The Effect of High Lung Conductivity on Electrocardiographic Potentials

Results from Human Subjects Undergoing Bronchopulmonary Lavage

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SUMMARY The effect of increased lung conductivity on ECG potentials was studied in human subjects undergoing pulmonary lavage of a whole lung. In this procedure, the air in the lung is replaced by physiologic saline solution, which is a highly conductive fluid. The same situation was simulated theoretically with an eccentric spherical model of the heart and torso. Both the experimental results and theoretical simulations show a decrease in body-surface potentials as the lung conductivity increases. In particular, a large decrease was observed in the posterior vector and the scalar Z lead both experimentally and theoretically. The model simulation shows that the scalar Z lead is maximal at a conductivity value that is very close to the typical normal lung conductivity, so that low voltages are predicted for low lung conductivities as well.

The electrical activity of the heart is manifest at the body surface through the intervention of the conducting medium in which the heart is embedded. The surface potentials, which are recorded as ECGs, therefore reflect both the cardiac generators and the properties of the inhomogeneous volume conductor. Using a theoretical model in which the major torso inhomogeneities were included, Rudy and Plonsey demonstrated the importance of the lung compartment in determining the distribution of body-surface potentials. The functional dependence of the surface potential on lung conductivity is dominated by the lung interaction with the surrounding muscle layer. When this interaction is taken into account, potentials lower than normal are obtained for abnormally high, as well as for abnormally low, lung conductivities. The model prediction of low surface potentials in cases of low lung conductivity is in keeping with the clinical findings of low ECG voltages in patients with obstructive lung disease, as well as with the reduction in potentials observed during experimentally induced over-inflation of the lungs. On the other end of the spectrum, abnormally high lung conductivity occurs clinically in cases of edema, pulmonary congestion or infiltration and was suggested by Lepeschkin as the main cause for the decrease in ECG voltages that accompanies these conditions. Both low and high lung conductivity were induced by Lepeschkin and coworkers in experimental animals by creating a left-sided complete pneumothorax (low conductivity), or by replacing the air in the left lung with physiologic saline (high conductivity). The ECG potentials decreased in both cases.

We studied the effects of increased conductivity of the right and left lungs on the measured surface potentials in human subjects, in which the geometry of the volume conductor differs markedly from that in dogs. The measurements were performed on patients undergoing pulmonary lavage with physiologic saline. We assumed that filling the lung briefly with saline does not modify the myocardial electrical sources and has only a minimal effect on the geometry of the volume conductor. This procedure allows the study of the isolated effect of increased lung conductivity. The same effect is simulated theoretically using the eccentric spheres model, and the simulation serves as the basis for interpreting the experimental results.

Methods

Experimental

Seven subjects were studied. In four subjects, a single segmental bronchus was filled with 100–150 ml of sterile physiologic saline (conductivity at body temperature is 0.0198 mho/cm). Because the filling had no significant effect on the ECG recordings, we concluded that this small volume of saline produced a perturbation that was too small to significantly affect the surface potentials. In three subjects, lavage of a whole lung was performed under general anesthesia, using a double-lumen Carlens endotracheal tube. Patient 1 (a 29-year-old male) had cystic fibrosis; Patients 2 (an 18-year-old female) and 3 (a 16-year-old female) suffered from alveolar proteinosis. To completely fill the lavaged lung with saline, the patient was first ventilated for 15 minutes with 100% oxygen, which is rapidly absorbed into the circulation. Saline was then instilled into the lung in 100-ml aliquots over 10–15 minutes to a filling pressure of approximately 25 cm H2O. The untreated lung was ventilated with 100% oxygen throughout the procedure. Scalar orthogonal ECG recordings and vectorcardiograms were obtained by the Frank system; electrodes were placed in the fourth interspace, as suggested by Langner and associates for the supine position. The potentials were measured before the lavage, while the lung was maximally distended with saline, and after the saline was aspirated. This sequence was repeated several times during the lavage, and in patient 1 on two occasions 2 days apart. In all cases the results were reproducible and the effect of lung filling was con-

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simulated heart spherical muscle shell because the activation spreads anterior the wall of the left ventricle is highly idealized, but highly realistic, representation of the source during most of the QRS. A heart with a radius of 5 cm in a torso with a radius of 12.5 cm was considered typical normal geometry. The minimal distance between the anterior chest wall and the anterior wall of the heart is typically 1 cm and this value was used in our calculations. The normal values used for conductivity variables are: blood = 0.006 mho/cm; myocardium = 0.002 mho/cm; lung = 0.0005 mho/cm; skeletal muscle = 0.00125 mho/cm; fat = 0.0004 mho/cm; pericardium = 0.001 mho/cm. A value of 0.0198 mho/cm was used for the conductivity of physiologic saline.

Experimental

The results from patient 1 are shown in figure 1. The lavage was performed on the right lung. In panel A the vectorcardiograms recorded before the lung was filled with saline are compared with those obtained during the lavage. The scalar lead recordings are shown in panel B. That this patient had cystic fibrosis is reflected in the “empty” vectorcardiograms, which show abnormally posterior vector and right ventricular hypertrophy. The most obvious change in the vectorcardiogram is an overall decrease in the potential magnitude when the lung is filled with saline. In particular, the sagittal plane vectorcardiogram shows a pronounced decrease in the posterior, anterior and inferior directions. The posterior and anterior vectors are affected the most; the maximal vector in the transverse plane (directed posterior and to the right) is reduced by 0.6 mV (22.2%), and the maximal vector in the sagittal plane (directed posterior and superior) is reduced by 0.85 mV (28.8%). In contrast, a small increase in the vector to the right is apparent in the frontal plane vectorcardiogram. The following changes are observed in the scalar leads: X lead — an increase of 0.14 mV (14%) in the S wave; Y lead — a decrease of 0.5 mV (29.4%) in the R wave and a decrease of 0.2 mV (16.6%) in the S wave; Z lead — a decrease of 0.8 mV (28.6%) in the R wave and 0.2 mV (25%) in the Q wave.

The results from patient 2 are shown in figure 2. The right lung was filled with saline. Again, the most apparent effect is a decrease in the posterior direction, clearly seen in the transverse and sagittal vectorcardiograms as well as in the scalar Z lead. The maximal decrease in the transverse plane vector is 0.95 mV (40%) and in the sagittal plane 0.7 mV (28.6%), while the R-wave amplitude in the scalar Z lead is reduced by 0.6 mV (27.3%). Also, the rightward vector (frontal and transverse plane vectorcardiograms) increased

\*The pericardial conductivity was provided by Dr. J. Clark from measurements of canine pericardium.
during late QRS, and this was manifest as a 0.4-mV increase in the S wave in the scalar X lead as well.

The effect of filling the right lung in patient 3 is shown in figure 3. An overall reduction of potentials is seen in the transverse and sagittal plane vectorcardiograms. As before, the most apparent decrease occurred in the posterior vector in the transverse plane (maximal decrease of 0.65 mV [35.1%]). The anterior vector also decreased (0.3 mV [35%] in the sagittal plane) early in the QRS. In contrast, the vector to the right increased late in the QRS. The above changes are reflected in the scalar leads as a 0.44-mV decrease in the R wave in the Z lead, a 0.2-mV decrease in the Q wave of the same lead, and a 0.26-mV increase in the S wave in the X lead.

In patient 3, the left lung was also filled with saline (fig. 4). The ECG changes caused by filling the left lung were qualitatively similar to those produced by filling the right lung (fig. 3). In both cases, the most striking effect is the decrease in the posterior direction reflected in the transverse and sagittal plane vectorcardiograms, as well as in the scalar Z-lead ampli-
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Figure 4. The effect of filling the left lung in patient 3. Format as in figure 1.

Quantitatively, the decrease in the posterior direction is much greater when the left lung is filled. The maximal decrease in the transverse plane is 1.25 mV (71.4%) for the left lung and 0.65 mV (36%) for the right lung. The decrease in the R-wave amplitude in the Z lead is 0.96 mV (66.6%) when the left lung is filled. The left lung lavage also caused a 0.26-mV (37.8%) decrease in the Z-lead Q wave, and a small (0.1-mV) increase in the X-lead Q-wave amplitude.

Theoretical

The following results were obtained for a double-layer source located in the posterior part of the spherical heart wall, midway between the endocardium and the epicardium. This location corresponds to activation of the posterior left ventricular wall. The central angle of the double layer was taken to be 60°. For this angle, the surface area of the source is sufficiently extensive to represent a typical activation wave.

The effect of increased lung conductivity on the surface potential distribution is shown in figure 5. Two cases are considered: A simulates the case with typical lung conductivity of 0.0005 mho/cm; B simulates the case of saline-filled lung by assigning the value of 0.0198 mho/cm (conductivity of physiologic saline) to the conductivity of the lung region. The result is a decrease in potential amplitude everywhere on the torso surface. Obviously, potential differences between any two surface electrodes are diminished as well. Quantitatively the peak potential (at θ = 0°) is reduced by 81%.

The largest experimentally measured change caused by the lung filling occurred in the Frank Z lead, so this lead was studied theoretically as well. Because of azimuthal symmetry, the Z-lead voltage for the eccentric spheres can be very well approximated by the potential difference between the midanterior (θ = 0°) and midposterior (θ = 180°) surface points. The computed Z-lead voltage (V_Z) as a function of lung conductivity is shown in figure 6. V_Z attains a maximum at a conductivity that is very close to normal physiologic values, so that voltages lower than normal are obtained for abnormally high as well as abnormally

Figure 5. The effect of increased lung conductivity on the surface potential distribution. The potential is plotted as a function of the polar angle θ. θ = 0° corresponds to the midsternal line, θ = 180° and θ = −180° is the same point, representing the midposterior line. (A) Typical lung conductivity. (B) Conductivity of lung region is equal to conductivity of physiologic saline.
low lung conductivities. For lung conductivity equal to the conductivity of saline, the reduction was 82%.

**Discussion**

The theoretical effect predicted by the eccentric spheres model is a reduction in the amplitude of ECG potentials everywhere on the body surface. The largest decrease occurred in the Z-lead voltage, which was reduced by 82% when the conductivity of the lung region was increased from 0.0005 mho/cm (normal) to 0.0198 mho/cm (saline conductivity). A similar phenomenon was observed experimentally in all cases. The most striking effect caused by filling either lung with saline is a large decrease in the posterior vector and in the R-wave amplitude in the Z lead. The average decrease for the four cases reported here is 39%; a maximal decrease of 66.6% occurred when the left lung was filled. That the largest effect is on the Z-lead voltages is consistent with the fact that most of the lung volume lies posterior to the heart. (This effect is even more pronounced when the lung is filled with saline and the subject is in the supine position, as was the case in these experiments.) Not surprisingly, the theoretical simulation predicts a larger decrease in Vz than the experimentally measured change. In the model, the conductivity of the whole lung region is set equal to the conductivity of saline, whereas in the experimental procedure, only the air volume in the lung is replaced by saline. As a result, the increase in conductivity of the lung region is smaller than in the experimental setting. A theoretical decrease in Vz of 66.9% (comparable to the maximal experimental decrease of 66.6%) occurred when the conductivity of the lung region was set equal to 0.01 mho/cm (50.5% of saline conductivity, or 20 times the normal conductivity of lung). The increase in the S wave in the X lead when the right lung was filled cannot be explained by the theoretical results. A possible cause is the asymmetry in the x direction in the experimental setting (only one lung filled) in contrast to the azimuthal symmetry of the theoretical model. Another possibility is a slight change in the heart position caused by the shift in the lung due to the weight of the saline. A shift of

In conclusion, identification of increased lung conductivity as the factor responsible for the decreased ECG potentials during pulmonary lavage was confirmed by the results of the theoretical simulation. Clinically, these results imply that abnormally low surface potentials are to be expected in cases of pulmonary congestion and edema. This information permits the interpretation of ECG effects in more complicated situations. For example, the decrease in surface potentials in congestive heart failure was attributed to the increase in the heart volume.18 Because an increase in heart size was shown to augment the potential,19 another factor acting to decrease the potential must be operative and must predominate in this situation. This factor is probably the pulmonary congestion caused by the congestive heart failure. In addition, the theoretical model simulations show that Vz is maximal at a nearly normal lung conductivity. A fivefold decrease in lung conductivity from the normal value reduces Vz by 56%. Clinically, therefore, low voltages are to be expected in conditions that produce abnormally high (pulmonary edema) as well as abnormally low (obstructive lung disease) lung conductivities.

**Acknowledgment**

The authors thank Rennie Fraenkel for her help in obtaining the experimental data.

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The Pathologic Correlates of the Electrocardiogram: Complete Left Bundle Branch Block

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SUMMARY To assess whether gross pathologic differences exist between hearts with left bundle branch block (LBBB) and left-axis deviation (LAXD) and those with LBBB and a normal frontal plane axis, we examined 70 hearts with LBBB in a series of 1410 sequential dissections (5%). Thirty-two hearts had LAXD and 34 had normal axes on the correlative ECG. Left ventricular enlargement occurred frequently (93%). No significant differences were found in age distribution, left ventricular weight, coronary anatomy or infarct location. Quantitative analysis revealed larger inferoposterolateral and apical infarcts in hearts with LBBB and LAXD (p < 0.01). The accuracy of various electrocardiographic signs of left ventricular enlargement and myocardial infarction in the presence of LBBB was assessed. Voltage criteria and QRS duration poorly define anatomic chamber enlargement. Anterior infarction is suggested by a q or pathologic Q wave in lead I, a q wave in leads I, V₂ and V₆, or notched S waves in V₅ or V₆. Pathologic Q waves or ST shifts in the inferior leads have high diagnostic specificity but low sensitivity for inferior infarction.

THE CLINICAL PRESENCE of complete left bundle branch block (LBBB) on the ECG is frequently associated with ischemic or hypertensive heart disease and cardiac enlargement. The prognosis of LBBB is excellent when detected at an early age in an otherwise normal subject. However, prospective long-term follow-up in an older population shows that newly acquired LBBB carries a 50% 10-year survival. Concurrent left-axis deviation (LAXD) and LBBB reportedly portend a greater incidence of myocardial dysfunction, more advanced conduction system disease and a greater cardiovascular mortality than in patients with LBBB and a normal frontal plane axis. Whether the presence of LAXD with LBBB indicates a greater degree of myocardial pathology remains unknown. LBBB can obscure the ECG findings of myocardial infarction (MI) and left ventricular enlargement (LVE); however, in the presence of LBBB, there may be subtle ECG changes that indicate the location of myocardial infarction or the presence of LVE. Some of these contentions remain controversial.

In this study we examined the gross pathologic correlates of LBBB to determine whether pathologic differences exist between hearts with LBBB and LAXD, and those with LBBB and a normal frontal plane axis. We also sought to establish the value of the ECG in detecting both LVE and the anatomic location of MI in the presence of LBBB.

Methods

During a 6-year period, information was gathered on 1500 autopsied male patients from a general hospital in whom pathologic examination of the heart was supported by correlative ECGs demonstrating supraventricular antegrade conduction. All hearts were dissected by two of the authors according to strict protocol. In this study, the presence of myocardial deficits, either fibrosis or necrosis, of any degree is described by the term infarction. The location of such myocardial deficits was recorded on detailed protocol sheets. The myocardium of the left ventricle was subdivided into septal, anterior, and inferoposterolateral areas using clear anatomic boundaries. Each of these areas was further subdivided into two basal, two central and one apical zone, for a total of 15 zones (fig. 1). The

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Supported in part by NHLBI grants HL-19768 and HL-16901, a grant from the Louisville and Jefferson County and Kentucky Heart Associations, and an award from the Veterans Administration.


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Circulation. 1982;65:440-445
doi: 10.1161/01.CIR.65.3.440

Circulation is published by the American Heart Association, 7272 Greenville Avenue, Dallas, TX 75231
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Print ISSN: 0009-7322. Online ISSN: 1524-4539

The online version of this article, along with updated information and services, is located on the World Wide Web at:
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