University of Wisconsin Hospital and Clinics

DEPARTMENT OF MEDICINE
Center for Health Sciences J5/215
University of Wisconsin—Madison
600 Highland Avenue • Madison, Wisconsin 53792

October 10, 1979

Mrs. Shirley Pekarske
W131 S6689 Kipling Drive
Hales Corners, WI 53130

Dear Mrs. Pekarske:

Bob Lemanske brought your letter which you sent to him and his wife to me and asked if I would communicate with you regarding the circumstances surrounding the sudden death of your husband. Some of the details are very sketchy because of the abrupt nature of the entire event. Apparently your husband collapsed at the Wisconsin Center at 702 Langdon Street and the fire rescue squad was called and when they arrived their report indicates that they found him pale, semi-conscious, with no obtainable blood pressure and a slow heart rate. They immediately began medication to bring his blood pressure back up to normal, however, this had very little effect. He apparently related to them that he had been having shortness of breath at night for the three or four days prior to this acute episode. They transported him immediately to University Hospitals where he was seen very briefly in the Emergency Room by Dr. Eliot Williams, the chief medical resident, and he was then transferred directly to the coronary care unit where Dr. Fulks and I took over the attempt at resuscitation. When initially seen an electrocardiogram had been obtained which showed a fast heart rate at 130 per minute and had changes suggesting an old anterior myocardial infarction (heart attack) and diffuse changes suggesting marked ischemia (lack of oxygen) to all of the rest of the heart muscle in spite of the fact that he had a reasonable heart rate he was never able to generate any significant blood pressure nor was his heart able to circulate blood adequately throughout his body because of the severe lack of oxygen to the heart muscle. Over approximately the next hour we administered a number of different drugs along with closed chest massage. Anesthesiology was present and assisted us with maintaining his respirations as best we could. We were never able to get an adequate cardiac output (blood flow throughout the body) and in spite of all of the things we did his condition deteriorated until he was unable to maintain any kind of electrical rhythm. The basic cause of death was the inability of the heart to pump blood. We were able over a considerable

period of time to maintain a heart rhythm but the muscle could not contract and therefore there was no significant blood flow. After an hour of closed chest massage in an attempt to maintain some blood flow it became apparent that we could not reverse the situation and there was evidence of irreversible brain damage and at that point further resuscitation attempts were discontinued.

I am enclosing with this letter a copy of the preliminary autopsy report as the final report is not yet available and will take some weeks to process all of the sections in detail. The autopsy confirmed the fact that there was evidence of an old myocardial infarct (heart attack) at sometime in the past but that the acute event was a widespread loss of blood supply resulting in a paleness of the entire heart muscle as well as focal areas of hemorrhages throughout the muscle suggestive of the extensive necrosis (death) of the muscle itself. These findings then are those of a massive acute heart attack which rendered the heart completely incapable of generating reasonable blood flow throughout the body, thus resulting in your husbands death.

I can understand that all of this undoubtedly comes as a severe shock. This unfortunately, however, is one of the major manifestations of coronary artery disease. Patients may be extremely well one day and may even pass rather rigorous physical exams but subclinical coronary artery disease is extremely difficult to detect and one of the major manifestations of coronary disease is sudden death due to the abrupt cessation of blood flow to the heart muscle.

Allow me to express my sincerest sympathy at your tragic loss and if there is anything further that I can supply in the way of information, I would be most willing to do so.

Sincerely,

Condon R. Vander Ark, M.D.
Associate Professor of Medicine
Acting Head, Cardiology Section

CVA:mlj
Encl.

Chart

UNIVERSITY OF WISCONSIN • Department of Pathology and Laboratory Medicine

B6/344 Clinical Science Center • 600 Highland Avenue • Madison, Wisconsin 53792

(*) Autopsy number: W-79-266

(N) Name: Robert Pekarske

(H) History number: 866018

(V) Service: Medical ICU

(W) Ward: F4/M5

(S) Hospital staff: Condon R. Vanderark, M.D.

(R) Hospital resident: Michael W. Fulks, M.D.

(I) Hospital intern: None

(Y) Age: 58 years

(X) Sex: Male

(D) Date/time of death: 9/14/79, 17:36

(A) Date/time of autopsy: 9/15/79, 09:15

(P) Prosector: Renee B. Warren, M.D.

(signature) Renee Warren

(F) Faculty: Robert W. Huntington III, MD

(signature) Robert W Huntington III

9:59 AM 17 Sept 79

CLINICAL DIAGNOSES.

1. History of hypertension.
2. EKG consistent with anteroseptal myocardial infarction.
3. Status post cardiorespiratory arrest.

GROSS ANATOMIC DIAGNOSES.

1. Myocardial infarction.
   a. Old anteroseptal myocardial infarction.
   b. Arteriosclerotic coronary artery disease.
      1. Left anterior descending coronary artery - 99%+obstruction.
      2. Left circumflex coronary artery - 60% obstruction.
      3. Right coronary artery - 20% obstruction.
   c. Cardiomegaly, 550 gm.
   d. Arteriosclerosis of aorta, grade 4/10.
2. Pulmonary edema and congestion; right 850 gm, left 650 gm.
3. Acute hemorrhagic gastritis.
4. Hepatomegaly, 2650 gm.
5. Fibro-congestive splenomegaly, 450 gm.
6. Arterionephrosclerosis, mild.

SUMMARY COMMENT.

At autopsy, there was evidence of an old anteroseptal myocardial infarction. In addition, there was a widespread paleness of the entire myocardium, intramural petechial hemorrhages and focal subendocardial hemorrhages, all being extremely suggestive of extensive myocardial necrosis, sufficient, indeed, to cause this man's acute demise. Severe arteriosclerotic obstruction of the coronary arteries appears to be the forerunner of the myocardial necrosis.