

MASSACHUSETTS INSTITUTE OF TECHNOLOGY

Department of Electrical Engineering, Department of Mechanical Engineering, Division of Biological Engineering, and the Harvard-MIT Division of Health Sciences and Technology

6.022J/2.792J/BEH.371J/HST542J: Quantitative Physiology: Organ Transport Systems

INTRODUCTORY CASE STUDY

INTRODUCTORY CASE STUDY

Mr. D is a 67-year-old man who was admitted to the hospital because of severe chest pain of 2 hours duration. The sensation of “squeezing” chest pain began suddenly in the afternoon while he was sitting down at home. He noticed that he was sweating, and he felt nauseous, but he did not vomit, he was not lightheaded, nor was he short of breath at that time.

He waited about 20 minutes, but the pain did not get better, and he began to feel some shortness of breath. He was now alarmed, and told his wife about his symptoms. She immediately called 911, and he was taken by ambulance to the hospital.

Past Medical History

Over the past year, the patient had had occasional chest discomfort when exerting himself for about a year. The feeling of tightness in the middle of his chest would always resolve by itself within a few minutes if he rested. In fact, yesterday he had developed the same pain while mowing the lawn, but as usual it had resolved quickly with rest. Nevertheless, he had begun to worry about the pains, and had made an appointment with his internist to be evaluated. The appointment is for next week, however!

Mr. D used to smoke, but quit 25 years ago. His father died of a “heart attack” at age 65. The patient’s cholesterol was 208 at his last check-up six months ago. He has not had high blood pressure or diabetes.

The patient had surgical removal of a malignant tumor of his colon eight years ago. Colonoscopy last year showed no evidence of recurrence.

Physical Examination

Upon admission the patient was alert and oriented, but acutely ill and anxious, and in considerable distress with continued chest pain and shortness of breath. His systolic BP was 90. (His usual was approximately 135/80.) His heart rate was regular at 100 bpm, with frequent extra beats. He appeared to be short of breath and his respiratory rate was 28. Examination of his chest revealed moist crackles (rales) three-quarters of the way up bilaterally. His jugular venous pressure was estimated to be 8 cm. of H₂O. His heart sounds were soft and distant, and there were no audible murmurs. A soft fourth heart sound was audible. The carotid upstroke was normal, and no bruits were heard. His peripheral pulses were thready and his hands and feet were quite cool.

Laboratory Tests

His **white blood cell count** was 11,000 (nl=4K-11K), **hematocrit** 48 (nl=36-48), and platelets were normal. Other labs were:

Sodium	135 (135-145)	CPK	70 (0-200)
Potassium	3.3 (3.5-5.3)	CPK-MB	6% (< 5%)
Chloride	108 (100-112)	SGOT	23 (0-40)
CO ₂	17 (21-30)	LDH	99 (90-275)
BUN	17 (6-20)	CTnT	0.4 (< 0.01)
Creatinine	1.6 (.8 - 1.2)		

The initial **arterial blood gases** (in room air) showed:

$$pO_2=49 \quad pCO_2= 34 \quad pH=7.42$$

He was given oxygen to bring inspired O_2 to 40%, and his blood gases were:

$$pO_2=60 \quad pCO_2= 34 \quad pH=7.42$$

The **electrocardiogram** showed sinus rhythm, frequent multi-form VPBs, and ST elevations in V1 to V6, with loss of precordial R-waves. (This was consistent with an acute anterior myocardial infarction.)

The **chest x-ray** showed bilateral pulmonary edema (fluid in the alveolar spaces).

Hospital Course

He was sent immediately to the cardiac catheterization laboratory where angiography revealed no lesions in the left main coronary artery, but severe (99%) obstruction in the proximal left anterior descending coronary artery. There was significant stenosis of the left circumflex artery. The right coronary artery was occluded proximally and filled via collaterals from the circumflex. Balloon angioplasty with stent placement was performed on the left anterior descending artery with near resolution of the stenosis. His pulmonary capillary wedge pressure was 26, and an intra-aortic balloon pump (IABP) was placed via the left femoral artery. He was transferred to the Coronary Care Unit, and at that time was free of chest pain.

He was treated with aspirin, heparin, IV nitroglycerine, an ACE (angiotensin converting enzyme) inhibitor, and lopressor to maintain a systemic arterial mean pressure between 65 and 70 mmHg. He was also given intravenous doses of the powerful diuretic, lasix, to treat his pulmonary edema.

His course in the CCU was complicated by congestive heart failure despite the IABP. His pulmonary edema led to worsening gas exchange, and his ABG deteriorated to:

$$pO_2=62 \quad pCO_2= 61 \quad pH=7.25$$

It was necessary to provide mechanical ventilation for the patient. He was intubated and ventilated at a rate of 12 breaths per minute with 100% O_2 , and PEEP=0. His ABG improved to:

$$pO_2=75 \quad pCO_2= 38 \quad pH=7.38$$

An echocardiogram performed on the second hospital day showed significant damage to the left ventricle, whose ejection fraction was only 20% (nl = >55%). The LV was enlarged with severe reduction in contraction of the anteroseptal areas. There was a possible thrombus in the LV apex.

A Swan Ganz catheter was placed on the third hospital day to assist in managing his fluid balance and his drug regimen. By careful management of the pulmonary edema and the use of positive inotropic drugs (to strengthen the heart's contractility) and by using a small amount of PEEP on the ventilator, his ABG improved to:

$$pO_2=320 \quad pCO_2= 38 \quad pH=7.38$$

He was weaned off the ventilator and the IABP was successfully weaned and removed by the fourth hospital day.

He was transferred out of the CCU to the "step-down" unit and remained stable. On the eighth hospital day he was discharged to a rehabilitation facility.

His discharge medications included:

Captopril - (an ACE inhibitor) for afterload reduction

Isordil - (a vascular dilator) to reduce venous pressure (preload) as well as afterload and dilate coronary arterioles to improve myocardial oxygen supply

Aspirin - to inhibit platelet function and decrease the likelihood of future arterial thrombosis

Lasix - a powerful diuretic to control fluid accumulation

KCl - to replace excess potassium loss associated with lasix use

Digoxin - a positive inotropic agent

Coumadin - an anticoagulant because of the presence of a blood clot in the dilated left ventricle, and the recent angioplasty.