ELEC ENG 3BB3:

Cellular Bioelectricity

Notes for Lecture 21

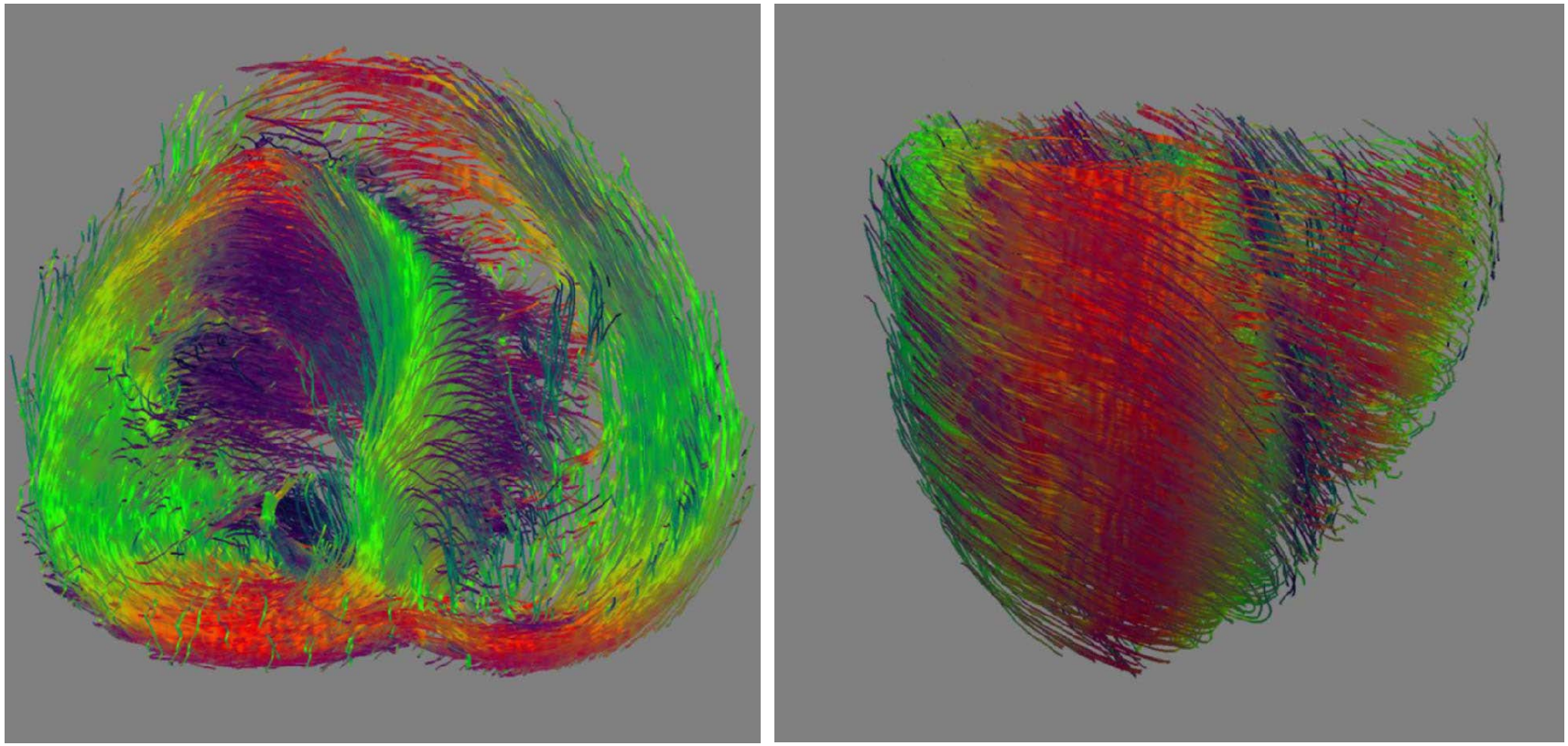
Thursday, February 27, 2014

Free wall activation of the heart:

Cardiac fibers tend to *spiral* around the heart. Consequently, fibers are oriented parallel to the endocardium and epicardium. Contraction of the muscle fibers produces a *wringing action*, which efficiently squeezes out (pumps) the blood.

Very rapid propagation occurs along the fiber axis, but a slower *wavefront* of activation progresses in a direction orthogonal to the fiber axis, due to anisotropic conductances.

Free wall activation of the heart (cont.):



(Zhukov and Barr, IEEE Visualization Conference 2003)

Free wall activation of the heart (cont.):

Hypothetical activation wave.

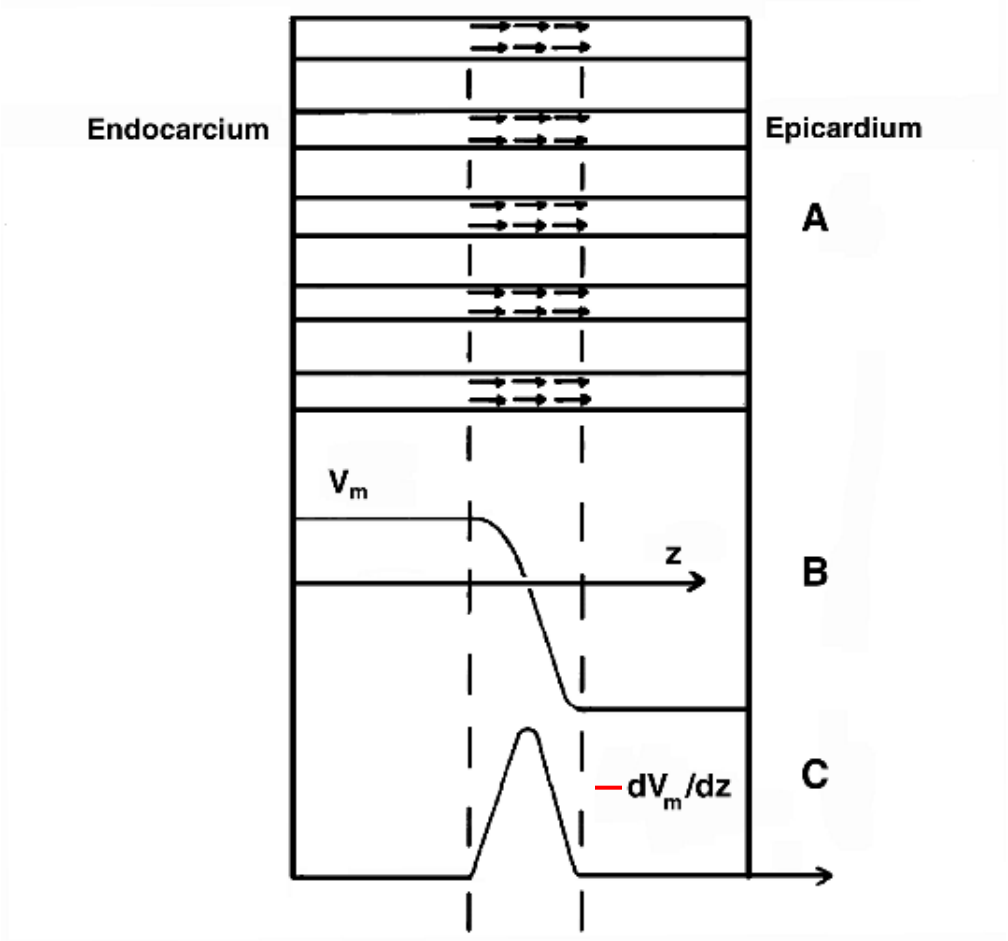


Figure 9.15. Hypothetical Fiber Orientation and Corresponding Activation Wave. A rise time of approximately 1 msec and a velocity of around 50 cm/sec means that the source region would be roughly 0.5 mm thick. From Liebman J, Plonsey R, Gillette PC. eds. 1982. *Pediatric electrocardiography*. Baltimore, MD: Williams and Wilkins.

Free wall activation of the heart (cont.):

Idealized temporal waveform for the cardiac action potential.

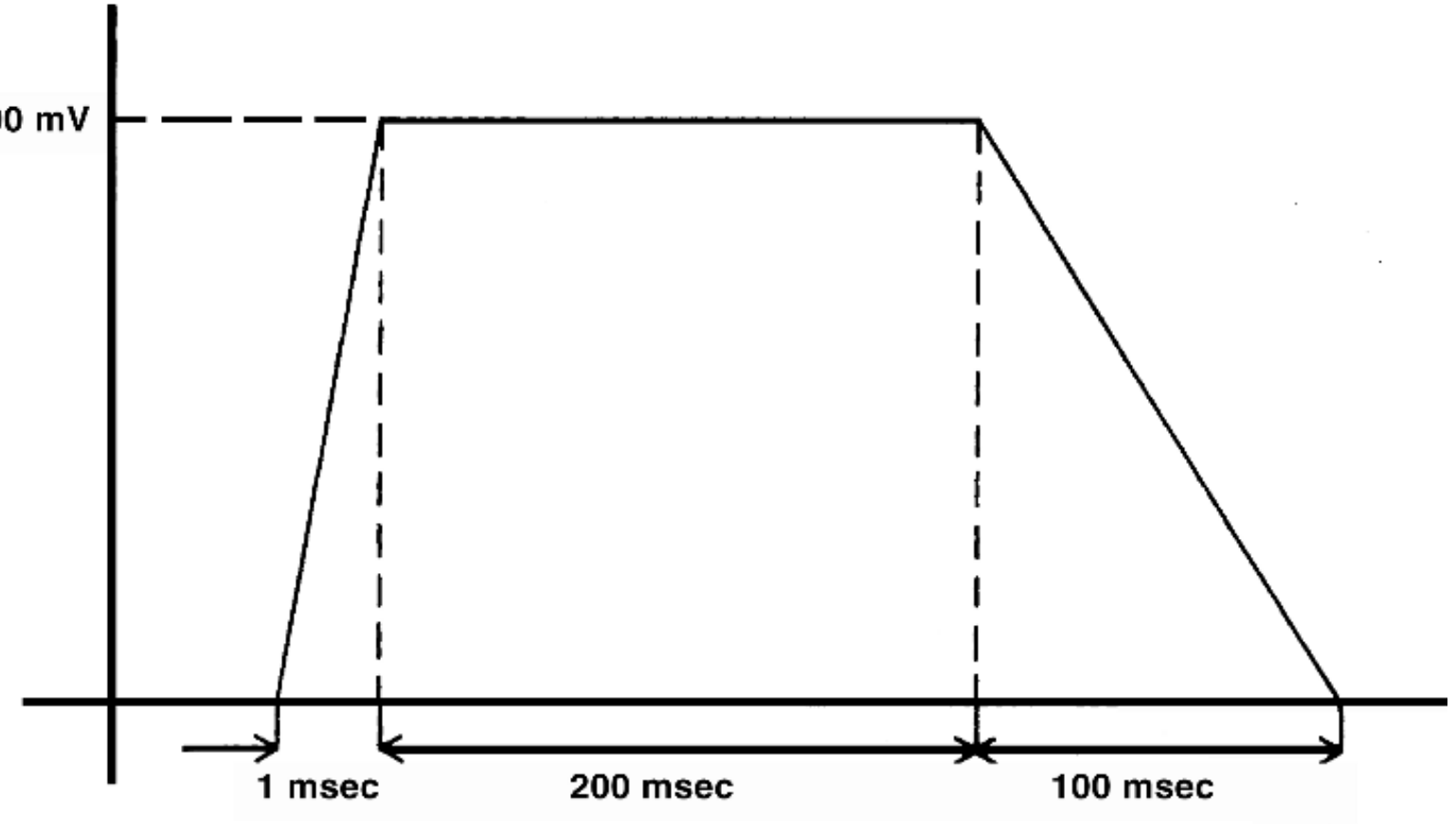


Figure 9.16. Idealized Temporal Cardiac Action Potential Consisting of Rapid Depolarization, Plateau, and Slow Recovery. (The rising phase is not drawn to scale.) Note that at an expected velocity of propagation of 50 cm/sec the spatial counterpart of this temporal action potential would require a tissue of at least 15 cm in thickness.

Source models:

The total source is that of a thick (» 0.5 mm) double layer whose axial density is proportional to ¡$\partial V_m/\partial z$.

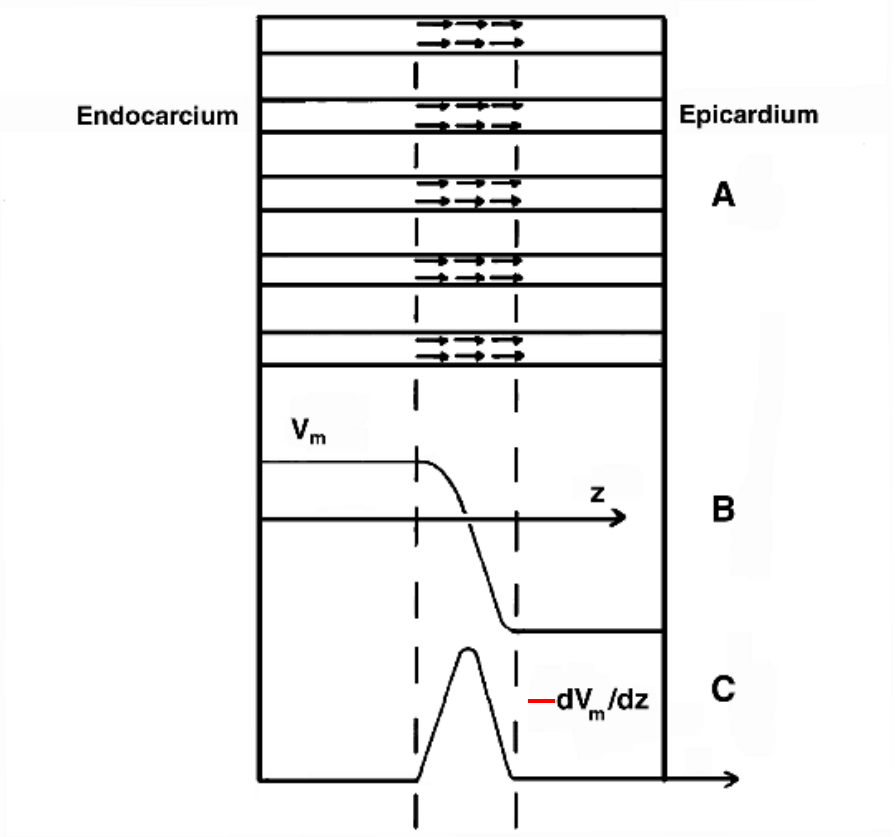


Figure 9.15. Hypothetical Fiber Orientation and Corresponding Activation Wave. A rise time of approximately 1 msec and a velocity of around 50 cm/sec means that the source region would be roughly 0.5 mm thick. From Liebman J, Plonsey R, Gillette PC. eds. 1982. *Pediatric electrocardiography*. Baltimore, MD: Williams and Wilkins.

Source models (cont.):

For field points outside the heart and at a distance large compared to 0.5 mm (e.g., on the body's surface), the axial extent of the source is unimportant and can be considered an ideal double-layer surface (i.e., zero axial thickness) and uniform in the lateral dimension.

Consequently, the activation wavefront (i.e., the isochrones illustrated in Fig. 9.12) can be interpreted as the site of a uniform double-layer source distribution with strength given by $v_{peak} - v_{rest}$.

Source models (cont.):

At any instant of time during heart activation, the source is a distribution of double-layer surfaces. A rough approximation to the source is the vector sum of all elements, as if they are all at the same location.

The resultant *single dipole* is referred to as the **heart vector** (or **heart dipole**):

$$\bar{H} = \int \bar{J}_i \, \mathrm{d}V, \qquad (9.83)$$

where $\bar{J}_i$ is the dipole moment per unit volume as a function of position in the heart.

ECG measurement and analysis:

The voltage measured between two body surface electrodes is known as a *lead voltage* V_ℓ.

For a dipole moment this depends on the lead location, heart location, heart vector, and torso volume inhomogeneities.

However, because the system is linear, it is possible to separate the heart vector from the other influences, such that:

$$V_\ell = \bar{H} \cdot \bar{\ell}, \qquad (9.87)$$

where $\bar{\ell}$ is known as the *lead vector*.

ECG measurement and analysis (cont.):

The *standard leads* (or limb leads) are placed at the extremities (wrists and ankles). The right leg is normally grounded to reduce noise, and the remaining electrodes give rise to three lead voltages:

$$V_{\rm I} = \Phi_{\rm LA} - \Phi_{\rm RA}\,, \tag{9.74}$$

$$V_{\rm II} = \Phi_{\rm LL} - \Phi_{\rm RA}\,, \tag{9.75}$$

$$V_{\rm III} = \Phi_{\rm LL} - \Phi_{\rm LA}\,, \tag{9.76}$$

where RA is right arm, LA is left arm and LL is left leg.

ECG measurement and analysis (cont.):

A typical lead voltage waveform is illustrated below.

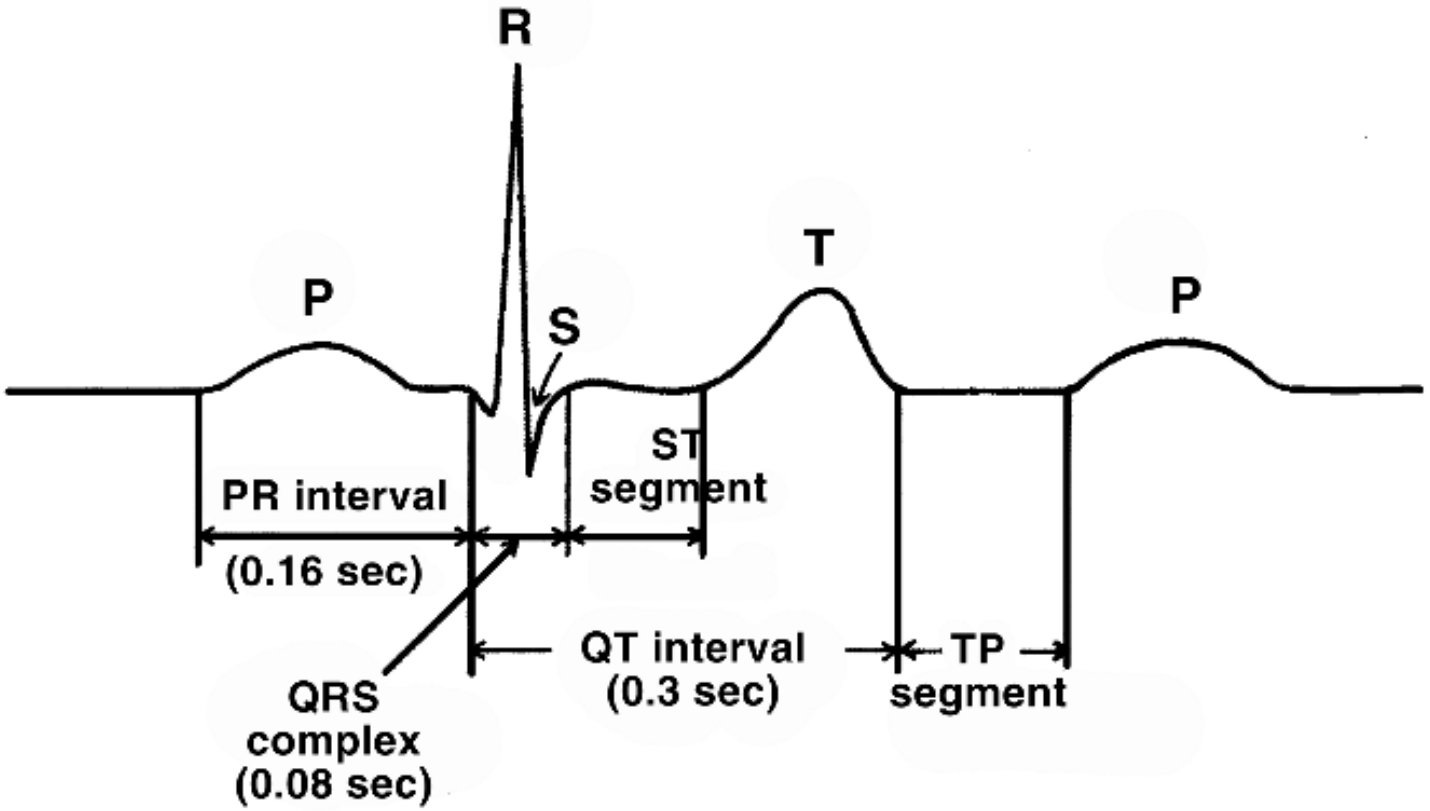


Figure 9.24. Significant Features of Standard (Scalar) Electrocardiogram. Durations given are typical values.

ECG measurement and analysis (cont.):

The three main components of the waveform can be attributed to three different components of heart activation:

- **P wave** — due to *atrial activation*, initiates contraction of the atria
- **QRS complex** — due to *ventricular activation*
- **T wave** — due to *ventricular recovery*

ECG measurement and analysis (cont.):

Other measures of the waveform based on these components that are useful for clinical diagnosis include:

- **PR interval** — a measure of AV conduction time
- **TP segment** — establishes the baseline
- **ST segment** — should be at baseline
- **QT interval** — the total duration of the ventricular systole
- **R–R interval** — reciprocal of heart rate

ECG measurement and analysis (cont.):

Analysis of the heart vector is achieved by considering the geometry of the standard leads and the corresponding lead vectors.

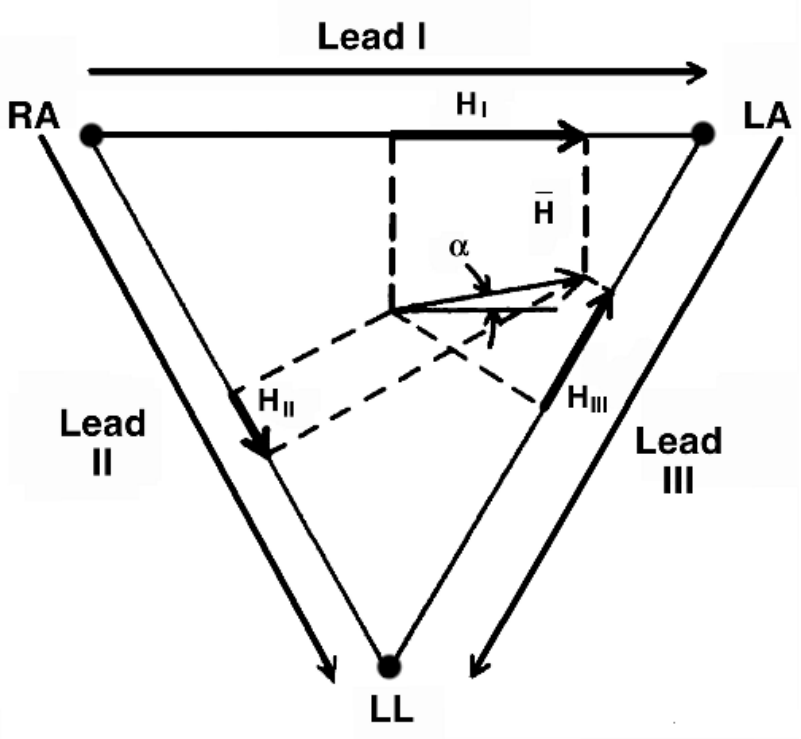


Figure 9.27. The Einthoven Triangle. The sides of the equilateral triangle describe the lead vectors for the limb leads, as shown. For the heart vector, $\overline{H}$, its projections on the triangle sides are labeled H_{I}, H_{II}, and H_{III} (choosing the subscript according to the respective lead); the sign of the lead voltages is found from the dot product of these projections and the corresponding lead vector (e.g., $V_{\mathrm{I}} = \overline{H}_{\mathrm{I}} \cdot \overline{L}_{\mathrm{I}}$). Note that in the illustrated case V_{I} is positive, V_{III} negative, and V_{II} positive. For simplicity in drawing, the lead vectors were given unit magnitudes.

ECG measurement and analysis (cont.):

Modern electronics allow for multiple electrodes to be placed on the torso for a more spatially-precise analysis of lead voltages.

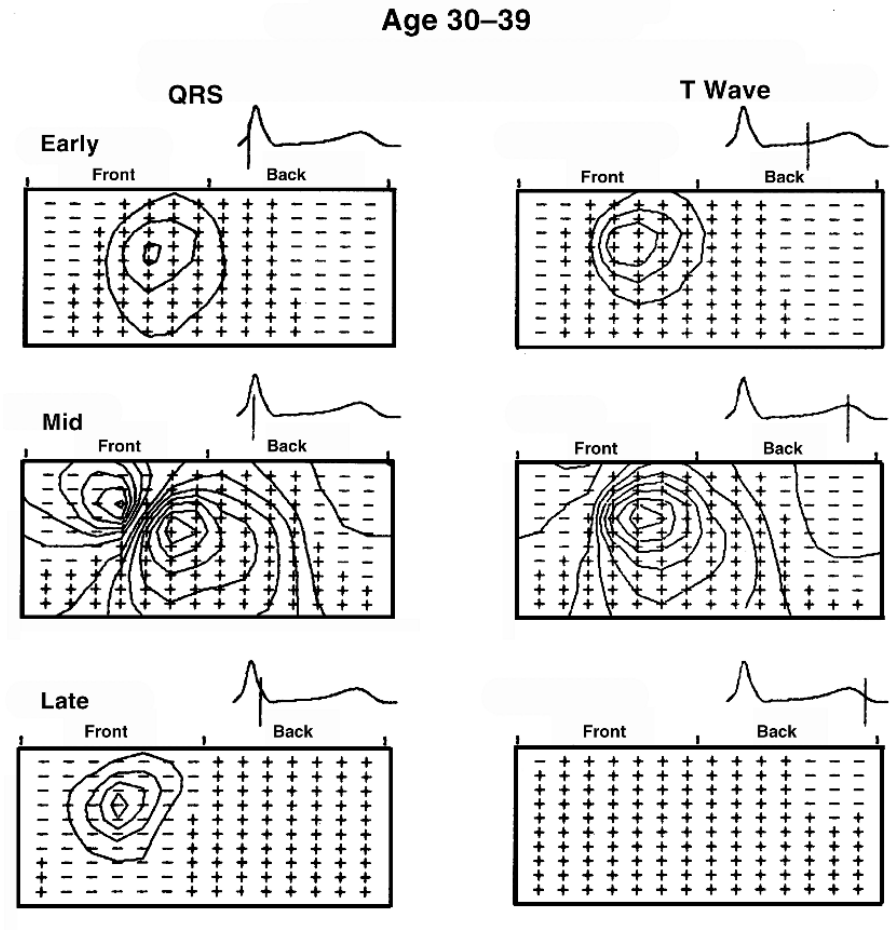


Figure 9.26. Body Surface Electrode Array and Potential Maps of QRS and T at Six Selected Time Instants. The electrodes and potentials on the anterior torso are displayed in the map from the left edge to the center; the potentials over the back (from left to right) continue in the map from the center to the right-hand edge. The map corresponds to the torso cut along the right mid-axillary line and unrolled. The top of the grid is at the level of the clavicles and the bottom at the level of the umbilicus. Shown is an average normal map compiled from subjects in the age group 30–39. Isopotential lines are 0.1 mV apart during QRS and 0.05 mV apart during T. From Green LS, Lux RL, Haws CW, Burgess MJ, Abildskov JA. 1986. Features of body surface potential maps from a large normal population. In *Electrocardiographic body surface mapping*, Ed RTh van Dam, A van Oosterom. Dordrecht: Nijhoff, with the kind permission of Kluwer Academic Publishers.

Luo-Rudy cardiac model:

Heart action potentials are two orders of magnitude longer in duration than action potentials in nerve or striated muscle.

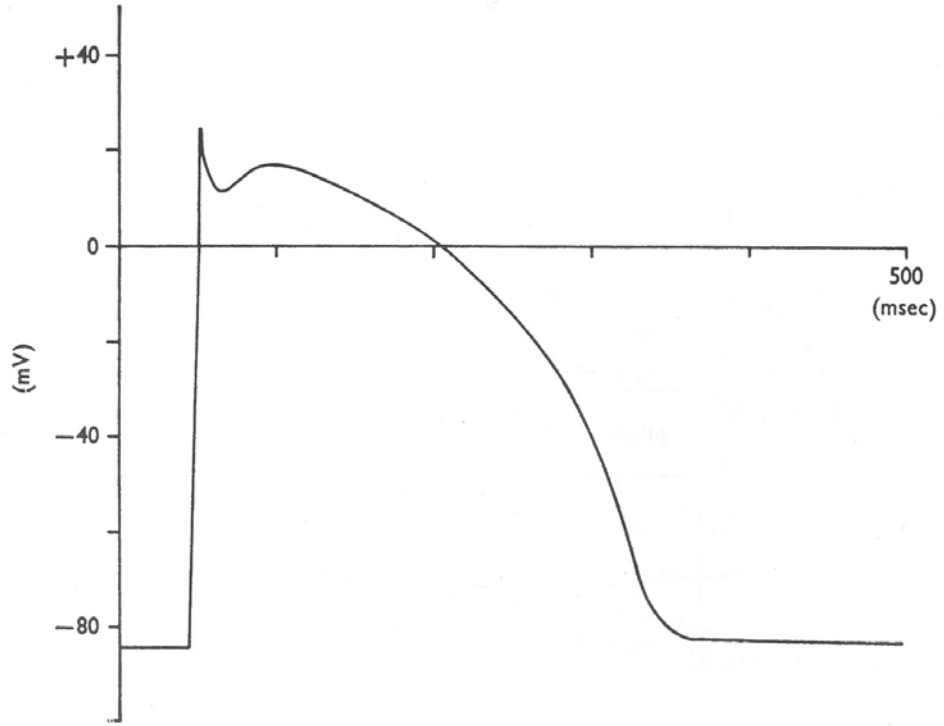


Figure 9.19. Typical Ventricular Cardiac Action Potential (Membrane Action Potential). This is obtained using the Beeler–Reuter model. From Beeler GW, Reuter H. 1977. Reconstruction of the action potential of ventricular myocardial fibers. *J Physiol* **268**:177–210.

Luo-Rudy cardiac model (cont.):

The *Luo-Rudy* (L-R) cardiac cell model greatly expands on the HH formulation.

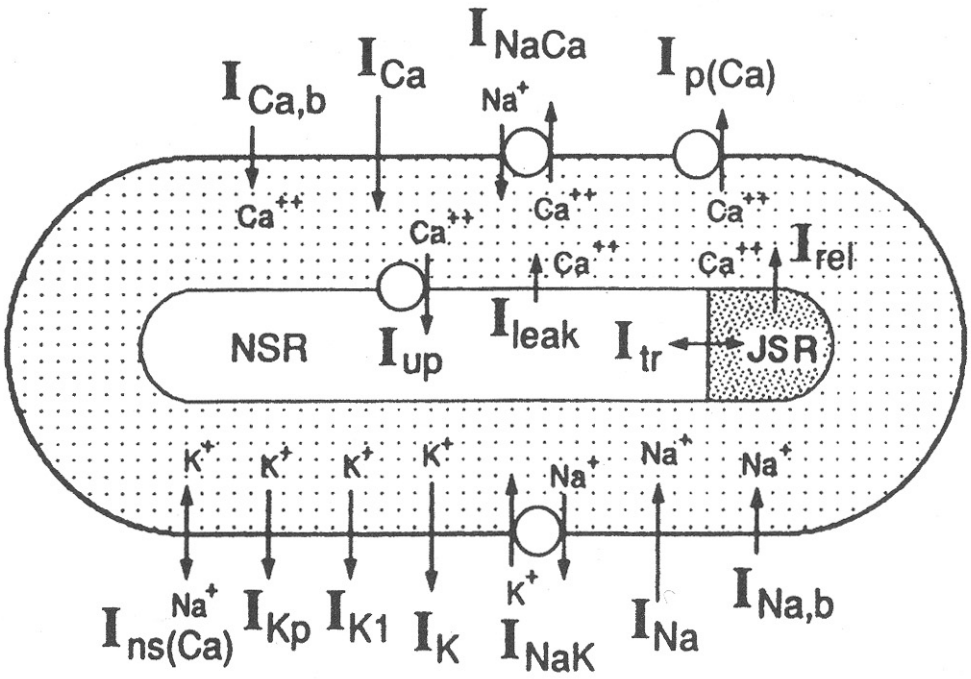


Figure 9.20. Schematic Diagram of Cardiac Cell Model. The abbreviations representing ionic currents are further described in the text. For those representing pumps and ion exchangers the Luo-Rudy paper should be consulted [26]. From Luo C-H, Rudy Y. 1994. A dynamic model of the cardiac ventricular action potential. *Circ Res* **74**:1071–1096.

Luo-Rudy cardiac model (cont.):

The L-R model includes:

- I_{Na}: a fast Na^+ current, which includes a slow inactivation parameter j in addition to the HH activation and inactivation parameters m^3 and h, respectively.
- I_K: a time-dependent potassium current, with an activation parameter X^2 and an inactivation parameter X_i. Additionally, the maximum potassium conductance is not assumed to be constant but rather depends on $[K]_o$, and the reversal potential allows for a small sodium current through the potassium channel.

Luo-Rudy cardiac model (cont.):

- I_{K1}: a time-independent potassium current, which again has a maximum potassium conductance that is not assumed to be constant but rather depends on $[K]_o$. This channel has an activation parameter that acts instantaneously.
- $I_{K\rho}$: a potassium plateau current, which is time independent and is insensitive to $[K]_o$.
- I_{Ca}: a calcium current, with instantaneous activation and inactivation parameters d and f, respectively. Its I-V relationship is described by the GHK current equation, and potassium and sodium flux through the channel is considered.