The Electrocardiogram after Mastectomy

By Charles S. LaMonte, M.D., and Alvin H. Freiman, M.D.

The characteristics of the electrocardiographic tracing are determined by three independent variables. The first of these involves the potentials generated by the myocardium during depolarization and repolarization. These potentials can be altered by a wide variety of physiologic states, as well as by intrinsic disease of the myocardium. The second factor relates to the properties of the extracardiac tissues that transmit the electrical potentials from the myocardial generator to the recording electrode. The final factor concerns the characteristics of the recording instrument.

Attempts to evaluate the second factor, namely, the effect of the transmitting tissues on the recorded electrocardiogram, are at present only qualitative. It is frequently difficult to determine whether an observed change or abnormality is due to an alteration in the potential generated by the myocardium or to modification of the impulse by the transmitting tissues. Furthermore, single well-defined changes in the transmitting tissues are uncommon. For example, Simonson has pointed out the variation in amplitude of the complexes as an interdependent function of age, body build, chest diameter, and sex. These considerations suggest the importance of evaluating the effect of discrete changes in the transmitting tissues on the recorded electrocardiogram. The present authors therefore compared retrospectively the electrocardiograms of 16 patients recorded prior to left mastectomy and at some time after surgery. In one or more precordial leads, 38 per cent showed an increase in voltage of the QRS complexes varying between 1.0 and 2.6 millivolts. However, because there was no control of precordial lead placement, sensitivity standardization, or changes in the patients' physical condition these changes in the recorded electrocardiograms could not be correlated solely with anatomic changes after left mastectomy. For these reasons a prospective study was undertaken to evaluate the effect of mastectomy on the electrocardiogram. In addition to clarifying the interpretation of electrocardiograms after such surgical procedures, it was thought that this investigation would demonstrate the effect on the electrocardiogram of a predictable alteration of the transmitting tissues.

Materials and Methods

The study was performed over a 5-month interval on all patients admitted for mastectomy to the James Ewing Hospital, on whom controlled electrocardiograms were obtained before and after surgery. Of 39 patients admitted to the study, 20 underwent left mastectomy; 19, right mastectomy. The mean age of the former group was 55.9 years; of the latter, 54.9 years. Table 1 lists the patients who underwent radical mastectomy and those, including men, who had a simple mastectomy.

One of the investigators saw each patient preoperatively and recorded a history and physical examination, with emphasis on symptoms or physical findings of cardiopulmonary disease. The following studies were performed: six-foot chest roentgenogram, complete blood count, urinalysis, and determination of the blood urea nitrogen and fasting glucose levels. When the patient was seen after surgery, the history and physical examination were repeated.

Preoperatively, and as soon after surgery as wound healing would allow, the same investigator recorded a 16-lead electrocardiogram on each patient. The two tracings were recorded within a time interval varying from 3 to 18 weeks. The recording instrument was a single-channel direct-writing machine.* The recorded leads were the conventional six limb leads and precordial leads.

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*Sanborn Visko Cardiette Model 100. Sanborn Company, Waltham 54, Massachusetts.
ELECTROCARDIOGRAM AFTER MASTECTOMY

V₃R through V₆ as prescribed by the New York Heart Association² and in the left midclavicular line in the third (V₄₃A), fourth (V₄₄R), fifth (V₄₅C), and sixth (V₄₆D) intercostal spaces. Positioning of the precordial leads in relation to ribs or intercostal spaces was not complicated postoperatively by the five partial rib resections, since the area of resection was relatively small. Prior to placement of the electrodes, electrode jelly† was applied to the patient’s skin with a wooden applicator to the point of mild erythema. The sensitivity of the recording machine was calibrated at the end of each lead at 1 cm. per millivolt, and was unchanged throughout the study. Except for one patient, the configuration of the precordial complexes in the same subject before and after operation was unchanged, and the difference between the two horizontal electrical axes of the heart was less than seven degrees. The single exception was a patient undergoing left mastectomy, who was thought on the basis of her electrocardiogram to have had a silent apical myocardial infarction. This patient was included in the study. An attempt to quantitate the amount of tissue removed at surgery was unrewarding because the surgical specimens contained varying amounts of axillary fat and tissue.

After the electrocardiograms had been examined for technical adequacy, the amplitude of all the complexes, namely, P, Q, R, S, peak-to-peak QRS, and T waves, was determined on three consecutive complexes and the mean value was recorded. All measurements were made from the lower border of the baseline to the lower border of the deflection inscribed. The arithmetic change in the amplitude of the complexes was determined by subtracting the values preoperatively from those postoperatively. For each complex, the mean change of amplitude was calculated in the group of patients undergoing left mastectomy and in those undergoing right mastectomy. Finally, the significance of difference between means was determined by Student’s t test to evaluate whether the postoperative increase in amplitude of a given complex was statistically significant and whether the increase in a given complex after left mastectomy was statistically greater than after right mastectomy. If the probability that a given increase could be due to chance alone was .05, this was considered only moderately significant. If the probability was .01 or .001 that chance alone could account for an increase, however, this was considered highly significant.

Results

The results of the investigation are summarized in table 2. This indicates the mean change in amplitude of each complex after either left or right mastectomy, the probability that an observed increase could be due to chance alone, and the probability that chance alone could account for the greater increase seen after left mastectomy than after right mastectomy.

Figure 1 demonstrates the change of amplitude of the precordial R wave after left or right mastectomy. Statistically significant increases in R-wave amplitude occurred in leads V₁ to V₄C after left mastectomy with statistically less significant increases in V₃R and V₆. After right mastectomy statistically significant increases were noted in leads V₁ and V₆. In leads V₃ through V₅ the increase was significantly greater after left than after right mastectomy.

Table 1

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*Radical mastectomy with chest wall resection involves excision of the breast tissue and overlying skin and areola, pectoral musculature, axillary fat and lymph nodes, short sections of the ribs and costochondral cartilage, and internal mammary lymph nodes. It generally involved tissue medial to the anterior axillary line and superior to the sixth intercostal space in the midclavicular line.

†Radical mastectomy entails resection of the breast tissue and overlying skin and areola, pectoral musculature and axillary fat and lymph nodes.

‡In simple mastectomy, the breast tissue alone is resected; skin and areola may not be excised.

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Table 2

Mean Change of Amplitude of Complexes after Left or Right Mastectomy, (Tenths of Millivolts)

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*P_L is the probability that chance alone could account for the increase in amplitude after left mastectomy.
†P_R is the probability that chance alone could account for the increase in amplitude after right mastectomy.
‡P_diff is the probability that chance alone could account for the greater increase seen after left mastectomy than after right mastectomy.
Figure 2 indicates the change of amplitude of precordial S waves after left or right mastectomy. These were statistically significant after left mastectomy in leads V₃R to V₄D and after right mastectomy in V₃R, V₁, and V₄. Only in V₂ was the increase after left mastectomy statistically significantly greater than after right mastectomy.

Figure 3 tabulates the change of amplitude of precordial QRS waves after left or right mastectomy. After left mastectomy statistically significant increases were present in leads V₃R to V₄C, and V₆. After right mastectomy the increases were statistically significant in leads V₃R, V₁, and V₆. The changes were statistically significantly greater after left mastectomy in V₂ to V₄C than after right mastectomy.

Figure 4 demonstrates the change of amplitude of precordial T waves after left or right mastectomy. These were statistically significant in leads V₁ to V₄C after left mastectomy and in leads V₃ to V₆ after right mastectomy. There was no statistical difference between these two groups.

Figure 5 compares the average change in
absolute amplitude with the mean percentage change of the precordial T waves after left and right mastectomy.

Figure 6 compares the average change in absolute amplitude with the mean percentage change of the precordial QRS complexes. In figure 6 it is worth noting the systematic trend from point to point on the chest wall after left mastectomy.

It is of interest to consider these changes in relation to the criteria for the electrocardiographic diagnosis of left ventricular hypertrophy. Among the 19 subjects who underwent right mastectomy, three satisfied the criterion of 3.5 millivolts or more in $S_{v1}$ plus $R_{v5}$ both before and after surgery. No additional patients satisfied this criterion after right mastectomy. In contrast preoperatively none of the 20 patients undergoing left mastectomy satisfied this or any other criterion for the diagnosis of left ventricular hypertrophy. After operation nine satisfied this criterion, and in some cases the sum of the voltages was nearly 6 millivolts.

**Discussion**

The present study examines the effect on the electrocardiogram of acute changes solely...
in the transmitting tissues—in this case, amputation of the breast and other soft tissue of the anterior thoracic wall. In the standard and augmented unipolar limb leads, no consistent change in any of the complexes was noted after either left or right mastectomy; there was no significant difference between the changes in the two operative groups.

**P Waves**

In the precordial leads, statistically significant increases in P-wave amplitude were noted after both left and right mastectomy. There was, however, no significant difference between the two groups. In view of the statistical equivalence of the two groups and the presence of more significant changes in other complexes, no definite conclusions concerning the changes observed in the P waves is warranted.

**T Waves**

Increases in the T waves were seen in most of the precordial leads after both left and right mastectomy, but there was no consistently greater change noted after left mastectomy. In addition, the evaluation of T-wave changes is more difficult in view of the inherent variability of these complexes under the influence of a wide range of physiologic conditions. For these two reasons the T-wave changes will not be discussed further.

**QRS Complexes**

Changes in the peak-to-peak amplitude of the QRS complexes sensitively summarize the changes in the R and S waves individually and support the discussion below. Large increases were recorded after left mastectomy over the entire precordium, most significantly in leads V_{3R}, V_{1}, V_{2}, V_{3}, V_{4A}, V_{4B}, and V_{4C}. In V_{4B} and V_{4C} increases in the individual complexes were as great as 3.5 millivolts. Changes of moderate or intermediate significance were recorded in V_{4D}, V_{5}, and V_{6} beneath which little, if any, tissue was excised. After right mastectomy, highly significant increases in the amplitude of the QRS complexes were noted in V_{3R} and V_{1}, and less significant increases in V_{5} and V_{6}. From a comparison of the changes in the two operative groups, two observations are pertinent. After left mastectomy, increases in the amplitude of complexes in leads V_{2} through V_{4}, and to a lesser significance in V_{5}, were strikingly greater than after right mastectomy. Secondly, after both left and right mastectomy, there were highly significant increases in the amplitude of complexes in V_{3R} and V_{1}, which were statistically equivalent after the two operations.

The significance of these findings is directly pertinent to the interpretation of the electrocardiogram after mastectomy. If precordial voltage criteria are utilized for the diagnosis of left ventricular hypertrophy, these data suggest that the occurrence of false-positive diagnoses after left mastectomy may approach 50 per cent. The study indicates that after mastectomy, standard criteria for electrocardiographic diagnosis may not apply. Perhaps after any alteration in the composition or configuration of the thorax, standard criteria should be employed with caution.

Perhaps of more fundamental importance, however, are the implications of these data regarding the effect of body build and especially chest wall configuration on the electrocardiogram. In addition to the effect of abnormalities of the chest on the electrocardiogram, the effect of normal variations in chest characteristics would appear to be important. As an example of this, Simonson et al. have noted that the precordial complexes, especially in leads V_{2} through V_{5}, are smaller in women than in men. Walker and Rose have reported that as youngsters pass through puberty, voltage in the precordial complexes in females diminishes markedly when compared to that in males. Finally, Selzer et al. have pointed out that erroneous electrocardiographic diagnoses, especially of ventricular hypertrophy on the basis of precordial voltage, are frequent in emaciated individuals. All of these studies, while providing valuable clues to the sources of electrocardiographic errors, are unable to isolate the variables concerned. For example, not only the dimensions but also the composition of the precordial tissues differ between

*Circulation, Volume XXXII, November 1965*
postpubertal males and females. The myocardial mass and perhaps the voltage that it generates may also differ. Similarly, with emaciation changes occur both in the tissues of the chest wall and in the myocardium itself.8

The present study has been able to isolate a single variable, namely, the excision of soft tissue from the anterior chest wall. There has been rigid control of precordial lead placement and of sensitivity standardization; and the groups of patients undergoing left or right mastectomy were comparable with respect to underlying cardiopulmonary conditions. There was no other consistent change in the patients' physical condition, and only in one instance was any change in the myocardium itself suspected.* The statistical analysis of the data corrects for the influence of this and of other, perhaps unrecognized, changes postoperatively in the patients' physiologic condition.

Although the data do not specifically indicate the reasons for the observed increase in voltage of the precordial complexes, they do justify some comments. One plausible reason for the increase, frequently suggested to explain variations in precordial voltage, is the proximity of the recording electrode to the myocardium. Although decreased distance satisfactorily can account for the increases observed in the left precordial leads after left mastectomy, it does not explain the increases seen in complexes over the right precordium after this operation. That there is a definite increase in voltage on the contralateral side, this study demonstrates conclusively. One might argue, however, that the study has failed to appreciate a decrease in distance between these right precordial leads and the myocardium—perhaps secondary to muscular atrophy or postoperative weight loss. This possible objection is probably not valid for the following three reasons: (1) Radical mastectomy is well tolerated by patients, requiring little bed rest and usually causing no general-ized weight loss or debilitation. (2) In the study group, the observed weight loss was 3 to 4 per cent of body weight, roughly the size of the surgical specimen. In some patients there was weight gain. (3) Muscular atrophy was unlikely to occur because the patients were placed on arm-motion exercises within a week of operation. Furthermore, if atrophy were to be expected, it would occur on the operated side, which the patient might favor because of pain; and there might be relatively more motion and muscular activity of the contralateral side. For these reasons, therefore, it is highly unlikely that there was a decreased distance between the myocardium and the right precordial leads after left mastectomy. Though decrease in distance alone does not adequately explain the increase observed in the precordial voltage postoperatively, investigations now in progress indicate that it is a highly significant factor.

A second possible explanation for the postoperative increase in precordial amplitude is the altered ability of the tissue to transmit the electrocardiographic impulse. This altered ability of the tissue might consist of diminished resistivity or of diminished capacitative impedance. Concerning tissue resistivity, Schwan and Kay9 and Nelson10 reported low values, varying in the range of 500 to 1,000 ohm centimeters. The present authors found, as would be expected, no difference in the recorded electrocardiogram potential when resistances up to 200,000 ohms were interposed between the skin and the electrocardiograph. The slight reduction of tissue resistance resulting from excision of tissue could not account for the increases of voltage reported above.

This investigation does not indicate the possible role of diminished capacitative impedance in the increased precordial voltage after surgery. In this connection it is worth noting that the T waves increased proportionately more than the QRS complexes in all leads; but the changes in the T waves were thought to reflect not only the influence of surgery but also the intrinsic variability of these complexes under a wide variety of

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*The patient suspected of an apical myocardial infarction.
physiologic circumstances. Nevertheless, neither diminished tissue resistivity nor decreased tissue capacitative impedance could explain the increase in the right precordial amplitudes after left mastectomy.

A third possible explanation for the increase in precordial voltage is an altered relationship between the heart and the thoracic volume conductor. From the work of Pipberger, Schmitt, Frank, and Frank and Kay, among others, it is justifiable to consider the electrical impulse from the myocardium equivalent to that which might be produced by a dipole properly positioned within the thorax. In a meticulously performed series of studies these various workers have shown the significant influence on surface potentials of the dipole position within the thorax models. This influence is greatest on the surface nearest the dipole, which according to Schmitt occurs at V₃. In the present study, following left mastectomy, the greatest increases in voltage were recorded in leads V₃ and V₄. In related work, while producing no change in the dimensions of his model, Nelson has been able to convert a volume conductor of infinite extent to one with finite boundaries. He has found in the latter situation, with the dipole position unchanged, a marked distortion of the equipotential lines within the model. Despite the fact that the greatest voltage increment occurred at the boundary nearest the dipole, all the surface potentials increased in the limited volume conductor.

These findings can be related to the present study by considering the thorax as a volume conductor of infinite, or relatively unlimited extent. Mastectomy may alter the characteristics of the volume conductor without affecting the position of the heart, or dipole-equivalent, within the thorax.

Summary

The electrocardiograms before and after operation have been compared in 20 patients undergoing left mastectomy and in 19 patients undergoing right mastectomy.

There are highly significant increases in the amplitude of the QRS complexes recorded over the precordium after left mastectomy, except in lead V₆.

There are highly significant increases in the amplitude of the QRS complexes of leads V₃R and V₄ after both left and right mastectomy.

These data are not completely explained by a diminution of distance between the recording electrode and the myocardium or of electrical resistance following the excision of soft tissue.

The possible relationship is suggested between the precordial voltage changes and the altered thoracic volume conductor.

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References

Galen, 131-201 A.D., Founder of Experimental Physiology

Galen, "the Serene," was an intellectual giant. He inherited, discussed, and synthesized all philosophical, scientific, historical, philological, and medical knowledge accumulated during eight centuries of Greek and Hellenistic civilization. His greatest achievement in cardiovascular physiology was his concept of the unidirectional movement of blood and air through the lungs, which endured until Harvey.

He was educated in the foremost philosophical systems of his time, Academic, Lycean, Epicurean, and Stoic, and also in mathematics and logic. Galen's education was strongly influenced by his father, a learned man of the highest personal qualities, and by his city of birth, Pergamon, in which the famous temple to Asklepios was located. At seventeen years of age, apparently prompted by his father's vivid dreams, he added the study of medicine to his study of philosophy. After several years of medical study, which included the dissection of animals, he left Pergamon to travel throughout Greece and Asia Minor, exploring the medical customs of each of the areas that he visited. Included in his itinerary was Alexandria, where he encountered the scientific tradition established by Herophilos and Erasistratos, and where he found human dissection used in anatomical teaching. After twelve years of study abroad, he returned to Pergamon to become physician to the gladiators, a position which provided ample experience in traumatic surgery and medicine. Four years later he moved to Rome, then at the zenith of its power. There he soon achieved great renown for his skills in medical practice and in dissection, and became the physician and friend of the Emperor, Marcus Aurelius. Although his medical colleagues were well aware of his scientific talents and accomplishments, they found his arrogance and ostentation insufferable. His scientific legacy consisted of many papers and books, but no disciples.—André Cournaud, M.D. Circulation of the Blood. Edited by Alfred P. Fishman, M.D., and Dickinson W. Richards, M.D. New York, Oxford University Press, 1964, pp. 11 and 12.

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