BRIEF COMMUNICATIONS

Magnetic Measurement of S-T and T-Q Segment Shifts in Humans

Part II: Exercise-Induced S-T Segment Depression

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SUMMARY. The direct-current magnetocardiogram, which shows the T-Q (baseline) shift, is used to clarify the cause of S-T depression induced by stress testing in the human heart. Measurements are made of the amount of baseline shift associated with the S-T depresion. Results are presented of a well-documented patient, with typical coronary artery disease, undergoing a two-step exercise test. Before exercise, there was no S-T or baseline shift. During exercise, the S-T segment became depressed and the baseline segment was simultaneously elevated, at about 70% of the S-T amplitude. After termination of exercise, the baseline elevation disappeared somewhat more rapidly than the S-T depression. These results were consistent in repeated tests of this patient. Because the baseline shift is a reflection of an injury current, these results confirm the belief that exercise-induced S-T depression is mostly due to an injury current which is interrupted during the S-T interval. The baseline shift seen here is the first non-invasive measurement of an injury current in the human heart, and its presence and time-course generally agree with measurements in the animal heart. This work also confirms that the direct-current magnetocardiogram, although not practical for clinical purposes, is useful as a research tool. (*Circ Res 53: 274-279, 1983*)

IN Part I of this work, it was indicated that there are two mechanisms by which the S-T segment shift on the electrocardiogram (ECG) can be produced. In the first, it is produced by a current flowing only during the S-T interval, and is called a "true" S-T shift. In the second mechanism, it is produced by a steady injury current which flows during the entire cardiac cycle, except that it is interrupted during the S-T interval; in this case it is called an "apparent" S-T shift. In principle, it would be possible to distinguish between the two shifts because the apparent S-T shift would be accompanied by a T-Q segment (baseline) shift, but on the ECG this T-Q shift cannot be seen because the baseline is filtered out to eliminate skin interference. Therefore, the ECG cannot distinguish between the two S-T shifts. However, the direct-current magnetocardiogram (dcMCG), which is insensitive to skin interference, does measure the T-Q shift; hence, it can distinguish between the two types. The dcMCG was used in Part I to verify that the S-T shift in early repolarization and left bundle branch block was a true S-T shift. It is used here again to clarify the cause of the S-T depression induced by exercise.

Exercise-induced S-T depression is believed to result from a subendocardial ischemia. Because animal experiments have shown that both true and apparent S-T shifts can be induced by ischemia, it is reasonable to believe that both types of S-T shifts would be present during stress testing in patients with coronary artery disease. However, this has never been experimentally verified. Our purpose here is to determine if both indeed are present, and to measure the time course of each. This information should aid in understanding the electrophysiology underlying exercise-induced ischemia.

Because there are stringent requirements for a dcMCG stress-test measurement, only one patient was studied here. However, this was a well-documented patient with typical coronary artery disease; therefore, if caution is used, the results can to some extent be generalized.

Methods

The Magnetic Technique

The details of the dcMCG and its use were described in Part I. Except in one aspect, they are similar here. In brief review, the dcMCG is measured in a magnetically shielded room with a two-channel detector, where the detecting coil in each channel is of the 2-D type (inset of Fig. 1). One channel measures the magnetic field gradient called $\Delta B_z/\Delta y$, due to current sources oriented in the x-direction. The other measures $\Delta B_z/\Delta x$, due to sources oriented in the y-direction; x, y, and z are standard vectorcardiographic axes, Δx and Δy are the mean separations between D's, and B_z is the z-component of the magnetic

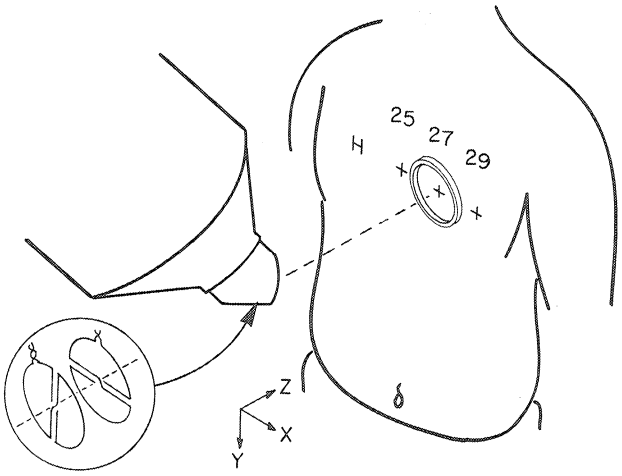

FIGURE 1. *Arrangement for measuring the dcMCG. The detector, described in Part I, is the 2-D SQUID system located in the tail of the dewar containing liquid helium. The 2-D detecting coils are shown in the inset; in practice they almost coincide, but are here separated along the z-axis for illustration only. The left coil measures* $\Delta B_z/\Delta x$*; the right coil measures* $\Delta B_z/\Delta y$*. Preliminary measurements were made equally at points H25, H27, H29, separated horizontally by 5 cm; H25 is on the midline, 10 cm above the xiphoid. For a measurement, the patient steps forward from his present out-of-range position, places one of the three points at the detector, then steps back again. Final measurements were made mostly at H27, where a plastic ring, which accurately locates the detector, was attached to the chest.*

field vector. The dcMCG cannot see z-oriented sources (normal to the chest), because they produce essentially zero external field. The aspect of the dcMCG which is different here, than in Part I, is the number of locations over the chest which are measured.

This difference involves extraneous dc fields arising from noncardiac sources in the torso, such as from the GI tract; these extraneous fields produce false T-Q shifts. It was noted that false fields could be handled in either of two ways. First, the cardiac S-T shift could be modulated (turned on-and-off as with coronary artery occlusion in a dog) so that any associated T-Q shift would also be modulated; the cardiac T-Q shift would therefore be separated from the unmodulated, steady false shift. In this case, the dcMCG need only be measured at a single location at the chest, provided that the S-T shift is clearly seen there. When the S-T shift could not be modulated, the second way is used; a map of T-Q shifts is plotted over the chest, and the cardiac T-Q shift is separated from the false shift because it is spatially centered over the heart. In Part I, no modulation was possible, therefore the map was used. In the case studied here, the S-T shift is modulated by exercise; therefore, a mapping is not necessary and the dcMCG is measured mostly at one location.

To ensure that any false T-Q level was indeed steady, so that the heart modulation would be effective, before every stress test the dcMCG was measured every 2 minutes for a period of 30 minutes. This protocol provided a T-Q control level (the "off") against which T-Q shift due to exercise (the "on") could be compared. The following criterion of steadiness was used. If the patient's T-Q fluctuations were <1 pT/cm during this period, the stress test was allowed to take place; if not, it was abandoned. Prior to a stress test, most of the same precautions described in Part I were taken to minimize extraneous fields; these included performing an overnight fast to decrease the GI electrical activity and using a magnetic eraser to demagnetize ferromagnetic particles in the lungs and GI tract.

Patient Selection, Stress Test Protocol

The records of several hundred patients were screened for the following factors: an exercise-induced S-T depression ≥2 mm in the limb leads, a history of left main coronary artery disease as disclosed by coronary arteriography, and a generally stable condition with no history of unstable angina or congestive heart failure. (The limb-lead requirement reflects the fact, as explained in Part I, that the MCG is sensitive only to sources oriented parallel to the frontal plane.) Five volunteer male patients were chosen as candidates for this study with their informal consent. Each was then measured for steadiness of extraneous fields, after an overnight fast, by monitoring the T-Q level for a 30-minute period. This was in duplication of the pre-exercise control period. It was found that there was considerable variation in steadiness, and in four patients the control-period criterion was not met, hence they were rejected from the study. In only one patient was the extraneous field found to be very stable. He was 63 years of age, and coronary arteriography had revealed three-vessel coronary artery disease. His prior stress tests had shown 2 mm S-T depression in aVF, but his resting ECG was normal. In every respect, he was a patient with typical coronary artery disease. It is this one patient who was extensively measured by us in a sequence of stress tests, and whose data constitute this study.

Certainly the data of only one patient is of limited value. However, considering the large screening effort required to produce one qualified patient, and that this is a first measurement of its type, the selection of further subjects at this time could not be justified. The fact that this patient's disease is typical and that he is well-documented warrants the presentation of these data.

The patient underwent a total of five exercise stress tests for this study; the first two were preliminary, while, in the last three tests, conclusive dcMCG data were accumulated. During the testing period he received no digitalis or other agents which could influence his cardiac electrophysiology. Before each test, blood and urine samples were analyzed, the 12-lead ECG was recorded, and he was examined by two physicians. To prepare for a test, he changed into nonmagnetic clothes (no zippers, etc.) During the test, standard safety procedures were always followed; a cardiologist, a trained nurse, and a respiratory therapist were present, along with a crash cart with drugs and defibrillator. During and after the exercise, the patient's blood pressure was repeatedly measured using a nonmagnetic sphygmomanometer. The test was terminated when there was a significant drop in blood pressure, or the patient began to experience mild discomfort.

The following details apply to the three data-recording tests. The type of test performed was the two-step test; this was used instead of the treadmill or bicycle ergometer because the moving steel components in these devices would produce magnetic disturbances. Each of the two (wooden) steps was 8 inches high, and the patient walked up and backed down these steps during two stages of exercise, paced by a metronome. During the first stage, the up-down time was 5 seconds, whereas, during the second, it was 4 seconds. Exercise was briefly interrupted every 2 minutes or so to perform the dcMCG measurement.

The dcMCG measuring arrangement is shown in Figure 1. Preliminary measurements were made at three locations in order to choose one best location for final data. Measurements at one location was desirable because the S-T and T-Q shifts could change rapidly in time, and their time course could best be followed by repeated measurements at one location. The pre-exercise T-Q shift was smallest and the S-T shift due to exercise was largest at H27; therefore, this was the location chosen. However, at this location, the S-T and T-Q shifts were a sensitive function of detector position and angular orientation. To minimize errors due to this sensitivity, a positioning ring was taped to the chest, as illustrated. Then, as the patient approached the detector for each measurement (see Part I), he was able to seat the detector into the ring at a fixed position; the ring also restricted the angular orientation. Most data shown here were accumulated using the ring at that one location.

The ECG leads aVF, VI, and V5 were continuously and simultaneously recorded using nonmagnetic foam-padded silver chloride electrodes, connected through a low noise cable. The two dcMCG signals and these ECG signals were monitored on a strip chart recorder, and recorded on magnetic tape for later study. The ECG bandwidth was 0.05–50 Hz, whereas that of the dcMCG was dc-50 Hz.

Results

The S-T and T-Q data were consistent in all five stress tests. Because the most extensive recording was made in test 5, the data from this test are presented here. Some raw traces are shown in Figure 2. Of the two magnetic channels, it is seen in Figure 2A that the largest changes due to exercise are in the $\Delta B_z/\Delta y$ column. There is an increasing S-T depression as exercise progresses, as well as a T-Q shift which accompanies the S-T depression. For example, after 4 minutes of exercise, the S-T depression is −2.0 pT/cm and the T-Q elevation is +1.5 pT/cm. In the V5 column, the S-T depression progresses similarly to that in $\Delta B_z/\Delta y$ (in those ECG traces not shown here, V1 contains no S-T shift, whereas aVF shows a somewhat lesser S-T depression than does V5). At 2 minutes post-exercise, the T-Q level in $\Delta B_z/\Delta y$ has essentially returned to zero, but the S-T depressions in that channel and in V5 are still apparent. At 8 minutes post-exercise, both have returned to the pre-exercise level. The similarity between the S-T shifts (and other features) in $\Delta B_z/\Delta y$ and V5 is to be expected, in that both signals are mainly sensitive to cardiac sources oriented along the x-axis.

The $-\Delta B_z/\Delta x$ column shows much smaller S-T and T-Q shifts than does the $\Delta B_z/\Delta y$ column. Because signals in the two columns are due to sources oriented perpendicularly to each other, there is no reason why their shifts should be similar; the absence of significant shifts in $-\Delta B_z/\Delta x$ indicates only that there is no significant source oriented in the y (vertical) direction. The absence of T-Q shift in this channel also allows it to serve as a control. It indicates that it was unlikely that the T-Q elevation in $\Delta B_z/\Delta y$ was induced by exercise in an organ other than the heart (an exercise-induced extraneous field), since such fields would have a significant $\Delta B_z/\Delta x$ component (see Part I); hence, this T-Q elevation is indeed related to the S-T depression. The small changes in QRS amplitude and waveform seen down both dcMCG columns are mostly a modulation due to respiration; this modulation is clearly seen in Figure 2B.

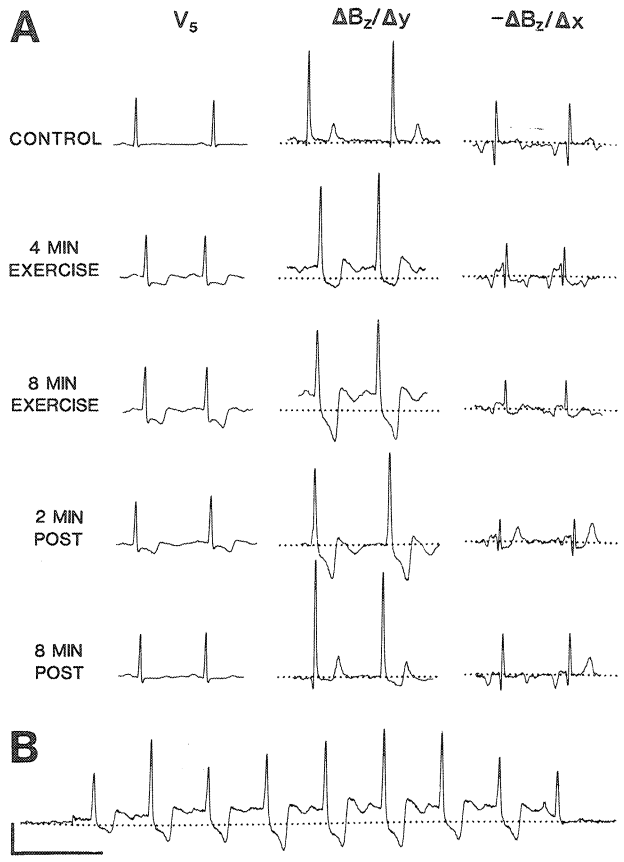

FIGURE 2. *Part A: sections of raw traces recorded before, during, and after stress test 5. ECG traces are in the left column, and dcMCG traces from location H27 are in the other two. The minus sign on $\Delta B_z/\Delta x$ produces upright QRS. The dotted lines are the magnetic zero level. Part B: a complete measurement of $\Delta B_z/\Delta y$ at 5.5 minutes of exercise, in test 5, showing how the magnetic zero was chosen. At the flat, leftmost part of the trace the patient is out-of-range. He then brings H27 up to the detector and remains there for about one respiration cycle, then steps back again to yield the flat, rightmost part. The two flats define the magnetic zero level. The vertical calibration bar is both 1 mV for V5 and 4 pT/cm for the dcMCG; the horizontal bar is 1 second.*

The time-course of the shifts in test 5 are plotted in Figure 3. During the control period, the T-Q fluctuations, which are due to time-changes in the extraneous fields, are seen to be within the acceptance criterion of <1 pT/cm in both magnetic channels. The average T-Q shift and the standard deviation (n = 15) were calculated and found to be −0.1 ± 0.5 pT/cm in $\Delta B_z/\Delta y$, and −0.1 ± 0.4 pT/cm in $\Delta B_z/\Delta x$; the standard deviations indicate the T-Q fluctuations to be expected after exercise begins. During the exercise period, the S-T shift in $\Delta B_z/\Delta y$ increases to a level of 3.6 pT/cm, whereas the T-Q

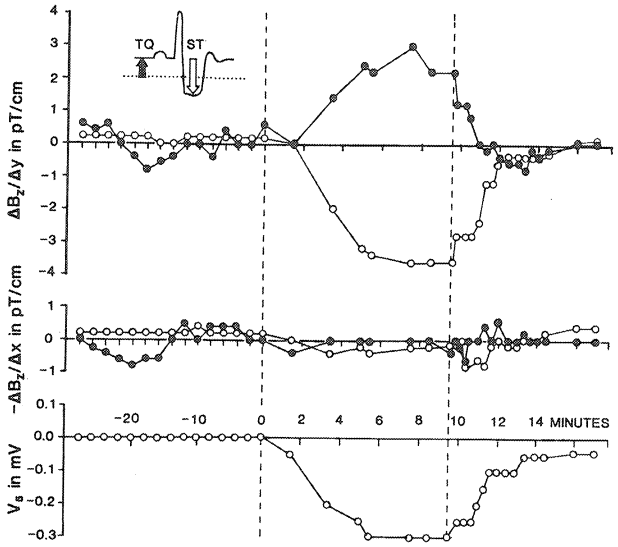

FIGURE 3. *Time course of the S-T shifts (black dots) and T-Q shifts (open dots) in test 5. The upper left diagram indicates how the T-Q shift (black arrow) and S-T shift (white arrow) were defined on the raw traces. The T-Q shift is the difference between the T-Q level and the magnetic zero reference (dotted line, subject out of range), whereas the S-T shift is the difference between the S-T level and the T-Q level. The two dcMCG plots are from location H27. The two vertical dashed lines indicate the beginning and end of exercise. Before exercise, where the time scale is compressed, the control measurements are made every 2 minutes.*

shift also increases but to a lesser extent, then decreases near the end of the exercise period. However, the largest T-Q excursion (3.0 pT/cm at 7 minutes) appears to be an upward extraneous-field fluctuation, and it is likely that the change of the T-Q curve is the same as that of the S-T curve, leveling off at about 2.5 pT/cm. In this interpretation, there is no true downward turn near the end of exercise. The difference of 1.1 pT/cm between S-T and T-Q levels is noted to be more than twice the standard deviation of fluctuations. Thus, it appears that the time course of the T-Q shift is similar to that of the S-T shift during this period, but its level is about 70% of the S-T level. During the exercise period, about 70% of the S-T shift in $\Delta B_z/\Delta y$ is an apparent shift, whereas about 30% is a true shift. No significant S-T or T-Q shifts take place in $-\Delta B_z/\Delta y$ during the exercise period, confirming the observation in Figure 2.

In the post-exercise period, the S-T segment recovery is not similar in $\Delta B_z/\Delta y$ and V5; the S-T shift vanishes more slowly in V5. Whereas these two shifts should behave similarly in time because they both respond to x-sources, some differences are expected because they are due to different angles-of-view. The 2-D coil samples a smaller area of the heart than does the V5 electrode, and the area viewed in $\Delta B_z/\Delta y$ appears to recover more rapidly than that viewed in V5. Also, in this period, the T-Q shift appears to vanish more rapidly than does the S-T shift in $\Delta B_z/\Delta y$. Although it is possible that the T-Q time course in test 5 is truly identical to that of S-T when account is taken of the extraneous-field fluctuations, the data in the other tests indicate that the T-Q level does vanish more rapidly; hence, we accept this interpretation. Therefore, it appears that the S-T apparent-true ratio changes from 70/30 to 0/100; that is, the S-T shift becomes a true shift as it vanishes.

Discussion

The presence of a T-Q elevation associated with S-T depression in the dcMCG during exercise is the first non-invasive measurement of injury current in the human heart, and indicates the presence of the apparent type of S-T shift (noted by us in a preliminary analysis (Savard et al., 1981)). In addition, some true S-T shift is present. During exercise, the apparent/true ratio is constant at about 70:30, but after cessation of exercise the ratio appears to change in favor of the true type of S-T shift. Of course these results are based on only one patient. However, because his data are consistent in repeated tests, and his clinical and electrocardiographic manifestations of coronary artery disease are typical, we cautiously suggest that stress testing of similar patients will yield similar results. In the following, we first compare these results to previous results, next we discuss aspects of the apparent S-T shift, then we comment on the true S-T shift.

Because the S-T shift seen here is related to ischemia induced by stress testing, we compare these results to experimental results dealing with S-T shifts from induced ischemia. Our results are in general agreement with the dcMCG measurement of the ischemic heart in intact dogs (Cohen and Kaufman, 1975), and with numerous dc potential measurements of exposed or isolated ischemic animal hearts. In the dcMCG measurement, when a dog's coronary artery was occluded, an S-T shift developed which at first was almost completely of the apparent type. After 15 minutes or so, a true S-T component became apparent. The dcMCG human and dog measurements agree, insofar as ischemia produced mostly an apparent S-T shift at first, and, after a time, some true S-T shift as well.

In dc potential measurements made directly on the heart, the presence of both types of shifts in response to coronary artery occlusion in dogs was reported by Samson and Scher (1960), Katcher et al. (1960), Printzmetal et al. (1961), and in the isolated pig heart by Kléber et al. (1978), Janse et al. (1980), and Moréna et al. (1980). There was evidence that the apparent type resulted from a loss of membrane resting potential, whereas the true type was caused by a loss of amplitude and change in shape of the action potential (Samson and Scher, 1960; Kléber et al., 1978). Most had shown a time course similar to that seen here. For example, in the isolated pig heart, the apparent S-T shift appeared at about 1.5 minutes after coronary artery occlusion, followed some 3 minutes later by the true type (Kléber et al., 1978;

Janse et al., 1980; Moréna et al., 1980). Similar results were reported by Katcher et al., (1960). However, several authors have reported the appearance of the true component a few minutes before the apparent one (Samson and Scher, 1960), or the appearance of only an apparent component (Vincent et al., 1977). We point out that one problem in these animal studies is that the measurements were made on the exposed heart. The interruption of the volume conductor could alter source currents hence relative S-T/T-Q potentials on the heart surface, in comparison to those on the surface of the intact torso; ultimately, it is the S-T shift measured on the surface of the intact human torso which needs clarification. In contrast, the dcMCG measurements were made over the intact torso, and therefore directly relate to surface ECG measurements. It follows that the time-course seen by the dcMCG in both dog and human, where the apparent S-T shift appears first, is a more reliable result than that determined from electrogram measurements.

Our results also confirm data in a brief report of electrogram measurements of an isolated human heart, due to ischemia induced by occlusion of a coronary artery (Janse and Kléber, 1981). The occlusion caused an S-T shift which was accompanied by a T-Q shift. At 3 minutes after occlusion, the T-Q shift was equal and opposite, whereas, at 4.5 minutes, an extra S-T shift was seen that seemed to be the true type; these appeared to remain stable for the remainder of the 45-minute occlusion.

The presence of the injury current is of clinical interest because it can cause harmful arrhythmias; this would take place at the border of an ischemic zone (Janse et al., 1980; Kléber et al., 1978). In particular, the injury current could reexcite cells (Hoffman, 1966; Han, 1969), enhance the automaticity of the Purkinje cells (Moe and Mendez, 1973; Jalife and Moe, 1976), or enhance early repolarization which would facilitate re-entry (Cranefield, 1975; Katzung et al., 1975). Although no arrhythmia was seen in the stress testing of our patient, it is known that exercise does produce arrhythmia in some patients, and this may be the cause. Stated otherwise, when an injury current (apparent S-T shift) is induced in a patient by exercise, there may be a greater risk of arrhythmia than when the S-T shift is largely of the true type.

Concerning the 30% of true S-T shift during exercise, its origin may be more complicated than a current flowing only during the S-T interval. This is an ideal origin, stated in Part I, where the current is due to a difference in action potentials between depolarized regions of the heart; the resting potentials are not directly involved. Further, the apparent shift is ideally due to an injury current which is *completely* interrupted so that zero current flows during the S-T interval. The actual conditions may be more complicated (Rush et al., 1982), in that the cells around the injured area may be in a polarized but non-excitable state due to a diminished resting potential; therefore, some injury current may flow during the S-T interval. As an example, we assume a resting potential of −50 mV (instead of the normal −90 mV). During the S-T interval, there is now a current between the injured area (at −50 mV) and the slightly depolarized potential in the normal region (say at +10 mV). Further, during the T-Q interval, the injury current is generated by the difference in resting potential between the normal and injured regions, which is now 90–50 mV (instead of the ideal 90–0 mV). As a result, the T-Q shift is no longer ideally equal and opposite to the S-T shift; the T-Q/S-T shift ratio is now −90-(−50)/+10-(−50) = −40/60, or about 70%, which happens to be what we see here. The 30% of true S-T shift that we see, then, could be an "ideal true" shift, or the non-ideal shift of our example, or a combination of the two. There are not enough data here to separate them.

The fact that the dcMCG can measure the T-Q shift suggests that it could be a most useful clinical tool. Unfortunately, our experience here confirms that in Part I; only in selected persons are the extraneous fields small enough to allow dcMCG measurement. Until a method is found to reduce these fields so that most patients would be measureable, the dcMCG will remain a research tool. However, in this regard it can be a unique tool, as shown by the work presented here. Further cases where it can be used, as mentioned in Part I, are the S-T shifts seen in ventricular aneurism, in left ventricular hypertrophy, and as a result of using digitalis.

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Part I of this work appears as the preceding paper in this issue.

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