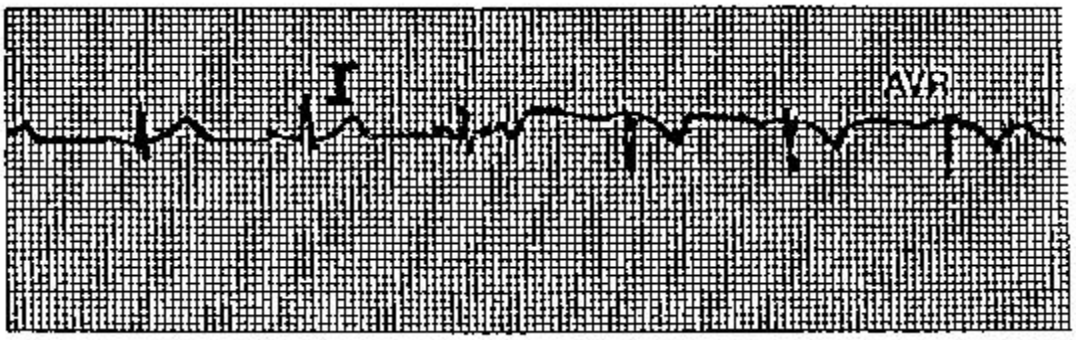
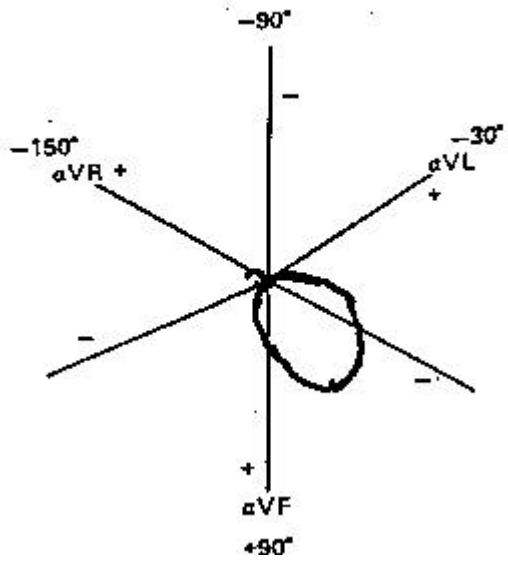
MEDICAL PHYSIOLOGY

Cardiovascular Conference 1 - Quiz 3A

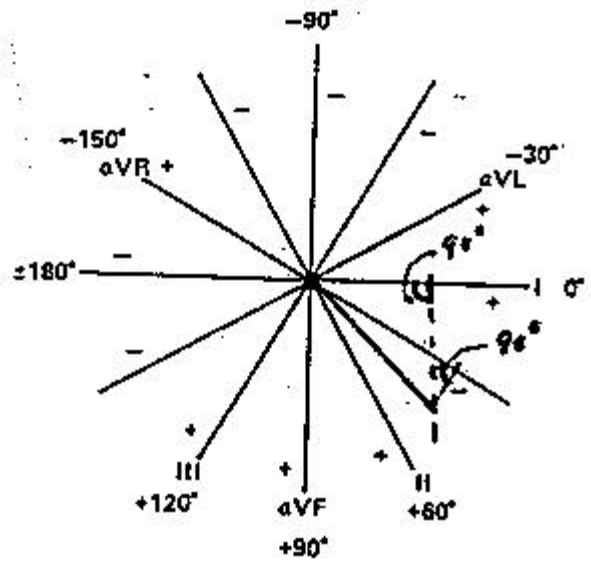
October 5, 2000



1. The above ECGs were recorded when GB was 16 years old. Using Lead AVR draw GB's ventricular depolarization vectorcardiogram.



2. Based on the above ECGs GB's mean electrical axis at the age of 16 was approximately plus 45 **degrees. Show how you arrived at your answer.**



3. The following data were obtained from GB 8 hours after administering Digoxin and doubling his Lisinopril dosage.

	Before Digoxin	After Digoxin
Brachial Artery Pressure(mmHg) =	160/100	130/ 70
Central Venous Pressure(mmHg) =	10	5
Heart Rate (bpm) =	85	75
Left Ventricular End Diastolic Volume (mL) =	220	180
Left Ventricular End Systolic Volume (mL) =	180	12

a. Digoxin (*increased*, decreased) **GB's Left Ventricular Ejection Fraction from** 0.18 **to** 0.33.

Ejection Fraction Before Digoxin = 40/220 = 0.18
Ejection Fraction After Digoxin = 60/180 = 0.33

b. GB's Systemic (Peripheral) Vascular Resistance before administering Digoxin was 2580 **dynes sec/cm5. Show your calculations**

SVR = PVR = ((Mean Arterial Pressure - Central Venous Pressure)/ Cardiac Output) x 1330
SVR = ((120 - 10)/ (85 x 40)/60) x 1330 = (110/56.70) x 1330 = 2580

4. How does Digoxin affect myocardial oxygen consumption?.

Digoxin increases the inotropic state resulting in an increase in myocardial oxygen consumption. In addition the increased inotropic state results in an increased cardiac output which increases arterial blood pressure and hence left ventricular pressure. This increases myocardial wall stress(tension) thereby increasing myocardial oxygen consumption. Off-setting these factors increasing myocardial oxygen consumption is a decrease in LVEDV which decreases wall stress and thus decreases myocardial oxygen consumption. However recall decreasing ventricular

volume only decreases wall stress by the cube root. Finally digoxin decreases the heart rate which will decrease myocardial oxygen consumption. The overall effect of digoxin will be to increase myocardial oxygen consumption. If you answered this question by referring to the data in question 3 in which the effects of digoxin on myocardial oxygen consumption were modified by doubling the dose of Lisinopril you may have concluded that myocardial oxygen consumption fell. Your answer would have been based on decreases in left ventricular systolic pressure, left ventricular volume and heart rate off-setting the increase in myocardial oxygen consumption associated with the digoxin induced increase in the inotropic state. It was not my intention that you refer to the data in question 3 but if you did and your explanations are correct you will receive full credit.

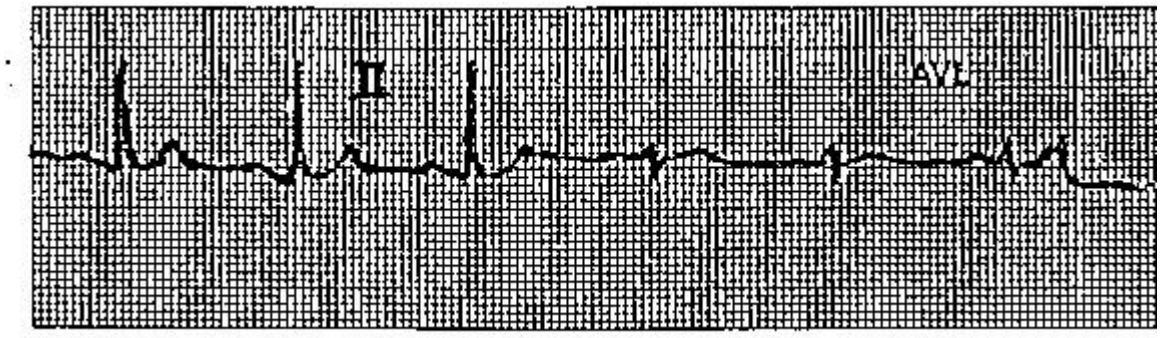
5. The size of GB's R wave in lead V6 when he was brought to the emergency room was larger than that recorded seventy-two hours later. Why did it decrease over this period of time?

Lead V6 is primarily "looking at" at depolarization-repolarization of the lateral left ventricular myocardium. At the time the ECG was obtained in the emergency room the affected region of the myocardium was ischemic/injured but still underging depolarization and repolarization. The magnitude of the R wave is directly proportional to the number of cells depolarizing. Seventy-two hours later these ischemic/injured cells have "died", i.e. they have no membrane potential and thus do not depolarize and repolarize. Consequently the number of electrically active cells "seen" by V6 is decreased leading to a decreased R wave amplitude.

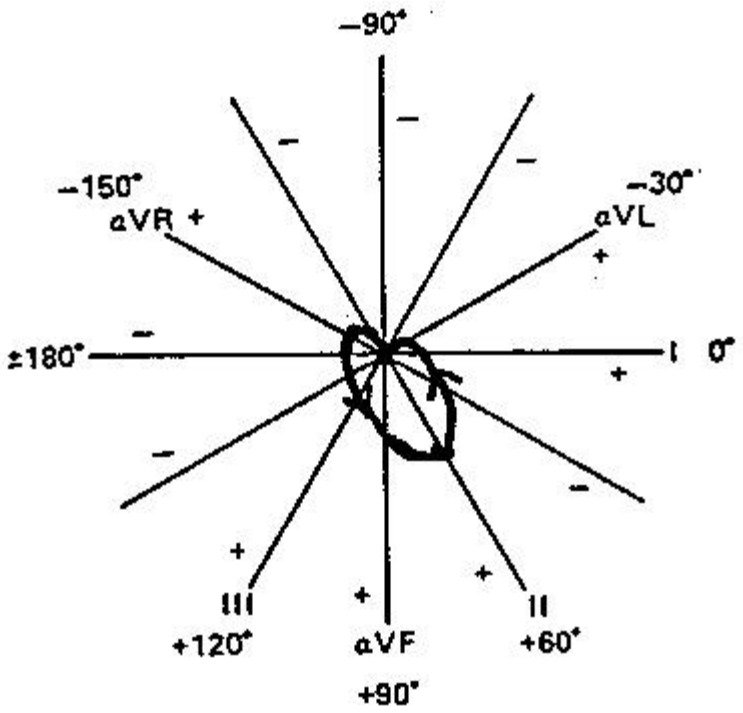
MEDICAL PHYSIOLOGY

Cardiovascular Conference 1 - Quiz 3B

October 3, 2000



1. GB's ECGs when he was 16 years old are shown above. Using Lead II draw his ventricular depolarization vectorcardiogram.



2. At that time GB's mean electrical axis was approximately plus 60 degrees. Show how you arrived at your answer.

The net QRS deflection in lead aVL is essentially zero. Therefore the mean electrical axis is either plus 60 degrees or minus 120 degrees. Since the net deflection in lead II is positive the mean electrical axis is plus 60 degrees.

3. The following data were obtained from GB 8 hours after administering Digoxin and doubling his Lisinopril dosage.

Brachial Artery Blood Pressure = 140/80 mmHg
Central Venous Pressure = 5 mmHg
Heart Rate = 75 bpm
Left Ventricular End Diastolic Volume = 180 mL
Left Ventricular End Systolic Volume = 120 mL

Based on the above data:

a. GB's left ventricular ejection fraction was 0.33**. (Show your calculations)**.
Ejection Fraction = Stroke Volume/End Diastolic Volume
Ejection Fraction = (180 - 120)/180 = 60/180 = 0.33

b. Systemic Vascular (Peripheral) Resistance was 1685 **dynes sec/cm5. (Show your calculations.)**
SVR = PVR = ((Mean Arterial Pressure - Central Venous Pressure)/ Cardiac Output)
SVR = (100 -5)/ (75x60/60) = 95/75 = 1.2666 mmHg/(mL/sec)
SVR = 1.2666 x 1330 = 1685 dynes sec/cm5

4. Describe how Lisinopril affects myocardial oxygen consumption.

Lisinopril blocks the conversion of Angiotensin I to Angiotensin II(AII). AII increases vascular resistance. Thus Lisinopril decreases vascular resistance. A major determinant of myocardial oxygen consumption is systolic left ventricular pressure, i.e. the greater systolic left ventricular pressure the greater myocardial oxygen consumption. Systolic left ventricular pressure is directly related to systemic vascular resistance. Thus decreasing systemic vascular resistance with Lisinopril decreases left ventricular systolic pressure resulting in a decrease in myocardial oxygen consumption.

5. When GB was brought to the emergency room marked ST segment elevations were noted in his precordial leads. Seventy-two hours later there were no ST segment elevations in his precordial leads. Explain why these ST segment changes occurred.

ST segment elevations and depressions result from some ventricular muscle cells being partially depolarized. Consequently during the T-Q segment of the ECG the ECG electrodes record an extracellular potential difference. During the ST segment there is no measureable extracellular fluid potential difference. Since the ST segment is referenced to the T-Q segment, the ST segment is elevated or depressed. The ST segment is elevated if the ECG leads are recording a negative T-Q extracellular potential difference. If the ECG leads are recording a positive T-Q extracellular potential the ST segment is depressed.