dr. Sivashanker's medical records indicate that Dr. Sivashanker did not perform stool guiac testing following discharge and that Patient 2 did not resume taking Coumadin until May 1995, when the Coumadin was reinstated at a neurologist's recommendation. Patient 2 remained in atrial fibrillation and continued to be at risk of recurring stroke throughout this time period, in part due to Dr. Sivashanker's failure to continue Coumadin therapy*

***Dr. Miller's Testimony***

60. Dr. Miller testified that Dr. Sivashanker's care and treatment of Patient 2 during the May 21, 1991, hospitalization had fallen below the minimal standard of care due to Dr. Sivashanker's failure to reinstitute anti-coagulation therapy. Dr. Miller testified that Patient 2's risk of additional stroke was high, based on her prior stroke, the blood clots found in her left ventricle, and the continued atrial fibrillation. (Tr. at 251-252).
61. Initially, Dr. Miller testified that Dr. Sivashanker's various reasons for discontinuing the Coumadin had not been sufficient. Dr. Miller testified that there had been no indication of a significant bleeding problem, such as a drop in blood pressure or blood count. Furthermore, Dr. Miller testified that the amount of blood noted by Dr. Sivashanker, streaks of blood on a glove during a rectal examination, was an insignificant amount of blood when considering termination of Coumadin. Dr. Miller further testified that there had apparently been no concern about the possibility of an ulcer, as that had not been addressed in the record in any way. Dr. Miller concluded that if there had been concern for any serious contraindication to the use of Coumadin, which was so strongly indicated in this patient, there should have been a comprehensive note indicating those concerns. (Tr. at 254, 485-486).

On cross-examination, however, Dr. Miller testified that Dr. Sivashanker's discontinuance of Coumadin had been appropriate. Dr. Miller clarified his earlier testimony and stated that Dr. Sivashanker's shortcoming had been in failing to restart Coumadin therapy after Patient 2 had been evaluated for potential bleeding sites. Moreover, Dr. Miller testified that Dr. Sivashanker should have clearly documented the rationale for his decision. Dr. Miller testified that it is the standard of care to document the rationale for treatment and the issues that may occur upon discharge. Dr. Miller explained that "it is important to know how the physician is planning to deal with potential adverse events after discharge. (Tr. at 490-493, 738-739).

62. Moreover, Dr. Miller testified that Dr. Sivashanker should have documented a plan for follow up and eventual reinstitution of Coumadin in an outpatient setting. Dr. Miller further noted that, in reviewing Dr. Sivashanker's post-discharge office notes, there is no mention of any GI bleeding or that a stool guaiac had been performed. (Tr. at 254-256).
63. Dr. Miller testified that Dr. Sivashanker's reinstitution of Coumadin in 1995 had been appropriate. Dr. Miller criticized Dr. Sivashanker's failure to prescribe Coumadin between 1991 and 1995, when Patient 2 had been at risk for additional stroke. (Tr. at 257-258).

***Dr. Sivashanker's Testimony***

64. Dr. Sivashanker stated that he had discontinued Coumadin, and had decided not to reinstitute its use upon discharge, for the following reasons:

- Patient 2 had had an active lower GI bleed, and Dr. Sivashanker had not located the source.
- Dr. Sivashanker had been concerned about the harm that could result had he restarted the Coumadin, had Patient 2 started to bleed, and had the gastroenterologist been unable to reach the source.
- Dr. Sivashanker had originally started Coumadin with the approval of the gastroenterologist so long as there was no active bleeding. Therefore, the bloody stools had been a contraindication to the use of Coumadin according to the gastroenterologist.
- Patient 2 had been hospitalized due to syncopal attacks. Moreover, she was trying to be more active, and getting out of her wheelchair. Dr. Sivashanker stated that Patient 2 could have sustained serious injuries had she passed out and fallen while taking Coumadin.
- In 1991, Patient 2 had presented with gynecological bleeding. In 1993, Patient 3 had presented with bleeding in her underwear. Dr. Sivashanker testified that these events had been contraindications to reinstating Coumadin.
- Patient 2 had had a “clear-cut” lacunar infarct hemorrhage as documented by CT scan on May 31, 1995. Despite the fact that the infarct was old, Dr. Sivashanker testified that Coumadin is contraindicated in a patient with a lacunar infarct.

(Tr. at 90-93, 100-101, 107, 110, 918-925, 950-951).

65. When asked if he had documented his rationale for not reinstating Coumadin therapy, Dr. Sivashanker again reviewed findings in the record and consultant reports. Dr. Sivashanker did not locate any specific documentation in the medical records setting forth his rationale for not reinstating Coumadin therapy upon Patient 2's discharge from the hospital. Dr. Sivashanker stated as follows:

I did not write exactly why I didn't restart. I assumed that I had enough reasons not to. But I can only say that here on the documentation of why it was contraindicated, which was not written in black and white here, but I still felt it was contraindicated.

(Tr. at 93-100, 925). Moreover, Dr. Sivashanker acknowledged that he had not addressed the risks of not using Coumadin anywhere in the record. (Tr. at 939).

66. Dr. Sivashanker also stated that Patient 2 had been discharged to a nursing home after hospitalization. Dr. Sivashanker testified that Patient 2 had been primarily managed by three nursing home physicians. Dr. Sivashanker further stated that he did not see Patient 2 in the nursing home, but that she sporadically came to his office for follow-ups. Dr. Sivashanker testified that Patient 2 had been managed by a number of physicians between 1991 and 1995, and none of the physicians had reinstated Coumadin. (Tr. at 102, 105).
67. Dr. Sivashanker testified that he had reinstated Coumadin in 1995, despite the continuing risk factors, for the following reasons:
- Patient 2 had not had any significant GI bleed between 1991 and 1995.
  - By 1995, Patient 2 had been wheelchair bound, and was not as likely suffer injuries due to a fall.
  - Patient 2's lacunar infarct had become stable.
  - Patient 2 had been evaluated by a neurologist and a cardiologist, who had recommended reinstating the Coumadin.
  - In 1995, Patient 2 had presented with new cardiovascular events, in the absence of active bleeding for a number of years.

(Tr. at 106-109, 964).

***Dr. Mahizhnan's Testimony***

68. Dr. Mahizhnan testified that, in order to diagnose the source of Patient 4's gastrointestinal bleeding, an arteriogram would have been appropriate. Dr. Mahizhnan testified, however, that Patient 2 had refused any further testing. On closer review of the record, however, Dr. Mahizhnan acknowledged that Patient 1 had only refused a colonoscopy. Dr. Mahizhnan further acknowledged that the record did not indicate whether an arteriogram had been offered or if the bleeding had been serious enough to warrant the test. (Tr. at 1106-1107).
69. Nevertheless, Dr. Mahizhnan testified that it had been appropriate to withhold Coumadin. Dr. Mahizhnan reasoned that Dr. Sivashanker had not located the source of the bleed and Patient 2 was falling frequently, which increased the risk of injury resulting in bleeding. Furthermore, Dr. Mahizhnan testified that, since Patient 2 had been taking Coumadin for more than a year, the problem with ventricular thrombi may have been resolved, decreasing the need for continued Coumadin. (Tr. at 1123-1125, 1336-1344).

On cross-examination, however, Dr. Mahizhnan acknowledged that Dr. Sivashanker had not documented that his reason for discontinuing Patient 2's Coumadin was the blood on the glove after rectal examination. Dr. Mahizhnan had made the assumption because it was implied. Dr. Mahizhnan further testified on cross-examination that it would have been appropriate to hold the Coumadin "for a few days" or until the source of bleeding had been determined. Dr. Mahizhnan acknowledged that Dr. Sivashanker had not documented his reasons for not reinstating Coumadin or for not pursuing further testing regarding the source of the bleeding. (Tr. at 1331-1332, 1340-1344).

***Dr. Beaver's Testimony***

70. Dr. Beaver testified that determining the risks and benefits of using Coumadin is a matter of judgment, and that experts often disagree. Dr. Beaver further testified that it had been reasonable for Dr. Sivashanker to discontinue Coumadin in order to evaluate Patient 2's gastrointestinal bleeding and syncopal episodes. (Tr. at 1170-1173).

Nevertheless, Dr. Beaver testified that a patient who has atrial fibrillation, has had a previous stroke due to embolus, and has had a left ventricular thrombus is at a fairly high risk for another stroke. Dr. Beaver further testified that that person should be anticoagulated unless there was a strong contraindication, such as bleeding. (Tr. at 1238-1239).

71. Dr. Beaver testified that most physicians would document their reasons for discontinuing Coumadin in a patient such as Patient 2. Moreover, the physician should included in the discharge summary the reasons for starting and stopping the drug. Dr. Beaver stated that the decision whether to anticoagulate is a significant medical decision. (Tr. at 1227-1230).

***Dr. Chinn's Testimony***

72. Dr. Chinn testified that Dr. Sivashanker had identified bleeding from the rectum during the hospital admission. Moreover, Dr. Sivashanker had identified two additional bleeding sites post-hospitalization. Dr. Chinn testified that these findings would "mitigate against" resuming anticoagulation therapy. (Tr. at 1417-1421).

**PATIENT 2'S SEPTEMBER 11, 1996, VISIT TO DR. SIVASHANKER'S OFFICE**

*Dr. Sivashanker's office note for September 11, 1996*

73. Dr. Sivashanker's office note for September 11, 1996, states that Patient 2 had presented with complaints of coughing up blood, pain in the right side of abdomen,

and bleeding from the vagina. The note also contains some vital signs, and a past medical history. There is no evidence that Dr. Sivashanker examined Patient 2 or listed a plan of treatment. The note dated September 11, 1996, does not continue to the following page in the medical record. (St. Ex. 2 at 16).

Nevertheless, the office record also contains an undated note at Page 4, which contains family and social history, medications, and a SOAP evaluation. In the observations, Dr. Sivashanker notes hemoptysis, rule out tuberculosis; rule out cervical carcinoma; rule out bleeding secondary to excess Coumadin. Dr. Sivashanker also decreased Patient 2's Coumadin to 1 mg every other day and ordered a PPD skin test. (St. Ex. 2B at 4). [Note: Dr. Sivashanker argued that Page 4 is a continuation of the September 11, 1996, note found at Page 16 in the exhibit. See Tr. at 114-116].

*Regarding the Board's allegation that, on September 11, 1996, Patient 2 had presented to Dr. Sivashanker's office with complaints of hemoptysis and vaginal bleeding; Dr. Sivashanker had failed to document how he addressed these symptoms*

***Dr. Miller's Testimony***

74. Dr. Miller testified that, if Page 4 is not a continuation of the entry for September 11, 1996, than the care Dr. Sivashanker provided on that date departed from the minimal standards of care. (Tr. at 283-284).

***Dr. Mahizhnan's Testimony***

75. Dr. Mahizhnan testified that Page 4 is a logical continuation of Page 16. Moreover, the treatment was appropriate for the symptoms with which Patient 2 had presented, but for the fact that Dr. Sivashanker did not order a chest x-ray. (Tr. at 1114-1121).

**PATIENT 3**

Patient 3, an 82-year-old woman, was admitted to St. Ann's Hospital on March 15, 1990. Dr. Sivashanker first met Patient 2 upon her admission to the hospital. (Tr. at 70; St. Ex. 3A at 3).

**PATIENT 3'S MARCH 15, 1990, HOSPITAL ADMISSION**

*Basis for the Admission; Hospital Course*

76. Patient 3 presented to the ER at St. Ann's Hospital on March 15, 1990. Patient 3 had vague complaints of chest pain and a cough of approximately two months duration. Patient 3 had been admitted at Mount Carmel Medical Center two months earlier for

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treatment of CHF and ASHD. Moreover, one year earlier, Patient 3 had been hospitalized with hallucinations and organic brain syndrome. Patient 3 was a resident of a nursing home under the care of a family physician. Her routine medications included Lasix, hydrochlorothiazide, Vasotec, and Indocin. Patient 3 was alert and oriented upon admission. (St. Ex. 3A at 6, 10).

In the ER, examination of Patient 3 revealed a “persistent congested cough,” ankle edema, and minimal jugular vein distention. The EKG demonstrated atrial fibrillation with a ventricular response of 120 to 130, although her usual rhythm was sinus. Arterial blood gases showed a pH of 7.46, a pCO<sub>2</sub> of 40 and a pO<sub>2</sub> of 69. A chest x-ray revealed an enlarged heart, pulmonary vascularity within normal limits, and “essentially clear” lungs. The radiologist listed his impression as “cardiomegaly without overt CHF.” Nevertheless, the ER physician wrote that there was “some cephalization of the vasculature.” Patient 3 received Lasix 40 mg IV. Her intake in the ER was 150 c.c. and her output was 1760 c.c. (St. Ex. 3A at 6-10, 39, 73).

In the ER, Patient 3’s serum sodium was 133 [normal range 142-151], her serum potassium was 4.0 [normal range 3.8-5.3], and her serum chloride was 97 [normal range 106-114]. Patient 3 was admitted to the intermediate care unit under the care of Dr. Sivashanker. Her orders included Lasix 40 mg PO daily and an IV of D<sub>5</sub>W at a KVO [keep vein open] rate. Dr. Sivashanker was consulted to care for Patient 3 because Dr. Sivashanker was the internist on call at the hospital. (St. Ex. 3A at 10, 39, 63).

In the History and Physical, Dr. Sivashanker wrote that Patient 3’s breath sounds were diminished and distant with a few scattered rales and rhonchi in the bases. Patient 3 also had an S3 gallop. Dr. Sivashanker listed diagnoses of chest pain, rule out unstable angina; atrial fibrillation with rapid ventricular response; senile dementia; and hypertension, uncontrolled. Dr. Sivashanker discontinued the order for daily Lasix. Instead, he ordered Lasix 40 mg IV four times a day PRN for increased chest congestion. His orders also included Capoten 12.5 mg three times a day. (St. Ex. 3A at 11, 40).

On March 16, 1990, Dr. Sivashanker noted that Patient 3 was agitated and confused, and that her lungs were clear. Patient 3’s serum sodium was 133, her serum potassium was 4.0, and her serum chloride was 97. Patient 3’s chest x-ray was “free of any infiltrate or failure.” Patient 3’s total fluid intake that day was 2309 c.c.; her total output was 1450 c.c. Patient 3 received no diuretics. (St. Ex. 3A at 18, 62, 74, 246, 257-258).

On March 17, 1990, Dr. Sivashanker noted that Patient 3 had a dry cough, that she was short of breath at rest, and that her chest had a few scattered rales. Patient 3’s total

fluid intake that day was 930 c.c.; her total output was 950 c.c. Patient 3 received no diuretics. (St. Ex. 3A at 18, 246, 257-258).

On March 18, 1990, Dr. Sivashanker noted a dry cough, "possibly secondary to Capoten." He stated that he would discontinue the Capoten to see if Patient 3's cough disappeared. Dr. Sivashanker also noted scattered rales. Patient 3's serum sodium was 128, her serum potassium was 4.0, and her serum chloride was 94. Patient 3's total fluid intake that day was 1320 c.c., and her total output was 1300 c.c. Patient 3 received no diuretics. (St. Ex. 3A at 18, 41, 62, 246, 257-258).

On March 19, 1990, Dr. Sivashanker noted confusion and disorientation. Patient 3's serum sodium was 128, her serum potassium was 3.6, and her serum chloride was 94. Dr. Sivashanker noted scattered rhonchi, no rales and no edema. Dr. Sivashanker questioned whether the confusion was secondary to hyponatremia. Dr. Sivashanker changed the IV to normal saline, with 10 mEq of potassium chloride per liter, to run at 100 c.c. per hour. He also ordered a CT of the brain, and consulted Dr. Friedman, a neurologist, and Dr. Grodner, a pulmonologist. (St. Ex. 3A at 19, 42, 62).

On March 19, 1990, Patient 3 was evaluated by Dr. Grodner. Dr. Grodner listed impressions of chronic bronchitis, early CHF and atrial fibrillation. He recommended a long acting theophylline, a beta-2 agonist aerosol, and Humibid LA. (St. Ex. 3A at 12).

Patient 3 was also evaluated by Dr. Friedman. Dr. Friedman noted that Patient 3 had become more confused since admission, that her gait had deteriorated, and that her serum sodium had decreased to 128. Dr. Friedman listed his impressions as senile dementia due to Alzheimer's disease or multiple small infarct dementia, with deterioration probably due to "ICU psychosis" and/or the decreasing sodium level. Patient 3's total fluid intake that day was 2432 c.c.; her total output was 3200 c.c. Patient 3 received no diuretics. (St. Ex. 3A at 13, 246, 259-260).

On March 20, 1990, an enhanced CT of the brain was completed. Dr. Friedman noted that the CT had demonstrated an infarction of the left parietal area. Dr. Sivashanker wrote that Patient 3 continued to be confused, and documented that her lungs were clear. Patient 3's total fluid intake that day was 2757 c.c.; her total output was 1800 c.c. She received no diuretics. (St. Ex. 3A at 13, 20-22, 248, 259-260).

On March 21, 1990, Dr. Sivashanker documented bilateral rhonchi. He ordered a cardiac consultation for CHF with Drs. Graham and Gorfinkle. Patient 3's serum sodium was 133, her serum potassium was 3.6, and her serum chloride was 103. Her chest x-ray demonstrated "a mild amount of blunting at the left costophrenic angle [which] could be due to a small effusion or pleural adhesions. The lungs themselves [were] well expanded and clear." (St. Ex. 3A at 20, 44, 62, 76).

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On March 21, at 6:00 p.m., Dr. Graham evaluated Patient 3. Dr. Graham noted that Patient 3 had upper airway noise, but that her lungs were otherwise clear. Dr. Graham diagnosed aortic insufficiency and questionable mitral regurgitation, secondary to hypertension or SBE. He recommended an echocardiogram and cultures. Patient 3's total fluid intake that day was 3601 c.c.; her total output was 1150 c.c. Patient 3 received no diuretics. (St. Ex. 3A at 14, 248, 259-260).

On March 22, 1990, Dr. Sivashanker noted that Patient 3 had been coughing less and suggested that the cough was unrelated to Capoten. He also noted that her lungs were clear and her sodium was 133. Dr. Sivashanker decreased the IV rate to KVO. (St. Ex. 3A at 22, 44, 45).

On March 22, Dr. Graham noted that an echocardiogram had revealed that Patient 3's aorta was dilated but there were no signs of dissection. Her diastolic murmur was unchanged. He further noted that an echocardiogram had shown "fairly good" left ventricular-right ventricular function" and "only mild to moderate" valvular abnormalities. (St. Ex. 3A at 23, 87).

During the day, the nurses' notes indicate that Patient 3's respirations were more labored, that Patient 3 had complained of cramping in the chest, and that Patient 3's "congestion" had increased. Her pO<sub>2</sub> was 66 at room air. At 6:00 p.m., Lasix 40 mg was given. An additional 40 mg was given at 4:00 a.m. the following day. Patient 3's total fluid intake was 2857 c.c.; her total output was 5000 c.c. (St. Ex. 3A at 24, 46, 151, 248, 259-260, 264; Tr. at 987-992).

On March 23, 1990, Dr. Graham wrote "She will be prone to fluid overload. Note the very high intakes of last 3 days. If that's avoided, then she won't need much else." (St. Ex. 3A at 24).

Dr. Sivashanker wrote that Patient 3 had developed chest rales during an episode of chest congestion the previous day. Dr. Sivashanker further noted that Patient 3 had been given Lasix 80 mg IV. Patient 3's serum sodium was 133, her serum potassium was 3.3, and her serum chloride was 96. Dr. Sivashanker noted bilateral rhonchi, no rales. At approximately 3:30 p.m., Dr. Sivashanker ordered Capozide 25/50 three times a day and Micro K. Dr. Sivashanker later ordered that the "hydrochlorothiazide" be decreased if Patient 3 got "too dry." Patient 3's total fluid intake was 2248 c.c.; her total output was 6200 c.c. (St. Ex. 3A at 24, 46, 62, 248, 263-264).

On March 24, 1990, Dr. Sivashanker noted mild wheezing. Patient 3's total fluid intake that day was 2426 c.c.; her total output was 2425 c.c. Patient 3 received three doses of Capozide 25/50 PO and no Lasix. (St. Ex. 3A at 25, 248, 263-264).

On March 25, 1990, Dr. Sivashanker noted scattered rhonchi. He stated that a chest x-ray had demonstrated no acute changes with left costophrenic blunting and possible small effusion. He listed an impression of CHF with hyponatremia. Dr. Grodner noted "tachycardia with gallop rhythm and some rales at bases [illegible] probably in CHF." Patient 3's total fluid intake that day was 1071 c.c.; her total output was 2950 c.c. Patient 3 received three doses of Capozide 25/50 PO and one dose of Lasix 40 mg IV. (St. Ex. 3A at 26, 77, 250, 263-264).

On March 26, 1990, Patient 3's serum sodium was 121, her serum potassium was 3.3, and her serum chloride was 84. Dr. Sivashanker wrote SIADH [syndrome of inappropriate anti-diuretic hormone secretion] secondary to CVA, chest mostly clear with few rhonchi. He noted that he would try a fluid restriction with increased saline IV, and would watch for deterioration of CHF. Dr. Sivashanker ordered that the IV rate be increased to 50 c.c. per hour, and a fluid restriction of 1000 c.c. per day. Later that day, he ordered that the IV rate be decreased to KVO if Patient 3 developed chest rales. A chest x ray revealed "Some interstitial edema with small left pleural effusion" when compared to the admission chest x-ray. Patient 3's total fluid intake that day was 2256 c.c.; her total output was 1000 c.c. Patient 3 received three doses of Capozide 25/50 PO and no Lasix. (St. Ex. 3A at 26, 47, 62, 78, 250, 263-264).

On March 27, 1990, 6:00 a.m., Patient 3's serum sodium was 119, her serum potassium was 3.1, and her serum chloride was 82. At 8:30 a.m., Dr. Sivashanker wrote that Patient 3 was very confused, and her lungs were "slightly congested with rhonchi and a few rales." He stated that he would consult with internal medicine and nephrology. He also increased the potassium chloride to 40 mEq per liter, and ordered Lasix 20 mg IV NOW, and every 4 hours PRN. (St. Ex. 3A at 27, 49, 61).

At 9:10 a.m., Dr. Williams, an intensivist, called a telephone order to the nursing floor. Dr. Williams ordered that Dr. Sivashanker's order for Lasix NOW and PRN be held. He also ordered immediate labwork, including ABGs, urine electrolytes and osmolality, and serum osmolality. Furthermore, Dr. Williams ordered potassium chloride, 50 mEq in a minimum amount of normal saline, and decreased the IV rate to KVO. (St. Ex. 3A at 49).

A 9:40 a.m., Patient 3's serum osmolality was 246 [normal range 280-300]. Her urine osmolality was 467 [normal range 0-2000]. A chest x-ray revealed cardiomegaly without overt cardiac decompensation. (St. Ex. 3A at 61, 79).

Dr. Williams later noted that Patient 3 had demonstrated mild pitting edema bilaterally, no JVD, a serum sodium of 119 and a serum potassium of 3.1. Dr. Williams stated that the hyponatremia "could be secondary to SIADH and/or over-diuresis with Lasix. Results of urine/serum osmolality will help determine etiology." He recommended urine electrolytes in addition to serum and urine osmolality. He also recommended

restricting fluids and discontinuing Lasix, providing oxygen therapy, changing Zantac to Carafate, supplementing potassium, and starting digoxin. (St. Ex. 3A at 16).

At 11:00 a.m., Patient 3's serum magnesium was 0.9 [normal range 1.3-2.1]. Patient 3 received magnesium sulfate IV over three hours. At 4:40 p.m., Patient 3's serum sodium was 119, her serum potassium was 3.9, and her serum chloride was 81. At 8:50 p.m., Dr. Sivashanker ordered 500 c.c. 3% saline with 20 mEq potassium chloride to run at a KVO rate, one time. Dr. Sivashanker ordered that Patient 3 be "No Code Blue." (St. Ex. 3A at 50, 61).

On March 27, 1990, [time not noted], Dr. Grodner wrote that the hyponatremia may be diuretic induced. (St. Ex. 3A at 28).

On March 27, 1990, [time not noted], Dr. Venkataraman, a nephrologist, saw Patient 3 and listed his impressions as symptomatic hyponatremia, "most likely secondary to reduced circulating volume or at least Na-Responsive." Dr. Venkataraman cited a normal to low central venous pressure, absence of edema, small rise in serum creatinine, and urinary parameters suggesting a pre-renal state [urine osmolality of 467, fractional sodium excretion of 0.1%]. Dr. Venkataraman suspected that the condition was "diuretic induced." Dr. Venkataraman recommended discontinuing the Capozide, Lasix, and fluid restrictions. He further suggested increased oral intake, and cautious but aggressive potassium replacement. Dr. Venkataraman further noted that if the sodium could not be replenished orally, he would recommend giving isotonic saline. (St. Ex. 3A at 15).

[Note: The date on Dr. Venkataraman's consultation is unclear and could be March 22 or March 27. Some testimony at hearing was based on the assumption that Dr. Venkataraman's consultation note was written on March 22. Nevertheless, a thorough review of the record supports a conclusion that Dr. Venkataraman's consultation note was written on March 27. Examples include the following: (1) Dr. Sivashanker ordered a consultation with a nephrologist on March 27, and Dr. Venkataraman is the only nephrologist who saw Patient 3 during his admission; (2) the lab values cited by Dr. Venkataraman were not reported by the lab until March 27; and (3) Dr. Venkataraman's recommendations are consistent with orders written by Dr. Sivashanker on March 28. See Tr. at 1456-1458].

Patient 3's total fluid intake that day was 2308 c.c.; her total output was 3300 c.c. She received three doses of Capozide 25/50 PO and no Lasix. (St. Ex. 3A at 250, 267).

On March 28, 1990, 5:30 a.m., Patient 3's serum sodium was 125, her serum potassium was 3.2, and her serum chloride was 88. Dr. Sivashanker noted that Patient 3 remained lethargic, and that her lungs were "clear mostly." He acknowledged Dr. Venkataraman's consult and wrote that "she will receive Lasix only if chest fills up

with fluid.” Dr. Sivashanker discontinued the Lasix, Capozide, and fluid restriction. He also ordered Micro K orally. (St. Ex. 3A at 28, 51, 61).

Later that day, Dr. Sivashanker wrote:

Repeat 3% saline with 20 potassium chloride at TKO, give 500 c.c.  
After IV infused, repeat lytes and continue to give above IV until  
[sodium]  $\uparrow$  135.

(St. Ex. 3A at 51). In addition, Dr. Sivashanker ordered that the nurses administer Lasix 20 mg IV PRN every four hours for chest congestion. (St. Ex. 3A at 51).

On March 28, 1990, 5:00 p.m., Patient 3’s serum sodium was 125, her serum potassium was 3.7, and her serum chloride was 89. Patient 3’s total fluid intake that day was 1716 c.c.; her total output was 1000 c.c. Patient 3 received one dose of Capozide 25/50 PO and no Lasix. (St. Ex. 3A at 61, 250, 267).

On March 29, 1990, Patient 3’s serum sodium was 131, her serum potassium was 3.9, and her serum chloride was 98. Dr. Venkataraman noted that Patient 3 was doing much better. He further noted that her clinical and laboratory parameters were consistent with diuretic induced hyponatremia. He added that “if diuretics are needed in future, Lasix can be used, but I would avoid thiazides as they have a much greater propensity to cause hyponatremia.” Dr. Venkataraman signed off the case at that time. Patient 3 received no diuretics that day. (St. Ex. 3A at 29, 60, 267).

On March 30, 1990, Dr. Sivashanker noted that Patient 3’s sodium was 135, and that Patient 3 was “alert and talking sensibly.” Patient 3’s serum sodium was 134 and her serum potassium was 4.1. Patient 3 received no diuretics that day. On April 1, Patient 3 was alert and oriented and her lungs were clear. On April 2, her serum sodium was 133 and her serum potassium was 5.8. (St. Ex. 3A at 29, 52, 59, 267).

On April 4, 1990, Patient 2’s serum sodium was 135 and her serum potassium was 4.2. She was alert and talking. Patient 3 was discharged from the hospital on April 9, 1990. (St. Ex. 3A at 1, 35, 59) [Note: the discharge summary was dictated January 14, 1991, ten months after Patient 3’s discharge. Dr. Sivashanker testified that the hospital had lost the original dictation and had asked Dr. Sivashanker to re-dictate it.]

*Regarding the Board’s allegation that, on March 23, 1990, Dr. Sivashanker had noted the presence of chest congestion and ordered 80 mg of intravenous Lasix, but he had continued Patient 3 on intravenous normal saline. A chest x-ray at that time showed no evidence of significant CHF. Since there was no evidence of significant CHF, the use of diuretics had been inappropriate.*

*Dr. Miller's Testimony*

77. Dr. Miller testified that Dr. Sivashanker's care and treatment of Patient 3 had fallen below the minimal standard of care due to his use of diuretics which subjected Patient 3 to severe hyponatremia and which could have caused serious medical complications. Dr. Miller testified that Dr. Sivashanker had used excessive amounts of diuretics which had resulted in dehydration and a serum sodium of 119, which is a potentially lethal condition. (Tr. at 258-259).
78. Dr. Miller noted that, on March 21, 1990, Patient 3 had been evaluated by Dr. Graham who noted no CHF on the chest x-ray. Dr. Miller testified that he interpreted Dr. Graham's comments to indicate that the Patient 3 did not have CHF at that time, despite symptoms noted over the course of several days which could have been indicative of CHF. These symptoms included rales, rhonchi, wheezing, moist cough, diminished breath sounds, dyspnea with exertion, and trace ankle edema. (Tr. at 581-592, 597).  
  
Dr. Miller further testified that the one time dose of Lasix on March 22, 1990, had been appropriate for Patient 3's deteriorating condition. (Tr. at 632-633). Nevertheless, Dr. Miller noted that over the next several days, Dr. Sivashanker ordered Capozide, with 50 mg of hydrochlorothiazide, three times a day, in addition to occasional doses of Lasix. Dr. Miller stated that Patient 3 had received 600 mg of hydrochlorothiazide and 100 mg of Lasix over a four day period. During that time, her serum sodium dropped to 119, a critical level. Moreover, between March 22 and March 27, Patient 3 suffered a net fluid loss of 11.3 liters, which, Dr. Miller testified, is "a sizable amount of fluid." Dr. Miller concluded that the fluid loss had dehydrated Patient 3, and the salt losses associated with the diuretics had resulted in the serum sodium dropping to 119. (Tr. at 264-266).
79. Dr. Miller testified that there is no evidence in the medical record that Patient 3 had been suffering from CHF between March 22 and March 27. Dr. Miller noted that there had been minimal effusions mentioned on chest x-rays, but the overall fluid balance refuted a finding of CHF. Moreover, Dr. Miller stated that, for some people who have significant heart disease, such findings are "really a baseline phenomenon." With such people, it is impossible to eliminate all traces of water, and attempting to do so puts the patient at serious risk for dehydration. (Tr. at 266-267).
80. Dr. Miller testified that he agreed with Dr. Venkataraman's March 27, 1990, consultation report, which suggested that diuretics had caused Patient 2's dehydration and hyponatremia. Dr. Miller further stated that, by giving salt and water, the problems could have been averted. (Tr. at 274-275).
81. Dr. Miller testified that, even if he had determined that Patient 3 had had CHF during this time period, he would have criticized Dr. Sivashanker's use of diuretics.

Dr. Miller testified that using Lasix would have been appropriate, without the three times daily use of Capozide. Dr. Miller testified that the use of the two agents concurrently was inappropriate, because using Lasix alone provides a much better effect, and using the thiazide three times a day had subjected Patient 3 to the unnecessary risks of hypothermia. (Tr. at 571, 573).

82. Dr. Miller testified that Patient 3 had been harmed by Dr. Sivashanker's use of excessive diuretics since Patient 3 had become confused and lethargic as a result of hyponatremia. (Tr. at 754-755).
83. Dr. Miller was presented with testimony of Dr. Sivashanker, indicating that the cause of Patient 3's hyponatremia had SIADH. Dr. Miller defined SIADH as "a clinical syndrome where the brain puts out a substance called ADH, which causes water retention in an inappropriate fashion." Dr. Miller testified that SIADH can be caused by a variety of factors including lung cancer, intestinal cancer, cerebral hemorrhage, and CVA. (Tr. at 647-648, 662).

Dr. Miller testified that SIADH is a well known complication of a stroke. Nevertheless, Dr. Miller testified that SIADH is a diagnosis that can be made only after eliminating a number of other possibilities. More specifically, Dr. Miller testified that, in the presence of dehydration and diuretic use, one can not make a diagnosis of SIADH. (Tr. at 267-271, 662).

Dr. Miller testified that SIADH may be considered in a patient who has suffered a CVA and who has a low serum sodium. Dr. Miller further testified that the main indicators are the serum sodium, urinary sodium and the urine osmolality. (Tr. at 663-664). Nevertheless, Dr. Miller testified that Patient 3's BUN rose from 5 to 15, and was an indication of a pre-renal state due to dehydration, rather than SIADH. Moreover, Dr. Miller testified that SIADH is a euvolemic or hypervolemic condition. (Tr. at 67-672).

#### ***Dr. Sivashanker's Testimony***

84. Dr. Sivashanker testified that, upon Patient 3's presentation to the ER on March 15, the ER physician had appropriately diagnosed CHF and treated Patient 3 with Lasix. In response to the Lasix, Patient 3 had diuresed profusely, and her cough and shortness of breath had improved. (Tr. at 120).

Dr. Sivashanker acknowledged that x-rays taken after Patient 3's diuresis had shown cardiomegaly without overt CHF. Nevertheless, Dr. Sivashanker testified that Patient 3 had continued to have CHF. Dr. Sivashanker noted that Patient 3 had had a "persistent congested cough," ankle edema, cardiomegaly, and minimal JVD, all of which are signs of CHF. Despite the fact that the chest x-ray had not shown definite

evidence of CHF, Dr. Sivashanker believed that there had been some cephalization of the vasculature which, Dr. Sivashanker stated, is a subtle finding which the radiologist may have missed. Moreover, Dr. Sivashanker testified that Patient 3's  $pO_2$  had been 69;  $pCO_2$  40; and pH 7.46, which ruled out any primary lung disease. Finally, Dr. Sivashanker testified that Patient 3 was a non-smoker with no history of lung disease. (Tr. at 120-124).

In addition, Dr. Sivashanker stated that Patient 3 was of advanced age and had chronically low blood pressure and chronic hyponatremia. Dr. Sivashanker stated that, in light of these factors and Patient 3's unstable blood pressure, Dr. Sivashanker had discontinued hydrochlorothiazide, ordered Lasix on an as-needed basis for episodes of pulmonary congestion and edema, changed Vasotec to a short acting Ace inhibitor, and discontinued Corgard, a negative inotropic beta-blocker which is seldom used for heart failure. (Tr. at 120-121, 996).

85. Dr. Sivashanker testified that on March 23, 1990, he had ordered diuretics to treat Patient 3's CHF. In support of his conclusion that Patient 3 had been suffering CHF, Dr. Sivashanker pointed to the nurses' notes documenting wheezing, rhonchi, and rales. Dr. Sivashanker also cited the chest x-ray which had revealed left costophrenic blunting and possible small effusion. Finally, Dr. Sivashanker cited Patient 3's  $pO_2$  of 66 on room air, and a  $pCO_2$  of 33, which suggested to Dr. Sivashanker that Patient 3 had been "blowing off the carbon dioxide by rapid breathing" due to the CHF. (Tr. at 980-987, 994-995).
86. Dr. Sivashanker testified that Patient 3 had developed an infarct on March 19, 1990, which was evidenced on a CT scan. Dr. Sivashanker stated that the infarct had precipitated the onset of SIADH, which had been the cause of the acute hyponatremia. Dr. Sivashanker added that the diagnosis of SIADH could be confirmed by the sodium levels, the serum and urine osmolality and the uric acid levels. (Tr. at 121, 129-130).

Dr. Sivashanker testified that that when a patient has SIADH, the patient's fluid volume is normal, as is the uric acid and BUN. In addition, sodium and plasma osmolality are low. (Tr. at 163-164).

87. Dr. Sivashanker testified that he had requested a nephrology consultation with Dr. Venkataraman on March 22, 1990. (Tr. at 130-131) [Note, however, the medical record suggests that the consultation with Dr. Venkataraman was obtained on March 27 rather than March 22, 1990. If the consultation was March 27, then much of Dr. Sivashanker's testimony regarding Dr. Venkataraman's opinion is inconsistent with the record.] Regarding Dr. Venkataraman's recommendations, Dr. Sivashanker testified as follows:

- On March 23, Dr. Sivashanker ordered Capozide in spite of Dr. Venkataraman's suggestions and despite the fact that Patient 3's sodium level was only 123. Dr. Sivashanker acknowledged that the hyponatremia may have been diuretic induced. Nevertheless, Dr. Sivashanker opined that the diuretics causing the hyponatremia would have been those ordered by Patient 3's family physician prior to her hospitalization not those ordered by Dr. Sivashanker. (Tr. at 131-132).
  - Dr. Sivashanker testified that he had ordered Capozide in spite of Dr. Venkataraman's suggestions because, when Dr. Venkataraman had seen Patient 3, her sodium level had been 123, not yet a critical level. Therefore, Dr. Venkataraman could not have appreciated the SIADH. (Tr. at 131-132).
  - Dr. Sivashanker testified that he had ordered Capozide in spite of Dr. Venkataraman suggestions because, as a nephrologist, Dr. Venkataraman had been concerned only about Patient 3's sodium. Dr. Sivashanker, however, had been concerned about the sodium as well as Patient 3's propensity for cardiac failure. He stated that Patient 3 had had a pleural effusion, as demonstrated on chest x-ray by left CP angle blunting and small effusion. Therefore, Dr. Sivashanker had needed to use strong diuretics to keep Patient 3 out of "full CHF." (Tr. at 132-134, 136-137).
  - Dr. Sivashanker testified that he "totally disagreed" with Dr. Venkataraman's conclusions. Dr. Sivashanker testified that signs of volume depletion include a low sodium and a low plasma osmolality, with a high uric acid and BUN. Dr. Sivashanker testified that Patient 3 did not fit the picture of volume depletion. (Tr. at 163).
  - Finally, Dr. Sivashanker testified that Dr. Venkataraman had made an error by noting certain factors, including a small rise in creatinine and urinary parameters, and in concluding that Patient 3 was in a pre-renal state. Dr. Sivashanker argued that, in order to diagnose a pre-renal state, the BUN must be 25 or more. Yet Patient 3's BUN was only 10. Therefore, because Dr. Venkataraman was "wrong with one thing, [Dr. Sivashanker] did not want to accept any of his explanations." (Tr. at 135-136).
88. Dr. Sivashanker stated that, because he had not agreed with Dr. Venkataraman's conclusions, he had consulted with Dr. Williams on March 27. Dr. Sivashanker acknowledged that Dr. Williams had also recommended discontinuing the Lasix. Nevertheless, Dr. Sivashanker had chosen to disregard Dr. Williams' recommendations as well. Dr. Sivashanker explained that Dr. Williams, like Dr. Venkataraman, had not been concerned with Patient 3's heart failure but had only

been concerned with her sodium. Therefore, Dr. Sivashanker, as the primary physician, could better see the total picture. (Tr. at 137-140, 158-161).

89. Dr. Sivashanker testified that he had ordered 500 c.c. of 3% sodium chloride on March 28. Dr. Sivashanker testified that, whenever a patient with CHF receives diuretics, there will be some hyponatremia. Therefore, normal saline is used to correct the hyponatremia while the Lasix is used to treat the heart failure. Dr. Sivashanker explained that, with the high concentration of sodium in the replacement fluids, the patient will retain more sodium despite the use of Lasix. (Tr. at 161, 164, 168-169).

Dr. Sivashanker opined that the SIADH had been “appropriately treated with saline and Lasix as needed. She responded nicely with a return of sodium to a baseline of 134 and was discharged in an improved medical condition.” (Tr. at 121).

90. Dr. Sivashanker acknowledged that he had not mentioned SIADH either in the discharge summary or the DRG sheet contained in the medical record. (Tr. at 172).

***Dr. Mahizhnan’s Testimony***

91. Dr. Mahizhnan testified that Dr. Sivashanker’s use of Lasix on March 23, 1990, had been appropriate. (Tr. at 1135).
92. Regarding SIADH, Dr. Mahizhnan testified that the basic physiology of SIADH is that the patient has an excess of anti-diuretic hormone [ADH]. Because there is an excess of ADH, the body retains fluid. The fluid however, is not intravascular, but extravascular, which explains why the patient does not go into severe CHF. It also explains why the patient requires diuretics to remove the excessive fluid. At the same time, sodium leaves the intravascular spaces with the fluid, and the patient becomes hyponatremic. Accordingly, the patient requires saline to increase the serum sodium. (Tr. at 1142).

Dr. Mahizhnan further testified that Patient 3 had developed SIADH on approximately March 27, ten days after suffering a stroke. Dr. Mahizhnan testified that the urine osmolality, serum osmolality, uric acid level, and BUN/creatinine along with Patient 3’s mental confusion, lethargy and weakness all supported the diagnosis of SIADH. Dr. Mahizhnan explained that in SIADH, the patient will present with hyponatremia, a urine osmolality higher than the serum osmolality, and normal to very low uric acid, BUN and creatinine. On the other hand, if hypovolemia is the result of diuretics or a pre-renal state, the BUN and creatinine will be high. Dr. Mahizhnan testified that that was not the case with Patient 3. Dr. Mahizhnan added that, when a patient is taking diuretics, it becomes more confusing, but it is not difficult to diagnose SIADH with the proper laboratory studies. (Tr. at 1135-1137, 1141).

Dr. Mahizhnan further testified that the treatment for SIADH is Lasix and saline. (Tr. at 1141).

***Dr. Beaver's Testimony***

93. Dr. Beaver testified that it is likely that Patient 3 was admitted with CHF since an S3 gallop had been detected, and her physical symptoms had been consistent with CHF. Dr. Beaver further noted that the daily intake and output records indicate that Patient 3 had been excreting significantly more fluid than she had been taking in; therefore, since she did not go into shock or have a drop in blood pressure, one could assume that she had had excessive fluid to begin with. (Tr. at 1242-1249).
94. Dr. Beaver further testified that the amount of diuretic Dr. Sivashanker prescribed for Patient 3 in the form of Capozide had not been excessive. Moreover, Dr. Beaver testified that a patient with CHF, even after acute symptoms have resolved, requires continuous diuretic therapy. Otherwise, the acute symptoms will recur. (Tr. at 1252-1254).
95. Dr. Beaver testified that, on a number of occasions, he has given diuretic with saline. He stated that, in a patient who has CHF and hyponatremia, simply providing saline to increase the serum sodium puts the patient at risk for an exacerbation of CHF. Dr. Beaver concluded that Dr. Sivashanker had acted appropriately in treating Patient 3. (Tr. at 1175-1178).

***Dr. Chinn's Testimony***

96. Dr. Chinn testified that the use of Lasix in a patient with a diagnosis of CHF is appropriate even if there are no signs of edema on a chest x-ray. Nevertheless, Dr. Chinn testified that the chest x-ray of March 26 indicated interstitial edema and a small pleural effusion which would support a diagnosis of CHF and would warrant the use of diuretics. (Tr. at 1426).
97. Dr. Chinn further testified that Patient 3 had probably been suffering a combination of SIADH and over-diuresis. Dr. Chinn further testified that Dr. Sivashanker's treatment of Patient 3 had provided an appropriate correction of her SIADH. (Tr. at 1433).

Dr. Chinn noted that, by March 27, the BUN and the creatinine had both risen slightly, but were both still normal. Dr. Chinn testified that in SIADH, the BUN and creatinine would remain normal. Nevertheless, the fact that the levels were rising was an indication of volume depletion and not necessarily a sign of SIADH. (Tr. at 1437-1438).

Dr. Chinn testified that hyponatremia can be caused both by over-diuresis and by SIADH. Nevertheless, Dr. Chinn testified that over-diuresis would not normally cause a sodium to drop as low as it did in this case. Dr. Chinn further testified that he would

have expected a higher BUN and creatinine if over-diuresis and volume depletion had been the sole cause of the hyponatremia. Therefore, it is reasonable to assume that SIADH played some role. (Tr. at 1449-1453).

*Regarding the Board's allegation that Dr. Sivashanker's order of March 28, 1990, to give sodium chloride intravenously until Patient 3's serum sodium level rose above 135, was inappropriate, as it subjected Patient 3 to the risk of CHF and neurological complications*

***Dr. Miller's Testimony***

98. Dr. Miller testified that Dr. Sivashanker had appropriately ordered normal saline, 0.9% sodium chloride, on March 26 and 27, 1990. Moreover, Dr. Miller testified Dr. Sivashanker had appropriately switched to 3% sodium chloride. Nevertheless, in the time frame of 1990, there was great concern regarding a too rapid correction of serum sodium. Therefore, the 3% sodium chloride solution needed to be given very carefully and very slowly. (Tr. at 276-277, 655).

Dr. Miller stated that Dr. Sivashanker's order to give 3% sodium chloride until the serum sodium reached 135 was inappropriate. Dr. Miller testified that the order had put Patient 3 at risk of receiving too much sodium and chloride. Dr. Miller testified that it would have been appropriate to use 3% sodium chloride until the serum sodium reached a safe level, such as 125, and then to switch to a more conventional solution, such as 0.9% sodium chloride. (Tr. at 277-278).

***Dr. Sivashanker's Testimony***

99. Dr. Sivashanker testified that he had intended the nurses to infuse IV fluid of 0.9% sodium chloride, rather than 0.3% sodium chloride, until the serum sodium reached 135. Dr. Sivashanker further testified that he had realized, at that time, there had been some confusion about the order as written. Therefore, at 2:30 p.m., he changed the IV fluids to one liter 0.9% sodium chloride with 40 mEq of potassium chloride at 40 c.c. per hour. Nevertheless, Dr. Sivashanker testified that he now realizes that ordering an IV of either 0.9% or 3% sodium chloride to be infused until a certain serum sodium level is reached, and leaving that decision to the discretion of the nurses, had been inappropriate. (Tr. at 1002-1009).

***Dr. Chinn's Testimony***

100. Dr. Chinn testified that 3% sodium chloride, hypertonic saline, is only given in rare circumstances to correct very severe hyponatremia, which was the case with Patient 3. Dr. Chinn further testified that hypertonic saline is usually given until the sodium rises to about 125 or 127. He explained that the hypertonic saline is used to get the patient out of danger, not to correct the sodium to normal. The danger of too much

hypertonic saline is fluid overload. Therefore, it is unusual to provide hypertonic saline until the sodium rises until 135. Dr. Chinn concluded that the order had been inappropriate in this case. (Tr. at 1454-1456).

*Regarding the Board's allegation that it had been inappropriate for Dr. Sivashanker to direct the nurses to use intravenous Lasix for chest congestion on an as-needed basis.*

***Dr. Miller's Testimony***

101. Dr. Miller testified that it had been below the minimal standard of care for Dr. Sivashanker to direct the nurses to use Lasix at their discretion four times a day for "congestion." Dr. Miller testified that, first of all, chest "congestion" is an ambiguous term which can be interpreted a number of ways. Moreover, if a nurse believes that a patient is demonstrating indications for diuretic therapy, the nurse should call the physician at that time, not "four times a day." Furthermore, as previously noted, Dr. Miller testified that it is not acceptable to write an "as needed" order for the administration of Lasix; the physician should determine when Lasix is needed, not the nurse. Finally, Dr. Miller testified that a patient in this situation should be evaluated by the physician on a daily basis to determine the need for anti-diuretic therapy, and a standing order is inappropriate. (Tr. at 260-263).

***Dr. Sivashanker's Testimony***

102. Dr. Sivashanker testified that, looking back, he realizes that it is inappropriate to direct the nurses to administer Lasix on an as needed basis. Dr. Sivashanker stated that such an order allows "room for error." Nevertheless, Dr. Sivashanker noted that there was no evidence of miscommunication with the nurses, and there was no error in the administration of the medication as a result of his order. (Tr. at 977-978).

***Dr. Mahizhnan's Testimony***

103. Dr. Mahizhnan testified that physicians generally do not order Lasix on a PRN basis, but that it sometimes does happen. (Tr. at 1128).

**PATIENT 4**

Patient 4, a 68 year old male, first reported to Dr. Sivashanker's office in March 1984. Patient 4 presented with a history of hypertension, COPD, possible myocardial infarction, and anemia. (St. Ex. 4 at 2). Dr. Sivashanker continued to care for Patient 4 over the years. (St. Ex. 4).

On February 27, 1990, Patient 4 presented to Dr. Sivashanker's office with complaints of chest pain, unrelenting since 5:00 a.m.; pallor; and coughing. Dr. Sivashanker diagnosed "rule out

pneumonia” with acute respiratory distress. Dr. Sivashanker arranged for Patient 4’s admission to the hospital. (St. Ex. 4 at 17).

#### **PATIENT 4’S FEBRUARY 27, 1990, HOSPITAL ADMISSION**

##### *Basis for the Admission; Hospital Course*

104. Patient 4 was admitted to St. Ann’s Hospital on February 27, 1990, under the care of Dr. Sivashanker. In his History and Physical, Dr. Sivashanker noted that Patient 4’s primary complaints were increasing nonproductive cough, respiratory distress, increased respiratory rate, increased pallor of the skin, and “pain in the chest wall due to coughing.” Dr. Sivashanker’s diagnoses included acute respiratory distress and cough secondary to COPD with acute bronchitis; hypoxia; and hypertension, uncontrolled. (St. Ex. 4A at 7-8).

Dr. Sivashanker admitted Patient 4 to a telemetry unit. Dr. Sivashanker ordered EKGs twice daily for two days, CK and LDH daily for two days, oxygen at two liters per minute, arterial blood gases, activity as tolerated, and a consultation with Dr. Grodner. Patient 4’s CK was 539 [normal range 0-235]; his LDH was 236 [normal range 100-250]; and his MB was 3.6 [normal 0-2.2; borderline 2.3-5.6; elevated >5.6]. The EKG revealed an intraventricular conduction defect. Dr. Sivashanker did not mention these results in his progress notes. (St. Ex. 4A at 16, 23, 30).

That evening, Patient 4 complained of acute dyspnea and midsternal chest pain. The nurse further noted that Patient 4’s CK had been elevated, and paged Dr. Sivashanker. In addition, the nurse administered nitroglycerin sublingually three times without relief. Shortly after administration of nitroglycerin, Patient 4’s blood pressure dropped to 90/60, and he was pale, dusky, and diaphoretic. Dr. Sivashanker ordered breathing treatments PRN, Nitropaste four times daily, and STAT ABGs. (St. Ex. 4A at 17, 46).

On February 28, 1990, Dr. Sivashanker noted that Patient 4 had developed acute respiratory distress and severe chest discomfort during the night. On chest examination, Dr. Sivashanker noted that Patient 4’s air exchange had improved. A chest x-ray revealed that Patient 4’s lungs were “slightly hyperexpanded,” but otherwise normal. Patient 4’s CK was 483; his LDH was 215; and his MB was 11.3. The EKG revealed non-specific ST-T wave changes. Dr. Sivashanker did not mention these results in his progress notes. (St. Ex. 4A at 11, 23, 27, 31).

On March 1, 1990, Dr. Sivashanker noted that Patient 4’s chest was clear. He also noted that he would order a repeat arterial blood gas and, if normal, he would plan discharge. Patient 4’s CK was 503; his LDH was 224; and his MB was 8.6. The nurses’ notes indicate that the enzyme results were called to Kathleen at

Dr. Sivashanker's office. Dr. Sivashanker did not mention these results in his progress notes. (St. Ex. 4A at 12, 23, 55).

On March 2, 1990, Dr. Sivashanker noted that Patient 4 had been lethargic and short of breath earlier that day. He further noted that Patient 4 continued to feel weak and tired. On March 3, Dr. Sivashanker noted that Patient 4 had been feeling better, but that Patient 4 continued to feel weak and tired. (St. Ex. 4A at 13, 14).

On March 4, 1990, Dr. Sivashanker noted that Patient 4's breathing was fair, and his air entry had improved. Dr. Sivashanker also noted that Patient 4's cardiac rhythm was normal sinus. On March 5, Dr. Sivashanker noted that Patient 4's chest was clear and his cardiac rhythm regular. Dr. Sivashanker ordered Patient 4 discharged from the hospital. (St. Ex. 4A at 14, 20).

In his discharge summary, Dr. Sivashanker wrote that Patient 4 had had chronic end-stage COPD with increasing respiratory distress, pallor, and pain in the chest wall due to coughing. Dr. Sivashanker stated that Patient 4 "did not have any cardiac-type of pain." Dr. Sivashanker noted that Patient 4's CK and MB had been elevated, "suggestive of possible non-transmural MI." Dr. Sivashanker's discharge diagnoses included severe respiratory distress secondary to acute exacerbation of COPD with acute bronchitis; and elevated MB, possible subendocardial myocardial infarction. (St. Ex. 4A at 5-6).

*Regarding the Board's allegation that, on February 27, 1990, Patient 4's creatine kinase (CK) level was elevated and the myocardial band (MB) fraction was 3.6. On February 28, 1990, Patient 4's CK level remained elevated and the MB had risen to 11.3. On March 1, 1990, Patient 4's CK level remained elevated and the MB was 8.6. Dr. Sivashanker performed three electrocardiograph tests during this time period which showed no Q waves or ST segment elevation. Patient 4 complained of chest pain during this time period. Despite Patient 4's symptoms and laboratory reports, Dr. Sivashanker failed to diagnose and treat appropriately Patient 4's acute myocardial infarction.*

#### ***Dr. Miller's Testimony***

105. Dr. Miller testified that Dr. Sivashanker's care and treatment of Patient 4 had departed from the standards of care in that Dr. Sivashanker had failed to diagnose Patient 4 as having suffered a subendocardial myocardial infarction. Dr. Miller testified that, during the hospitalization of February 27, 1990, Patient 2 had had elevated cardiac enzymes and a normal EKG, which was indicative of a subendocardial myocardial infarction. Dr. Miller acknowledged that Dr. Sivashanker had written "possible subendocardial MI" in his discharge diagnoses. Nevertheless, Dr. Miller criticized Dr. Sivashanker's care and treatment of Patient 4 because Dr. Sivashanker had not changed Patient 4's therapy in any way after the enzyme elevation was confirmed by

the lab. Dr. Miller explained that, even though a subendocardial myocardial infarction is less severe than a transmural myocardial infarction, it should have been treated as an acute cardiac event. (Tr. at 285-291, 686, 691-692).

106. Dr. Miller further noted that Patient 4's complaints may have been considered atypical for a cardiac event. Nevertheless, Dr. Miller testified that "typical classic chest pain in people with heart attacks is probably the exception rather than the rule." A physician must assume that chest pain is cardiac in origin until proven otherwise. Therefore, when the MB came back elevated, the burden of proof falls to the physician to prove that it is not cardiac pain. (Tr. at 291-294).

***Dr. Sivashanker's Testimony***

107. Dr. Sivashanker testified that Patient 4 had not been a candidate for having cardiac problems. Dr. Sivashanker testified that Patient 4 was a 74 year old male who suffered from anemia and chronic COPD from a history of heavy smoking. Nevertheless, Dr. Sivashanker testified that Patient 4 was an unlikely candidate for an acute myocardial infarction, because his cholesterol levels were excellent; his CK was only slightly elevated; and he did not have a significant family history of coronary artery disease. Moreover, his EKGs were normal. (Tr. at 174-176).

Nevertheless, despite the fact that Patient 4 was an unlikely candidate for cardiac disease, Dr. Sivashanker testified that he had treated Patient 4 as a "rule out MI." Dr. Sivashanker had ordered bedrest, telemetry, nitroglycerin, aspirin, and Cardizem. (Tr. at 174-176). [Note: the medical record indicates that Dr. Sivashanker ordered activity as tolerated, and the nurses' notes indicate that Patient 4 spent time out of bed.]

108. Dr. Sivashanker testified that he had not been aware of the elevated MB until the day after Patient 4 had been discharged from the hospital. Dr. Sivashanker stated that no one had notified him of the results prior to the report being issued. (Tr. at 175-176).

Dr. Sivashanker acknowledged, however, that the nurses' notes reflect the elevated MB of 8.6 on March 1, 1990, and that his office had been notified of the results. Moreover, Dr. Sivashanker acknowledged that, as a physician, he had had a responsibility to monitor the enzyme levels as they were drawn. (Tr. at 178, 180-181).

109. Moreover, Dr. Sivashanker testified that, despite his failure to learn of the MB when it was drawn, the elevated level would not have made any difference in his management of Patient 4. Dr. Sivashanker testified that this MB had been low, only 2.34, and other enzymes, i.e., the SGOT, LDH and SGPT had been normal. Dr. Sivashanker concluded that the elevated MB had been a "red herring." (Tr. at 179, 182).

110. In addition, Dr. Sivashanker later testified that he had made a diagnosis of acute myocardial infarction on the basis of the elevated MB. When asked where he had documented that in the record, Dr. Sivashanker referred to the hospital's computerized DRG sheet. When reminded that the DRG sheet is created by the hospital, Dr. Sivashanker referred to his discharge summary, where he had diagnosed "possible subendocardial myocardial infarction." Dr. Sivashanker admitted that he had not mentioned the possibility of a subendocardial myocardial infarction any place else in the medical record. (St. Ex. 4A at 3, 6; Tr. at 1010-1012, 1024).
111. Dr. Sivashanker testified that he had treated Patient 4 as if he had suffered a myocardial infarction, despite the fact that he had not yet diagnosed him as having had a myocardial infarction. Dr. Sivashanker stated that the things he ordered in consideration of the possibility of a myocardial infarction included the telemetry bed, serial cardiac enzymes, serial EKGs, arterial blood gases, oxygen, aspirin, nitroglycerin, and Cardizem. Nevertheless, Dr. Sivashanker testified that he had allowed Patient 4 activity as tolerated because Patient 4 had not presented with complaints of chest pain. [Note: both Dr. Sivashanker's office records and the hospital record indicate that Patient 4 had been suffering chest pain since 5:00 a.m. on February 27, 1990.] (Tr. at 1015).
112. Dr. Sivashanker further testified that he had not considered thrombolytic therapy for Patient 4 because Patient 4 had not presented with classic chest pain or an ST elevation. (Tr. at 1030-1033). When asked if Patient 4's complaints of chest pain, respiratory distress, increased respiratory rate, and pallor could have been symptoms of a myocardial infarction, Dr. Sivashanker stated that they could not. Dr. Sivashanker explained that Patient 4 had complained of coughing with his chest wall pain which is not a classic presentation for a myocardial infarction. (Tr. at 1021-1023).
113. Dr. Sivashanker testified that his discharge orders for Patient 4 had been "no heavy lifting, outpatient treadmill testing and further cardiac work-up" which had been appropriate discharge orders for Patient 4 because "he didn't have any complications or any problems." (Tr. at 1029-1030).
114. Later, Dr. Sivashanker testified that, despite all of his prior testimony, he did not think Patient 4 had had a myocardial infarction at all. (Tr. at 1025-1027). Dr. Sivashanker explained that he had had other explanations for the elevated MB, such as skeletal muscle damage from increased respiratory muscle use. Dr. Sivashanker admitted, however, that he had not documented these considerations in the record. Dr. Sivashanker further testified that, with a myocardial infarction, other enzymes will also be elevated, such as the LDH, ALT, or AST. Dr. Sivashanker testified that none of these were present in Patient 4's case. (Tr. at 1031).
115. Finally, even later, when discussing Patient 4's hospital admission in December 1990, Dr. Sivashanker acknowledged that, at that time, he had referred to Patient 4's previous

acute myocardial infarction. Dr. Sivashanker testified that he may have been referring to the events of February 1990 or other events. He could not remember. (Tr. at 1036).

***Dr. Mahizhnan's Testimony***

116. Dr. Mahizhnan testified that Patient 4 had not had a myocardial infarction in February 1990. Dr. Mahizhnan based his opinion on the fact that, to diagnose a myocardial infarction, there must be some EKG changes and Patient 4 did not demonstrate any changes on his EKG. Nevertheless, Dr. Mahizhnan stated that he did not know the reason for the elevated MB, and suggested that it may have been a false positive or the result of a muscle injury. (Tr. at 1135, 1147-1148).

Dr. Mahizhnan further testified that, even if Patient 4 had suffered a subendocardial myocardial infarction, Dr. Sivashanker's treatment had been appropriate because Dr. Sivashanker had provided aspirin and a calcium channel blocker. Dr. Mahizhnan further testified that there had been no indication for thrombolytic therapy, and there had been no need to do any invasive procedures, such as angioplasty or emergency cardiac catheterization. (Tr. at 1146-1147).

***Dr. Beaver's Testimony***

117. Dr. Beaver testified that the fact that the MB had changed implies that there may have been a small amount of cardiac damage. (Tr. at 1258-1259).

***Dr. Sivashanker's Post Discharge Office Records***

118. On September 17, 1990, Patient 4 was hospitalized. The reason for the hospitalization was not documented, but at the time of the hospitalization, his a CK was 414 [normal range 0-235] and his MB was 3.9 [normal 0-2.2; borderline 2.3-5.6; elevated >5.6]. (St. Ex. 4 at 110).

**PATIENT 4'S DECEMBER 10, 1990, HOSPITAL ADMISSION**

***Basis for the Admission; Hospital Course***

119. Patient 4 presented via ambulance to the ER at Grady Memorial Hospital on December 10, 1990, at 8:26 a.m. Patient 4 complained of shortness of breath and chest discomfort, but denied "chest pain." Patient 4 received one nitroglycerin sublingually, which "almost" relieved his chest discomfort. St. Ex. 4D at 9-11). At 9:04 a.m., Patient 1's CK was 387 [normal range 61-224]; and his LDH was 185 [normal range 94-172]. The EKG was interpreted as "normal sinus rhythm with a tachycardic rate and PACs. Cor pulmonale. Clockwise rotation. Probable atrial

overload as well.” The ER diagnosis was rule out myocardial infarction, COPD. (St. Ex. 4D at 9-11, 31, 46). [Note: the hospital record contains special cardiac profile reports, but they are illegible. See St. Ex. 4D at 36-39].

Patient 4 was admitted to the intensive care/cardiac care unit under the care of Dr. Sivashanker. Dr. Sivashanker signed the pre-printed “CCU Arrhythmia Orders” and the pre-printed “Routine ICU/CCU Admission Orders.” (St. Ex. 4D at 50-51). At 6:21 p.m., his CK was 349; his MB was 13, relative index 3.7; and his LDH was 185. (St. Ex. 4D at 31).

The nurses’ notes indicate that Patient 4 had an episode of midsternal chest pressure and dyspnea at 2:00 a.m. After administration of sublingual nitroglycerin, Patient 4’s blood pressure dropped and his sinus rhythm converted to a junctional rhythm. The family was called. (St. Ex. 4B at 9-11, 31, 46, 72).

Dr. Sivashanker wrote that Patient 4 had complained of shortness of breath and pressure-like substernal chest pain without radiation. Dr. Sivashanker further noted that Patient 4 had felt that his pain was in his lungs. Dr. Sivashanker stated that the EKG had not shown any acute changes and that the MB was elevated “secondary to MI.” Moreover, Dr. Sivashanker wrote that the TPA [thrombolytic therapy] criteria had not been met, and that sublingual nitroglycerin had caused a drop in Patient 4’s blood pressure. Dr. Sivashanker noted that he would carefully use topical nitroglycerin. (St. Ex. 4D at 12-13).

On December 11, 1990, Dr. Sivashanker noted that the CK was 259, the LDH 148, and the MB 3. Patient 4 had not complained of chest pain or shortness of breath. The EKG revealed nonspecific ST and T wave changes. (St. Ex. 4D at 14, 31-33, 47). Later that day, Dr. Sivashanker noted that he had discussed Patient 4’s condition with Patient 4 and Patient 4’s ex-wife. Dr. Sivashanker wrote as follows:

[Patient 4] does not want any CPR or respirator to be used in case of emergency. However, he agreed to endotracheal intubation and/or medications for cardiac arrhythmias in case his condition should worsen. [Patient 4 was] made No Code Blue except for endotracheal intubation and medications for cardiac arrhythmias.

(St. Ex. 4D at 15). Dr. Sivashanker transferred Patient 4 to the telemetry unit, and prescribed Voltaren for “chest wall pain.” (St. Ex. 4D at 53-56).

On December 12, 1990, Dr. Sivashanker noted Patient 4 had had a “rough night.” Dr. Sivashanker stated that Patient 4 had coughed all night and that his chest wall was sore. Patient 4’s CK was 431. Again, the EKG revealed nonspecific ST and T wave changes. Dr. Sivashanker questioned pericarditis. (St. Ex. 4D at 15, 30).

On December 15, 1990, Dr. Sivashanker noted that Patient 4 had had “some vague substernal pain” radiating to the back with epigastric tenderness. Dr. Sivashanker added Carafate to Patient 4’s medications. (St. Ex. 4D at 17).

On December 17, 1990, Dr. Sivashanker discharged Patient 4 to home. Dr. Sivashanker diagnosed acute myocardial infarction, COPD, and acute bronchitis. He instructed Patient 4 to “resume previous activity” other than “climbing stairs and physical activity.” Dr. Sivashanker planned a stress test and Holter monitor. (St. Ex. 4D at 19, 60).

*Dr. Sivashanker’s Post Discharge Office Records*

120. Patient 4 was evaluated by Dr. Beaver on January 28, 1991. Dr. Beaver listed an impression of arteriosclerotic heart disease with apparent recent non-Q-wave myocardial infarction. Dr. Beaver performed a cardiac catheterization which demonstrated good left ventricular function, and an ejection fraction of approximately sixty percent. In addition, the cath revealed “a lesion of perhaps 60 to 80% involving a relatively small diagonal branch of the LAD but otherwise no fixed coronary artery lesions.” (St. Ex. 4 at 141-143, 165-166).
121. During a September 1991 hospital history and physician, Dr. Sivashanker noted in past medical history that Patient 4 had had a myocardial infarction in 1961. Dr. Sivashanker did not mention the events of February and December 1990. (Tr. at 155).

*Regarding the Board’s allegation that, on December 10, 1990, Patient 4 had been admitted to the hospital with shortness of breath and chest pain. On admission, Patient 4’s CK level was twice the normal level, and the MB was 13. Patient 4 was admitted to the Intensive Care Unit and treated with a nitroglycerin patch and Cardene. Dr. Sivashanker did not request a cardiology consultation. Moreover, Dr. Sivashanker failed to treat Patient 4 appropriately, or Dr. Sivashanker failed to document Dr. Sivashanker’s rationale for conservative treatment of Patient 4*

***Dr. Miller’s Testimony***

122. Dr. Miller testified that Dr. Sivashanker’s care and treatment of Patient 4 during the December 10, 1990, hospitalization had been below the accepted standard of care. Dr. Miller testified that this was the second myocardial infarction Patient 4 had experienced within ten months. Moreover, Patient 4 had been placed in the ICU, required a nitroglycerin patch, and suffered tachycardia, possibly of ventricular origin. Dr. Miller stated that Patient 4’s cardiac condition was complicated, and required more than what an internist in a community hospital could offer. Dr. Miller concluded

that a cardiologist should have been consulted and more aggressive management should have been initiated. (Tr. at 294-297, 686, 694).

123. Finally, Dr. Miller testified that, if Dr. Sivashanker had had reason to treat Patient 4 conservatively, such as Patient 4's request that nothing further be done, the reasons for treating the patient conservatively should have been documented in the medical record. (Tr. at 295-296).

***Dr. Sivashanker's Testimony***

124. Dr. Sivashanker testified that Patient 4 had complained of substernal chest pain with shortness of breath and cough. The pain was "pressure-like" and did not radiate. Dr. Sivashanker testified that the MB was elevated, so he called it a possible myocardial infarction. Nevertheless, Dr. Sivashanker testified that Patient 4's chest pain was atypical and his EKG normal. Dr. Sivashanker further argued that a later cardiac catheterization had revealed "a non-significant occlusion of the coronary artery." Therefore, Dr. Sivashanker had concluded that it had been merely a "possible MI." Dr. Sivashanker admitted, however, that he had not written a diagnosis of "possible M.I." but instead had written "acute M.I." (Tr. at 183-186).
125. Dr. Sivashanker testified that he had not consulted a cardiologist because Patient 4 had not presented with a complicated cardiac problem. The EKG had been normal, and the chest pain had been stable. Dr. Sivashanker testified that, practicing in a small town, an internist generally manages his patients' problems unless a complication arises. Dr. Sivashanker testified that Patient 4 had presented with a simple case, and an internist was capable of managing it. (Tr. at 186-187, 1034-1035).

***Dr. Mahizhnan's Testimony***

126. Dr. Mahizhnan testified that, using the same rationale he had discussed regarding Patient 4's previous admission, Dr. Mahizhnan did not believe that Patient 4 had had a myocardial infarction in December 1990.

Dr. Mahizhnan further testified that, even if Patient 4 had suffered a myocardial infarction, there had been no need for cardiac consultation. Dr. Mahizhnan testified that a board certified internist is qualified to treat patients with uncomplicated myocardial infarctions. Finally, Dr. Mahizhnan testified that Dr. Sivashanker had treated Patient 4 appropriately, even if he had suffered a myocardial infarction. (Tr. at 1148-1151).

127. On cross-examination, however, Dr. Mahizhnan testified that he agreed with Dr. Sivashanker's discharge diagnosis of "acute myocardial infarction, possibly subendocardial." (Tr. at 1350-1351).

***Dr. Beaver's Testimony***

128. Dr. Beaver testified that the American College of Cardiologists recommends that persons with subendocardial myocardial infarctions be managed conservatively. After a period of recovery, a stress test should be performed to determine if any additional intervention is required. Dr. Beaver testified that an internist or a family practitioner is capable of managing such a patient. Dr. Beaver concluded that Dr. Sivashanker had treated Patient 4 appropriately. (Tr. at 1178-1182).

Nevertheless, on cross-examination, Dr. Beaver testified that when a patient has repeated incidents of subendocardial myocardial infarction or cardiac ischemia, the physician may want to evaluate the patient more aggressively, as with a cardiac catheterization. (Tr. at 1260-1261).

**DR. SIVASHANKER'S APRIL 9, 1992, LETTER TO PATIENT 4**

*Regarding the Board's allegation Dr. Sivashanker's office records indicate that Dr. Sivashanker notified Patient 4 in a letter of April 9, 1992, that until Patient 4's outstanding bill of \$1,004.96 was paid Dr. Sivashanker could no longer see him. Dr. Sivashanker inappropriately terminated Dr. Sivashanker's patient-physician relationship with Patient 4*

***Dr. Sivashanker's Office Records for April 9, 1992***

129. Dr. Sivashanker's office chart for Patient 4 contains the following letter, dated April 9, 1992:

Dear [Patient 4]:

You have been my patient for over eight years now and I regret having to write this letter. Unfortunately, until your outstanding bill of \$1,004.96 is taken care of, I cannot see you again. I recommend you call Pam at the billing company at [phone number] to set up a payment schedule. I hope to see you again soon.

Sincerely, S.E. Sivashanker, M.D.

(St. Ex. 4 at 172).

Dr. Sivashanker did not see Patient 4 again until September 1998. (St. Ex. 4 at 32).

***Dr. Miller's Testimony***

130. Dr. Miller testified that, when terminating a patient relationship for financial reasons, the standard of care requires that "all other efforts at correcting the financial debt should be exhausted." In addition, the physician should allow a period of time before terminating the patient from the practice to allow the patient time to find another physician. Moreover, the terminating physician should assist the patient in finding another physician, and should provide records for the new physician. Dr. Miller concluded that the process Dr. Sivashanker used to notify Patient 4 that he had been terminated from Dr. Sivashanker's practice was below the standard of care. (Tr. at 297-299).

**FINDINGS OF FACT**

- A. The evidence presented at hearing supports the following allegations made by the Board in the March 10, 1999, Notice of Opportunity for Hearing in the matter of Saravana E. Sivashanker, M.D.:

1. Regarding Patient 1:

- a. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 1, a 59-year-old male with a history of mental retardation, hypogonadism, osteoporosis, diabetes, and chronic obstructive pulmonary disease [COPD].
- b. On May 19, 1988, Patient 1 was admitted to the hospital for urological surgery.
- c. In his discharge summary for the May 19, 1988, hospitalization, Dr. Sivashanker noted that Patient 1's myocardial band (MB) isoenzyme levels were positive, however, the laboratory reports in the patient's chart indicate that the MB levels were normal.
  - Dr. Sivashanker testified that he had stated in the discharge summary that the MB had been positive which suggested an acute myocardial infarction. Nevertheless, Dr. Sivashanker argued that, because he had not listed a myocardial infarction in the discharge diagnoses, it meant that he had not "strongly" believed that Patient 1 had really had a myocardial infarction.

Dr. Sivashanker's reasoning is utterly without merit. In fact, throughout the hospitalization, Patient 1 had not even demonstrated an elevated CK and no MB was ever calculated. Therefore, not only did Dr. Sivashanker record inaccurate information in his progress notes, but he also relied on

that inaccurate information in his discharge summary and continued to rely on it at hearing. Such carelessness is highly inappropriate.

- d. On January 5, 1990, Dr. Sivashanker admitted Patient 1 to the hospital for exacerbation of COPD and CHF.
- e. During the January 5, 1990, hospitalization, Dr. Sivashanker inappropriately directed the nurses to use intravenous Lasix for chest congestion on an as-needed basis.
  - The testimony supports a finding that Dr. Sivashanker inappropriately ordered the administration of Lasix on a “PRN” basis. As noted by Dr. Beaver, the problem with such an order is that it allows nurses to make medical decisions, which they are not trained to do. Moreover, such an order leaves room for error and allows the possibility of inappropriate treatment being administered by the nurses.
- f. During the January 5, 1990, hospitalization, Dr. Sivashanker inappropriately continued to prescribe intravenous Lasix concurrently with intravenous saline in order to correct iatrogenic hyponatremia, hypokalemia, and hypotension. Dr. Sivashanker interpreted and treated this clinical syndrome as early cardiogenic shock, when Patient 1 actually had volume depletion, pre-renal azotemia, hyponatremia, and hypokalemia from excessive use of diuretics.
  - By January 17, Patient 1’s serum sodium had dropped to 120, his potassium was 2.7, his BUN was 41, his creatinine was 1.9, and his blood pressure was 78/60. As noted by Dr. Miller these are serious conditions which resulted from excessive use of diuretics. In fact, both Dr. Beaver and Dr. Chinn acknowledged that diuretics had played a role in the significant volume depletion, hypotension, and electrolyte imbalance.

Dr. Sivashanker testified that the hypovolemia and electrolyte imbalances had been caused by Patient 1’s not eating or drinking adequate fluids and severe diarrhea. As noted by Dr. Miller, however, even if that was true, giving diuretics to such a patient makes no medical sense in the absence of overt heart failure.

Nevertheless, the hospital record does not support Dr. Sivashanker’s conclusions or the conclusions of Dr. Sivashanker’s experts that diarrhea had contributed to the hypovolemia and electrolyte imbalances. There is no evidence in the record that Patient 1 had ever experienced diarrhea, let alone constant diarrhea, other than Dr. Sivashanker’s January 19, 1990, notation “running diarrhea.”

Although the severe electrolyte balance was evident by January 17, Patient 1 had not even experienced a “loose” stool until January 19. In fact, a review of the nurses’ notes and the intake and output records indicates that on January 16, 1990, Patient 1 had had one solid stool. On January 17, Patient 1 had one large soft stool late in the day. On January 18, Patient 1 had one loose stool. On January 19, Patient 1 was incontinent of one loose stool. There are no additional stools documented in the records. Accordingly, the hospital record does not support Dr. Sivashanker’s contention that Patient 1 had experienced any diarrhea at all.

2. Regarding Patient 2

- a. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 2, a 75-year-old woman with a history of stroke, hypertension, and atrial fibrillation.
- b. Patient 2 was admitted to the hospital on January 27, 1990, due to a left hemispheric stroke. An upper gastrointestinal endoscopy showed that Patient 2 had an “active” duodenal ulcer. Dr. Sivashanker prescribed Coumadin therapy for Patient 2. However, Dr. Sivashanker inappropriately failed to document in the medical records Dr. Sivashanker’s rationale for treating Patient 2 with Coumadin, in light of the active duodenal ulcer.
  - The hospital record contains significant documentation by consultants regarding Patient 2’s need for Coumadin, as well as the risks and benefits of such therapy. Nevertheless, Dr. Miller and Dr. Beaver agreed that Dr. Sivashanker, as the primary physician, should have documented his own rationale for choosing to anti-coagulate a patient with an embolic stroke, atrial fibrillation, and an active duodenal ulcer.
- c. On May 21, 1991, Dr. Sivashanker admitted Patient 2 to the hospital for syncope. Patient 2 had chronic atrial fibrillation. A previous out-patient Holter monitor had shown multifocal premature ventricular contractions and bradycardia. At admission, Patient 2 was taking Coumadin and baby aspirin for stroke prevention. The Coumadin and aspirin were discontinued after a digital rectal examination showed “streaks of blood on glove.” To evaluate this finding, Dr. Sivashanker performed a flexible sigmoidoscopy and a barium enema, which showed only diverticuli.
- d. When discharged on May 31, 1991, Patient 2 was still in atrial fibrillation, but Dr. Sivashanker did not prescribe Coumadin. Dr. Sivashanker should have

reinstated Coumadin therapy for Patient 2 at discharge or in the out-patient setting if follow-up stool guiacs were negative.

- The evidence supports a finding that Dr. Sivashanker's care and treatment of Patient 2 during the May 21, 1991, hospitalization had fallen below the minimal standard of care due to Dr. Sivashanker's failure to reinstitute anti-coagulation therapy. Patient 2's risk of additional stroke was high, based on her prior stroke, the blood clots found in her left ventricle, and the continued atrial fibrillation.

Moreover, the hospital record did not indicate that Patient 2 presented with a high risk of bleeding. The record noted one instance of blood on a glove during a rectal examination. Nevertheless, Patient 2's hemoglobin, hematocrit, and vital signs failed to reflect any significant blood loss.

Furthermore, although the source of the bleeding was never identified, it did not appear that Dr. Sivashanker was very concerned with locating the source. Dr. Sivashanker attempted a flexible sigmoidoscopy, but was unsuccessful because the bowel had not been evacuated. Dr. Sivashanker did not suggest repeating the test after proper bowel preparation.

Dr. Sivashanker did offer to perform a colonoscopy which, Dr. Sivashanker testified, Patient 2 refused. This statement is not supported by the hospital record which indicates that, although Patient 2 had at first refused, she later agreed to have the test performed. Nevertheless, the test was not performed.

Dr. Mahizhnan testified that an appropriate follow-up examination would have been an arteriogram. But as noted by Dr. Mahizhnan, there was no indication that an arteriogram had been offered or that the bleeding had been serious enough to warrant the test.

In addition, there was no indication that Dr. Sivashanker ever performed guiac testing after discharge from the hospital until four years after discharge.

Finally, despite strenuously arguing the reasons why Patient 2 absolutely warranted the initiation of Coumadin in January 1990, Dr. Sivashanker did not even document his rationale for discontinuing the Coumadin in 1991. Moreover, Dr. Sivashanker failed to document his reasons for not reinstituting Coumadin after evaluating the bleeding, or his plans to monitor Patient 2 after discharge.

Accordingly, the evidence supports a finding that there was not sufficient medical justification to withhold Coumadin in Patient 2, in light of the risks associated with that decision.

- e. Dr. Sivashanker's medical records indicate that Dr. Sivashanker did not perform stool guiac testing following discharge.
- Prior to discharge in May 1991, Dr. Sivashanker wrote that he would not pursue Patient 2's gastrointestinal bleeding any further, other than to continue guiac testing. Nevertheless, there is no indication that Dr. Sivashanker performed any guiac testing post-discharge prior to the one test performed in 1995.
- f. Patient 2 did not resume taking Coumadin until May 1995, when the Coumadin was reinstated at a neurologist's recommendation. Patient 2 remained in atrial fibrillation and continued to be at risk of recurring stroke throughout this time period due, in part, to Dr. Sivashanker's failure to continue Coumadin therapy.
- Dr. Sivashanker failed to prescribe Coumadin to Patient 2 between 1991 and 1995. As noted above, Dr. Sivashanker's reasons for not reinstating Coumadin had not been sufficient.
  - Between 1991 and 1995, Patient 2 had been at risk for additional stroke. In fact, Patient 2 suffered a lacunar infarct sometime between 1991 and 1995.
  - Among his reasons for not reinstating Coumadin, Dr. Sivashanker testified that Patient 2 had been discharged to a nursing home after the 1991 hospitalization; that Patient 2 had been primarily managed by three nursing home physicians, and that he had seen Patient 2 only sporadically when she went to his office for follow-up visits. Dr. Sivashanker argued that Patient 2 had been managed by a number of physicians between 1991 and 1995, and none of the physicians had reinstated Coumadin.

Dr. Sivashanker's position is refuted by the medical records. Patient 2 was discharged to her daughter's home, and not to a nursing home. Patient 2 did not enter a nursing home until sometime between July 1992 and April 1995. Moreover, there is no indication that Patient 2 was seen by any other primary physician, or that her potential for embolic stroke and/or bleeding had been monitored by another physician.

3. Regarding Patient 3

- a. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 3, an 82-year-old woman with a history of CHF, arteriosclerotic cardiovascular disease, and organic brain syndrome.
- b. Patient 3 was admitted to the hospital on March 15, 1990, with the diagnosis of rapid atrial fibrillation and secondary mild CHF.
- c. On March 23, 1990, Dr. Sivashanker noted the presence of chest congestion and ordered 80 mg of intravenous Lasix, but continued Patient 3 on intravenous normal saline. A chest x-ray showed no evidence of significant CHF. Since there was no evidence of significant CHF, Dr. Sivashanker's use of diuretics was inappropriate.
  - The evidence supports a finding that Dr. Sivashanker's use of diuretics was inappropriate in light of the facts that CHF, if present, was not significant and that Patient 3 was already suffering severe hyponatremia and other electrolyte imbalance.
  - Dr. Miller testified that Dr. Sivashanker's use of diuretics had directly caused Patient 3's severe electrolyte imbalance. Dr. Chinn acknowledged that, despite the possibility that Patient 3 had been suffering from SIADH, Dr. Sivashanker's use of diuretics had also contributed to Patient 3's hyponatremia. In fact, even Dr. Sivashanker acknowledged that the hyponatremia may have been diuretic induced.
  - Dr. Sivashanker consistently disregarded the advise of consultants. For example, on March 23, 1990, Dr. Graham, the cardiologist, advised that Patient 3 should be maintained by limiting fluid intake, rather than administering diuretics. Nevertheless, Dr. Sivashanker continued to provide diuretics and fluids.

Dr. Williams, the intensivist, advised that Patient 3's hyponatremia may have been the result of over-diuresis with Lasix. He recommended restricting fluids and discontinuing Lasix. Dr. Sivashanker acknowledged that Dr. Williams had recommended discontinuing the Lasix. Nevertheless, Dr. Sivashanker had chosen to disregard Dr. Williams' recommendation because Dr. Sivashanker believed that Dr. Williams had not been concerned with Patient 3's heart failure but had only been concerned with her sodium. Therefore, Dr. Sivashanker believed that he, as the primary physician, could better see the total picture.

Furthermore, Dr. Venkataraman, the nephrologist, suggested that Patient 3's hyponatremia had been induced by the use of diuretics. Dr. Venkataraman recommended discontinuing the Capozide, Lasix, and fluid restrictions. Dr. Sivashanker chose to disregard Dr. Venkataraman's advice for a variety of reasons, none of which are convincing. For example, Dr. Sivashanker testified that, even if the hyponatremia had been diuretic induced, it was the diuretics prescribed to Patient 3 by her family physician prior to the hospitalization, rather than the diuretics ordered by Dr. Sivashanker. In addition, Dr. Sivashanker argued that Dr. Venkataraman, like Dr. Williams, had been concerned only about the patient's sodium, while Dr. Sivashanker had been concerned about the sodium as well as Patient 3's propensity for cardiac failure. Finally, Dr. Sivashanker argued that Dr. Venkataraman had made an error by concluding that Patient 3 was in a pre-renal state; therefore, because Dr. Venkataraman had been "wrong with one thing, [Dr. Sivashanker] did not want to accept any of his explanations."

Dr. Sivashanker continued to administer diuretics in an inappropriate manner despite consistent and reasoned advice of a number of consultants.

- Finally, Patient 3 was harmed by Dr. Sivashanker's use of excessive diuretics since Patient 3 had become confused and lethargic as a result of hyponatremia.
- d. Dr. Sivashanker's order of March 28, 1990, to give sodium chloride intravenously until Patient 3's serum sodium level rose above 135 was inappropriate, as it subjected Patient 3 to the risk of CHF and neurological complications.
- Dr. Sivashanker testified that he now realizes that ordering an IV of either 0.9% or 3% sodium chloride to be infused until a certain serum sodium level is reached, and leaving that decision to the discretion of the nurses, had been inappropriate.
  - Dr. Chinn also agreed the order had been inappropriate in light of Patient 3's propensity to develop CHF.
- e. It was inappropriate for Dr. Sivashanker to direct nurses to use intravenous Lasix for chest congestion on an as-needed basis.

4. Regarding Patient 4

- a. In the routine course of his practice, Dr. Sivashanker undertook the care of Patient 4, a 68-year-old male. From 1984 to 1992, Dr. Sivashanker treated Patient 4 for hypertension, diabetes, COPD, and heart disease.
- b. Patient 4 was admitted to the hospital on February 29, 1990, with a history of chest pain, cough, progressive shortness of breath, and an exacerbation of COPD.
- c. On February 27, 1990, Patient 4's CK was elevated and the MB was 3.6. On February 28, 1990, Patient 4's CK level remained elevated and the MB had risen to 11.3. On March 1, 1990, Patient 4's CK remained elevated and the MB was 8.6. Dr. Sivashanker performed three electrocardiograph tests during this time period which showed no Q waves or ST segment elevation. Patient 4 complained of chest pain during the this time period. Despite Patient 4's symptoms and laboratory reports, Dr. Sivashanker failed to diagnose and appropriately treat Patient 4's acute myocardial infarction
  - In this case, Dr. Sivashanker consistently ignored the signs of cardiac distress, including repeated episodes of mid-sternal chest pain, acute shortness of breath, pallor and diaphoresis, elevated cardiac enzymes and non-specific ST and T wave changes. In fact, Dr. Sivashanker did not even mention the elevated cardiac enzymes in his progress notes.

Moreover, when asked if Patient 4's complaints of chest pain, respiratory distress, increased respiratory rate, and pallor could have been symptoms of a myocardial infarction, Dr. Sivashanker stated that they could not. Dr. Sivashanker argued that Patient 4 had complained of coughing along with chest wall pain which is not a classic presentation for a myocardial infarction. Therefore, Dr. Sivashanker reasoned, Patient 4 had not presented with a myocardial infarction.

Nevertheless, as noted by Dr. Miller, a physician must assume that chest pain is cardiac in origin until proven otherwise. Moreover, because the MB came back elevated, the burden of proof fell to Dr. Sivashanker to prove that it was not cardiac pain. Dr. Sivashanker did not do so.

- Dr. Sivashanker's testimony regarding this hospitalization is often inconsistent and/or is contradicted by the hospital records. For example, Dr. Sivashanker testified that Patient 4 had not been a candidate for cardiac

problems. Nevertheless, Dr. Sivashanker's records indicate that Patient 4 had suffered a myocardial infarction in 1961.

Furthermore, Dr. Sivashanker testified that, when he admitted Patient 4 to the hospital, he had allowed Patient 4 activity as tolerated because Patient 4 had not presented with complaints of chest pain. Nevertheless, both Dr. Sivashanker's office records and the hospital record indicate that Patient 4 had been suffering chest pain since 5:00 a.m. that morning.

Moreover, Dr. Sivashanker tried to argue that he had not been notified of the elevated cardiac enzymes until after Patient 4's discharge from the hospital. Nevertheless, the nurses' notes indicate that his office had been called with the results as soon as the laboratory had completed the test.

In addition, Dr. Sivashanker testified that he had diagnosed an acute myocardial infarction; but when asked where he had documented that in the record, Dr. Sivashanker could only point to the hospital's computerized DRG sheet. Dr. Sivashanker then admitted that he had only diagnosed "possible subendocardial myocardial infarction."

On the other hand, despite arguing that he had diagnosed an acute myocardial infarction, Dr. Sivashanker later testified that Patient 4 had not had an myocardial infarction at all. Dr. Sivashanker reasoned that the MB had been low, only 2.34, and other enzymes, i.e., the SGOT, LDH and SGPT, had been normal. Therefore, Dr. Sivashanker concluded that the elevated MB had merely been a "red herring."

- d. On December 10, 1990, Patient 4 was admitted to the hospital with shortness of breath and chest pain. On admission, Patient 4's CK level was twice the normal level, and the MB was 13. Patient 4 was admitted to the Intensive Care Unit and treated with a nitroglycerin patch and Cardene. Dr. Sivashanker did not request a cardiology consultation. Patient 4 exhibited symptoms of an acute myocardial event. However, Dr. Sivashanker failed to treat Patient 4 appropriately, or Dr. Sivashanker failed to document Dr. Sivashanker's rationale for conservative treatment of Patient 4.
  - Patient 4 was suffering repeated episodes of subendocardial myocardial infarction. In fact, Dr. Sivashanker's records indicate that Patient 4 had suffered a third event in September 1991. In light of the repeated events, one only three months earlier, it was inappropriate for Dr. Sivashanker to manage Patient 4 as an uncomplicated subendocardial myocardial infarction and without the assistance of a cardiology consultation.

- e. Dr. Sivashanker's office records indicate that Dr. Sivashanker notified Patient 4 in a letter of April 9, 1992, that until Patient 4's outstanding bill of \$1,004.96 was paid Dr. Sivashanker could no longer see him. Dr. Sivashanker inappropriately terminated Dr. Sivashanker's patient-physician relationship with Patient 4.
- B. The following allegations made by the Board in the March 10, 1999, Notice of Opportunity for Hearing were not supported by the evidence presented in this matter.
1. Regarding Patient 1
    - a. Dr. Sivashanker's final diagnosis for Patient 1's May 15, 1988, hospitalization was acute lung edema, but the medical records do not support this diagnosis. Instead, the medical records support a diagnosis of COPD decompensation, which Dr. Sivashanker failed to recognize and treat appropriately.
      - Dr. Miller's testimony that Patient 1 had not had CHF during this admission is not convincing. Dr. Miller testified that Patient 1 had not had CHF, despite the findings of a cardiologist who saw Patient 1 at that time and despite documented findings consistent with CHF. Dr. Miller stated that he had based his conclusion on his belief that the 1987 cardiac catheterization had indicated no cardiac problems and on his belief that Patient 1 had had no previous cardiac problems.

Regarding the 1987 cardiac catheterization, Dr. Chinn's testimony was more persuasive. Dr. Chinn testified that the cardiac catheterization had revealed pulmonary hypertension and a diminished cardiac output. Dr. Chinn explained that pulmonary hypertension will likely lead to right heart failure. Dr. Chinn further testified that the reduced cardiac output was most likely caused by left ventricular failure. Both of these conditions are consistent with CHF.

Moreover, Dr. Miller's conclusion that Patient 1 had had no cardiac history was refuted by the medical records, which document previous hospitalizations for CHF and multiple myocardial infarctions. Finally, Dr. Miller's testimony that such previous records were "not relevant" to Patient 1's condition in 1988 was preposterous, given that Dr. Miller had based his opinion, in part, on Patient 1's lack of a cardiac history.
    - b. Throughout the May 19, 1988, hospitalization, Dr. Sivashanker ordered Lasix and nitroglycerin for Patient 1.

- The testimony at hearing revealed that Dr. Mortera and Dr. French had ordered nitroglycerin for Patient 1. In addition, Dr. Mortera and Dr. French had ordered Lasix prior to May 22, 1988. Finally, Dr. Miller's testimony regarding the propriety of prescribing those medications did not pertain to Dr. Sivashanker.
- c. During the January 5, 1990, hospitalization, Dr. Sivashanker treated Patient 1 for CHF, when there was no clinical evidence of CHF.
- After first testifying that there had been no evidence of CHF during Patient 1's January 5, 1990, hospitalization, Dr. Miller later stated that, in light of the information pointed out to him during cross-examination, his opinion had changed since preparing his expert report and since testifying on direct examination. At that time, Dr. Miller concluded that the medical record supported a conclusion that Patient 1 had been suffering from CHF.

Moreover, as noted by Dr. Sivashanker, the hospital record documented numerous symptoms consistent with a diagnosis of CHF, including rales, frothy sputum, tachypnea, orthopnea, jugular vein distention, an S3 gallop, hypotension, systolic murmur, pedal edema and pleural edema. Moreover, Patient 1 had had an extensive cardiac history, including myocardial infarctions, chronic hypotension related to CHF, and decreased cardiac output documented by the cardiac catheterization.

## 2. Regarding Patient 2

On September 11, 1996, Patient 2 presented to Dr. Sivashanker's office with complaints of hemoptysis and vaginal bleeding. Dr. Sivashanker failed to document how Dr. Sivashanker addressed these symptoms.

- A review of Dr. Sivashanker's office record for Patient 2 suggests that the undated Page 4 is a logical continuation the note for the entry dated September 11, 1996. Moreover, the treatment documented on Page 4 is appropriate for the complaints with which Patient 2 presented on September 11, 1996.

## CONCLUSIONS OF LAW

The conduct of Saravana E. Sivashanker, M.D., as described in Findings of Fact A.1 through A.4, constitutes "[a] departure from, or the failure to conform to, minimal standards of care of similar practitioners under the same or similar circumstances, whether or not actual injury to a patient is established," as that clause is used in Section 4731.22(B)(6), Ohio Revised Code.

### **PROPOSED ORDER**

It is hereby ORDERED that:

1. The certificate of Saravana E. Sivashanker, M.D., to practice medicine and surgery in the State of Ohio shall be **SUSPENDED** for an indefinite period of time, but not less than six months.
2. Within thirty days of the effective date of this Order, Dr. Sivashanker shall provide a copy of this Order by certified mail, return receipt requested, to the proper licensing authority of any state or jurisdiction in which he currently holds any professional license. Dr. Sivashanker shall also provide a copy of this Order by certified mail, return receipt requested, at the time of application to the proper licensing authority of any state in which he applies for any professional license or reinstatement of any professional license. Further, Dr. Sivashanker shall provide this Board with a copy of the return receipt as proof of notification within thirty days of receiving that return receipt.
3. Within thirty days of the effective date of this Order, Dr. Sivashanker shall provide a copy of this Order to all employers or entities with which he is under contract to provide physician services or is receiving training, and the Chief of Staff at each hospital where Dr. Sivashanker has privileges or appointments. Further, Dr. Sivashanker shall provide a copy of this Order to all employers or entities with which he contracts to provide physician services, or applies for or receives training, and the Chief of Staff at each hospital where Dr. Sivashanker applies for or obtains privileges or appointments.
4. The Board shall not consider reinstatement of Dr. Sivashanker's certificate to practice unless all of the following minimum requirements have been met:
  - a. Dr. Sivashanker shall submit an application for reinstatement, accompanied by appropriate fees.
  - b. Upon submission of his application for reinstatement, Dr. Sivashanker shall provide acceptable documentation of satisfactory completion of a course on maintaining adequate and appropriate medical records, such course to be approved in advance by the Board or its designee. Any courses taken in compliance with this provision shall be in addition to the CME requirements for relicensure for the CME acquisition period(s) in which they are completed.
  - c. Upon submission of his application for reinstatement, Dr. Sivashanker shall provide acceptable documentation of Dr. Sivashanker's enrollment in a post-graduate training

program in the area of internal medicine of at least six-months duration. Such program shall be approved in advance by the Board.

- d. In the event that Dr. Sivashanker has not been engaged in the active practice of medicine and surgery for a period in excess of two years prior to application for reinstatement, the Board may exercise its discretion under Section 4731.222, Ohio Revised Code, to require additional evidence of his fitness to resume practice.
5. Upon reinstatement, Dr. Sivashanker's certificate shall be subject to the following PROBATIONARY terms, conditions, and limitations for a period of at least five years:
- a. Dr. Sivashanker shall not request modification of the terms, conditions, or limitations of probation for at least one year after imposition of these probationary terms, conditions, and limitations.
  - b. Dr. Sivashanker shall obey all federal, state, and local laws, and all rules governing the practice of medicine and surgery in Ohio.
  - c. Dr. Sivashanker's certificate shall be LIMITED to participation in the post-graduate training program which was approved by the Board prior to reinstatement. The limitation shall not be terminated until Dr. Sivashanker provides the Board with acceptable documentation verifying successful completion of such program.
  - d. Prior to the termination of the limitation set forth in paragraph 5.c, above, Dr. Sivashanker shall submit to the Board and receive its approval for a plan of practice in Ohio which, until otherwise determined by the Board, shall be limited to a supervised structured environment in which Dr. Sivashanker's activities will be directly supervised and overseen by a monitoring physician approved in advance by the Board.

The monitoring physician shall monitor Dr. Sivashanker and his patient charts. The chart review may be done on a random basis, with the number of charts reviewed to be determined by the Board. The monitoring physician shall provide the Board with reports on Dr. Sivashanker's progress and status and on the status of his patient charts on a quarterly basis, or as otherwise directed by the Board. All monitoring physician reports required under this paragraph must be received in the Board's offices no later than the due date for Dr. Sivashanker's quarterly declaration. It is Dr. Sivashanker's responsibility to ensure that the reports are timely submitted.

Dr. Sivashanker shall obtain the Board's prior approval for any alteration to the practice plan approved pursuant to this Order.

In the event that the approved monitoring physician becomes unable or unwilling to serve, Dr. Sivashanker shall immediately notify the Board in writing and shall make arrangements for another monitoring physician as soon as practicable.

Dr. Sivashanker shall refrain from practicing until such supervision is in place, unless otherwise determined by the Board. Dr. Sivashanker shall ensure that the previously designated monitoring physician also notifies the Board directly of his or her inability to continue to serve and the reasons therefor.

- e. Dr. Sivashanker shall appear in person for interviews before the full Board or its designated representative within three months of the date in which probation becomes effective, at three month intervals thereafter, and upon his request for termination of the probationary period, or as otherwise requested by the Board.

If an appearance is missed or is rescheduled for any reason, ensuing appearances shall be scheduled based on the appearance date as originally scheduled. Although the Board will normally give him written notification of scheduled appearances, it is Dr. Sivashanker's responsibility to know when personal appearances will occur. If he does not receive written notification from the Board by the end of the month in which the appearance should have occurred, Dr. Sivashanker shall immediately submit to the Board a written request to be notified of his next scheduled appearance.

- f. Dr. Sivashanker shall submit quarterly declarations under penalty of Board disciplinary action or criminal prosecution, stating whether there has been compliance with all the conditions of probation. The first quarterly declaration must be received in the Board's offices on the first day of the third month following the month in which probation becomes effective, provided that if the effective date is on or after the 16th day of the month, the first quarterly declaration must be received in the Board's offices on the first day of the fourth month following. Subsequent quarterly declarations must be received in the Board's offices on or before the first day of every third month.
- g. In the event that Dr. Sivashanker should leave Ohio for three consecutive months, or reside or practice outside the State, Dr. Sivashanker must notify the Board in writing of the dates of departure and return. Periods of time spent outside Ohio will not apply to the reduction of this probationary period, unless otherwise determined by motion of the Board in instances where the Board can be assured that the purposes of the probationary monitoring are being fulfilled.
- h. If Dr. Sivashanker violates probation in any respect, the Board, after giving him notice and the opportunity to be heard, may institute whatever disciplinary action it deems appropriate, up to and including the permanent revocation of his certificate.

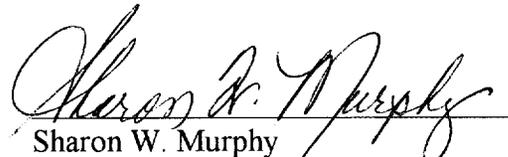
Report and Recommendation

In the Matter of Saravana E. Sivashanker, M.D.

Page 77

6. Upon successful completion of probation, as evidenced by a written release from the Board, Dr. Sivashanker's certificate will be fully restored.

This Order shall become effective thirty days from the date of mailing of notification of approval by the Board.

  
Sharon W. Murphy  
Attorney Hearing Examiner

July 12, 2000

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SARAVANA E. SIVASHANKER, M.D.

Dr. Egner directed the Board's attention to the matter of Saravana E. Sivashanker, M.D. She advised that objections were filed to Hearing Examiner Murphy's Report and Recommendation and were previously distributed to Board members.

**DR. SOMANI MOVED TO APPROVE AND CONFIRM MS. MURPHY'S PROPOSED FINDINGS OF FACT, CONCLUSIONS, AND ORDER IN THE MATTER OF SARAVANA E. SIVASHANKER, M.D. MR. BROWNING SECONDED THE MOTION.**

Dr. Egner stated that she would now entertain discussion in the above matter.

Dr. Somani stated that he looked at this case and saw this as a question of quality of care of poor patients by Dr. Sivashanker. These cases go back ten or more years. Dr. Somani stated that he is a little concerned about the testimony from the expert on behalf of the Board and the testimony of the cardiologist, the nephrologist and others who testified on behalf of Dr. Sivashanker. Obviously, when the medical professions review a case that's about ten years old, it becomes very difficult to go back to the thinking of the medical professionals at that time in light of what has been learned over the intervening ten to twelve years. Sometimes the discussion does reflect the current thinking of how a case would be managed today versus how it would have been managed ten years ago.

Dr. Somani stated that he was somewhat surprised that the testimony of Dr. Beaver and Dr. Miller was in many respects similar, but there were some points they disagreed on regarding how the care should have been provided. His reading of the cases and some of the discussion and details suggested to him that there may have been some degree of discussion about how the case should have been managed, but he didn't get the impression that there was gross negligence in all of these cases. He felt that the Proposed Order to suspend the license is inappropriate. This is an old case of patient care of ten to twelve years ago. He cannot support the proposed suspension.

**DR. SOMANI MOVED TO DISMISS THIS CASE.** The motion died for lack of a second.

Dr. Egner stated that Dr. Sivashanker testified at hearing that he had given up all of his hospital privileges, but actually his privileges were revoked at two hospitals. He was unable to maintain a private practice. She believes that minimal standards cases always go back a fair amount of time. Dr. Egner agreed that it is difficult sometimes to say that this happened ten years ago and now we're going to render an opinion; however, by the very nature of these cases, she thinks that happens all of the time. It takes a while for the Board to be made aware of the cases. Many times these cases are very long in investigating so that the Board can be fair by gathering all of the information that the Board needs about the patient and the physician. Timeliness is not always a paramount issue here. What is an issue is that these patients were inappropriately cared for and the care did not meet the minimum standards, and that a period of re-education is in order. Then Dr. Sivashanker will be monitored for a period of five years. The suspension itself is not all that long. The Board is giving Dr. Sivashanker a chance to get back into practice and practice the kind of medicine that the citizens of Ohio deserve.

July 12, 2000

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Dr. Stienecker agreed with Dr. Egner. He added that when the Board has a physician whose privileges have been revoked at one or two hospitals and who tends to practice medicine, as indicated by his records, by committee, without really maintaining control of all of these patients and not really making reasonably good or adequate decisions based upon the information that he's getting from others, the physician needs some kind of re-education process, at least to satisfy the Board's mandate to protect the public and ensure that this physician is functioning up to par.

Dr. Stienecker referred to Dr. Sivashanker's objections, where he states, concerning Patient 2, that: 1. There was no history of bleeding, 2. He was following protime; and 3. He was adjusting the protime as per the Coumadin. Dr. Stienecker stated that he thinks the Board ought to allow Dr. Sivashanker to adjust that. He would hope that Dr. Sivashanker was adjusting the Coumadin as per the protime and not vice-versa. He stated that Dr. Sivashanker ought to have a chance to change that before it goes into the record forever.

Dr. Stienecker noted that the Hearing Examiner's Proposed Order states that "upon application for reinstatement (Dr. Sivashanker) would provide acceptable documentation of enrollment in a post-graduate training program in the area of internal medicine." Dr. Stienecker stated that he would also like to see some kind of requirement that Dr. Sivashanker not only enroll, but actually complete the course, and that this was, in fact, a six-month residency program as required.

Dr. Egner stated that that seems appropriate. She asked whether that the Board needs to work on a proposed amendment.

Dr. Stienecker stated that the language of the Proposed Order takes it as an act of faith that if Dr. Sivashanker enrolls into a program, he'll go ahead and complete it. The Order as written doesn't require that.

Dr. Egner noted that paragraph 5C does limit Dr. Sivashanker's certificate to participate in the post-graduate training program approved by the Board prior to reinstatement. Dr. Sivashanker will have to verify successful completion of such program. He cannot do this retraining without a license. The reinstatement will be of a limited license.

Dr. Steinbergh suggested that the Board might be able to help this physician by directing him in the same way it directed Dr. Nguyen, a similar case. It allowed Dr. Nguyen to develop a program that was acceptable to the Board and ultimately return to practice. She agrees that retraining is appropriate. This was a difficult case to review. The experts on both sides disagreed somewhat, but Dr. Stienecker's observation is clear that this gentleman really was not in control of who was providing medical care. She believes the retraining issue is the critical one.

Dr. Bhati stated that this was a very lengthy case. He indicated that he was not particularly concerned with the first case. However, the other cases establish that Dr. Sivashanker needs more help. He needs retraining, attitudinal

July 12, 2000

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changes, and he needs to learn how to use a consultant properly. He totally ignored two consultant opinions.

A vote was taken on Dr. Somani's motion to approve and confirm:

VOTE:	Mr. Albert	- abstain
	Dr. Bhati	- aye
	Dr. Talmage	- aye
	Dr. Somani	- nay
	Mr. Browning	- aye
	Dr. Stienecker	- aye
	Dr. Agresta	- aye
	Dr. Steinbergh	- aye

The motion carried.

## SCOTT THOMAS STEWART, P.A.

Dr. Egner directed the Board's attention to the matter of Scott Thomas Stewart, P.A. She advised that objections were filed to Hearing Examiner Murphy's Report and Recommendation and were previously distributed to Board members.

Dr. Egner continued that a request to address the Board has been timely filed on behalf of Mr. Stewart. Five minutes would be allowed for that address.

Mr. Gartland stated that he and Mr. Stewart welcome the opportunity to talk to the Board today. Mr. Gartland stated that he believes the Board needs to hear from Mr. Stewart; therefore, he will defer the time to Mr. Stewart.

Mr. Stewart thanked the Board for the opportunity to address it. Mr. Stewart indicated that he has been interested in the medical field since he was a small child. His mother was a nurse, and he had an uncle who was a pharmacist. He entered the U.S. Army in 1988 to become an army medic to give him an opportunity to have job training and a chance to help care for people. It was during this time that he was first exposed to physician assistants, and he served in Korea during Desert Storm with a gentleman who encouraged him to become a P.A. and took him under his wing. He was unable to fulfill those goals while he was in the service. He left the service in 1992, at which time he moved his family to Dayton and applied to and enrolled in P.A. school at the Kettering College of Medical Arts in Dayton, Ohio. Mr. Stewart stated that he learned a tremendous amount of medical knowledge there. During that time his respect grew for both physicians and P.A.s.

Mr. Stewart continued that he was very excited upon graduation and his accepting his first job in Columbus. This job gave him an opportunity to establish a career and support his family. It allowed them to buy their first home and enroll their children in a good school district.



# State Medical Board of Ohio

77 S. High Street, 17th Floor • Columbus, Ohio 43266-0315 • 614/ 466-3934 • Website: www.state.oh.us/med/

March 10, 1999

Saravana E. Sivashanker, M.D.  
216 Lake Bluff Drive  
Columbus, OH 43235

Dear Doctor Sivashanker:

In accordance with Chapter 119., Ohio Revised Code, you are hereby notified that the State Medical Board of Ohio intends to determine whether or not to limit, revoke, suspend, refuse to register or reinstate your certificate to practice medicine and surgery, or to reprimand or place you on probation for one or more of the following reasons:

- (1) In the routine course of your practice, you undertook the care of Patient 1, as identified on the attached Patient Key (Key confidential - to be withheld from public disclosure), a 59-year-old male with a history of mental retardation, hypogonadism, osteoporosis, diabetes, and chronic obstructive pulmonary disease (COPD).
  - a) On 5/19/88, Patient 1 was admitted to the hospital for urological surgery. Throughout the hospitalization, you ordered Lasix and nitroglycerin for Patient 1. However, chest x-rays did not show congestive heart failure. In addition, you noted in your discharge summary that Patient 1's myocardial band (MB) isoenzyme levels were positive, however, the laboratory reports in the patient's chart indicate that the MB levels were normal.

Your final diagnosis was acute lung edema, but the medical records do not support this diagnosis. Instead, the medical records support a diagnosis of COPD decompensation, which you failed to recognize and treat appropriately.

- b) On 1/5/90, you admitted Patient 1 to the hospital for exacerbation of COPD and for congestive heart failure. You treated Patient 1 for congestive heart failure, when there was no clinical evidence of congestive heart failure.

Moreover, it was inappropriate for you to direct the nurses to use intravenous Lasix for chest congestion on an as-needed basis.

In addition, you inappropriately continued to prescribe intravenous Lasix concurrently with intravenous saline in order to correct iatrogenic hyponatremia, hypokalemia, and hypotension. You interpreted and treated

*Mailed 3/11/99*

this clinical syndrome as early cardiogenic shock, when Patient 1 actually had volume depletion, pre-renal azotemia, hyponatremia, and hypokalemia from excessive use of diuretics.

(2) In the routine course of your practice, you undertook the care of Patient 2, as identified on the attached Patient Key (Key confidential - to be withheld from public disclosure), a 75-year-old woman with a history of stroke, hypertension, and atrial fibrillation.

- a) Patient 2 was admitted to the hospital on 1/27/90, due to a left hemispheric stroke. An upper gastrointestinal endoscopy showed that Patient 2 had an "active" duodenal ulcer. You prescribed Coumadin therapy for Patient 2. However, you inappropriately failed to document in the medical records your rationale for treating Patient 2 with Coumadin, in light of the active duodenal ulcer.
- b) On 5/21/91, you admitted Patient 2 to the hospital for syncope. Patient 2 had chronic atrial fibrillation. A previous out-patient holter monitor showed multifocal premature ventricular contractions and bradycardia. At admission, Patient 2 was taking Coumadin and baby aspirin for stroke prevention. The Coumadin and aspirin were discontinued after a digital rectal examination showed "streaks of blood on glove." To evaluate this finding, you performed a flexible sigmoidoscopy and a barium enema, which showed only diverticuli.

When discharged on 5/31/91, Patient 2 was still in atrial fibrillation, but you did not prescribe Coumadin. You should have reinstated Coumadin therapy for Patient 2 at discharge or in the out-patient setting if follow-up stool guaiacs were negative. Your medical records indicate that you did not perform stool guaiac testing following discharge and that Patient 2 did not resume taking Coumadin until May 1995, when the Coumadin was reinstated at a neurologist's recommendation. Patient 2 remained in atrial fibrillation and continued to be at risk of recurring stroke throughout this time period, in part due to your failure to continue Coumadin therapy.

- c) On 9/11/96, Patient 2 presented to your office with complaints of hemoptysis and vaginal bleeding. You failed to document how you addressed these symptoms.
- (3) In the routine course of your practice, you undertook the care of Patient 3, as identified on the attached Patient Key (Key confidential - to be withheld from public disclosure), an 82-year-old woman with a history of congestive heart failure, atherosclerotic cardiovascular disease, and organic brain syndrome.

Patient 3 was admitted to the hospital on 3/15/90, with the diagnosis of rapid atrial fibrillation and secondary mild congestive heart failure.

It was inappropriate for you to direct the nurses to use intravenous Lasix for chest congestion on an as-needed basis.

On 3/23/90, you noted the presence of chest congestion and ordered 80 mg. of intravenous Lasix, but continued Patient 3 on intravenous normal saline. A chest x-ray at that time showed no evidence of significant congestive heart failure. Since there was no evidence of significant congestive heart failure, the use of diuretics was inappropriate.

Your order of 3/28/90, to give sodium chloride intravenously until Patient 3's serum sodium level rose above 135 was inappropriate, as it subjected Patient 3 to the risk of congestive heart failure and neurological complications.

(4) In the routine course of your practice, you undertook the care of Patient 4, as identified on the attached Patient Key (Key confidential - to be withheld from public disclosure), a 68-year-old male. From 1984 to 1992, you treated Patient 4 for hypertension, diabetes, chronic obstructive pulmonary disease (COPD), and heart disease.

a) Patient 4 was admitted to the hospital on 2/27/90, with a history of chest pain, cough, progressive shortness of breath, and an exacerbation of COPD.

On 2/27/90, Patient 4's creatine kinase (CK) level was elevated and the myocardial band (MB) fraction was 3.6. On 2/28/90, Patient 4's CK level remained elevated and the MB fraction had risen to 11.3. On 3/1/90, Patient 4's CK level remained elevated and the MB was 8.6. You performed three electrocardiograph tests during this time period which showed no Q waves or ST segment elevation. Patient 4 complained of chest pain during this time period. Despite Patient 4's symptoms and laboratory reports, you failed to diagnose and treat appropriately Patient 4's acute myocardial infarction.

b) On 12/10/90, Patient 4 was admitted to the hospital with shortness of breath and chest pain. On admission, Patient 4's CK level was twice the normal level, and the MB fraction was 13. Patient 4 was admitted to the Intensive Care Unit and treated with a nitroglycerin patch and Cardene. You did not request a cardiology consultation. Patient 4 exhibited symptoms of an acute myocardial event. However, you failed to treat Patient 4 appropriately, or you failed to document your rationale for conservative treatment of Patient 4.

- c) Your office records indicate that you notified Patient 4 in a letter of April 9, 1992, that until Patient 4's outstanding bill of \$1004.96 was paid you could no longer see him. You inappropriately terminated your patient-physician relationship with Patient 4.

Your acts, conduct, and/or omissions as alleged in paragraphs (1) - (4) above, individually and/or collectively, constitute "[a] departure from, or the failure to conform to, minimal standards of care of similar practitioners under the same or similar circumstances, whether or not actual injury to a patient is established," as that clause is used in Section 4731.22(B)(6), Ohio Revised Code.

Pursuant to Chapter 119., Ohio Revised Code, you are hereby advised that you are entitled to a hearing in this matter. If you wish to request such hearing, the request must be made in writing and must be received in the offices of the State Medical Board within thirty (30) days of the time of mailing of this notice.

You are further advised that you are entitled to appear at such hearing in person, or by your attorney, or by such other representative as is permitted to practice before this agency, or you may present your position, arguments, or contentions in writing, and that at the hearing you may present evidence and examine witnesses appearing for or against you.

In the event that there is no request for such hearing received within thirty (30) days of the time of mailing of this notice, the State Medical Board may, in your absence and upon consideration of this matter, determine whether or not to limit, revoke, suspend, refuse to register or reinstate your certificate to practice medicine and surgery or to reprimand or place you on probation.

Copies of the applicable sections are enclosed for your information.

Very truly yours,



Anand G. Garg, M.D.  
Secretary

AGG/bjs  
Enclosures

CERTIFIED MAIL #Z 395 591 253  
RETURN RECEIPT REQUESTED