

Ischemic Polarcardiographic Changes Induced By Exercise

A New Criterion

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SUMMARY

The polarcardiographic responses to exercise in normal men, young and middle-aged, have been compared with those of men who show ischemic responses on the electrocardiogram. Changes in the ST-vectors are the most significant. These changes have been reduced to a single numerical quantity based on the spatial magnitude of the vector at the end of the QRS complex and the spatial direction of a vector occurring at a clearly specified time during the period of the ST-segment. The assets of polarcardiography are that it enables study of *direction and magnitude* of ST-vectors in a time sequence. Thus it provides information of clinical importance which cannot be obtained by either electro- or vectorcardiography.

Additional Indexing Words:

Exercise ECG

ST-segment depression

Myocardial ischemia

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VARIOUS COORDINATE SYSTEMS may be employed to define the heart vector.¹ Rectangular coordinates have been used most frequently because they are readily available as the x, y, and z signals obtained from a vectorcardiographic lead system. However, the use of polarcoordinates goes back to Einthoven's description of an electrical axis. Spherical polar coordinates are conceptually more appropriate in problems relating to the magnitude and direction of the heart vector, just as are height, latitude, and longitude in relation to the globe. Because polarcoordinates of the heart vector can be readily derived from rectangular coordinates by an

analog computer, *viz.*, the polarcardiograph, there is no longer any need for cardiologists to restrict themselves to a coordinate system which, in this application, is cumbersome.

Whereas the x, y, and z signals, when fed to an oscillograph, yield the corresponding x, y, and z orthogonal electrocardiograms (ECGs), so the outputs from the polarcardiograph give polarcardiograms (PCGs),* which are tracings against a time scale of the spatial magnitude and direction of the heart vectors as they change throughout the cardiac cycle.

The resting PCG has been described in detail and the value of this type of display has been demonstrated, particularly in the diagnosis of myocardial infarction.¹⁻³ The PCG responses to exercise in normal subjects have also been reported.^{4, 5}

The value of the ECG as an indicator of myocardial ischemia induced by exertion has been repeatedly confirmed. The present paper describes the PCG changes induced by exercise in subjects showing an ischemic ECG response and raises the possibility of the PCG providing a superior indicator of myocardial ischemia.

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* Where confusion with the abbreviation for a phonocardiogram might exist, the abbreviations PoCG and PhCG have been suggested.¹ When polarcardiograms became routine at the Vancouver General Hospital they were immediately christened "polars."

Method and Materials

Subjects were exercised in stages, either on a treadmill or on a bicycle ergometer. ECGs were derived from a bipolar chest lead between the conventional V_5 position and a point just below the right scapula (lead CB_5).

The x, y, and z signals were derived from the Frank lead system. In subjects being studied in the upright position, the chest electrodes were placed at the level of the sternal ends of the fifth intercostal spaces; a protractor was used to locate the C electrode.

The equipment employed for polarcardiography consists of a bedside recorder, a playback unit, a polarcardiograph computer, and an oscillograph. It is manufactured by Totemite, Inc., P.O. Box 875, Bow, Washington 98232, and costs approximately \$12,000. It can be operated by an ECG technician after very little instruction and can easily be incorporated into any Heart Station.⁶ The bedside recorder places the x, y, and z signals on magnetic tape together with the patient's name and any other information. The size and ease of operation of the bedside recorder are about the same as that of a standard electrocardiograph. The tape is replayed via the playback unit to send the recorded x, y, and z signals to the polarcardiograph, which is a small, special-purpose, analog computer. The polarcardiograph automatically transforms the rectangular coordinates, represented by the x, y, and z signals, into the corresponding polar coordinates, represented by magnitude, latitude, and longitude signals, which are

then led to the oscillograph to give PCGs. The procedure takes only a few minutes. In addition to PCGs, the x, y, and z signals are used to simulate the 12-lead ECG^{1, 6, 7, 8} (a simulator is available from Totemite, Inc.); they may also be fed to a conventional vectorcardiograph.

The subjects for analysis were drawn from a series of over 150 studied during exercise by polarcardiography at the University of Washington and 22 subjects, similarly studied, at the University of Rotterdam. Twenty-nine young men, mean age 22.3 (SD 3.4) and 27 middle-aged men, mean age 48.7 (SD 5.0), from the University of Washington, all apparently normal with respect to history, physical examination, blood pressure, and exercise response, as determined by criteria previously reported were selected for study.⁹ In addition, this analysis includes studies on 14 men showing significant ST-segment depression after exercise; the resting ECGs were normal in 11, three showed T wave flattening or inversion. PCG changes were observed immediately after exercise, except in one case where tracings were obtained one minute after exercise. All but two subjects were exercised on the treadmill; those two, as well as the subjects in Rotterdam, were exercised on the bicycle ergometer. From the 22 Rotterdam subjects, 12 were selected as having typical ischemic ECG responses to exertion. Thus, a total of 26 subjects, mean age 53.7 (SD 9.8), with ECG recordings that reflected ischemia are included in this study.

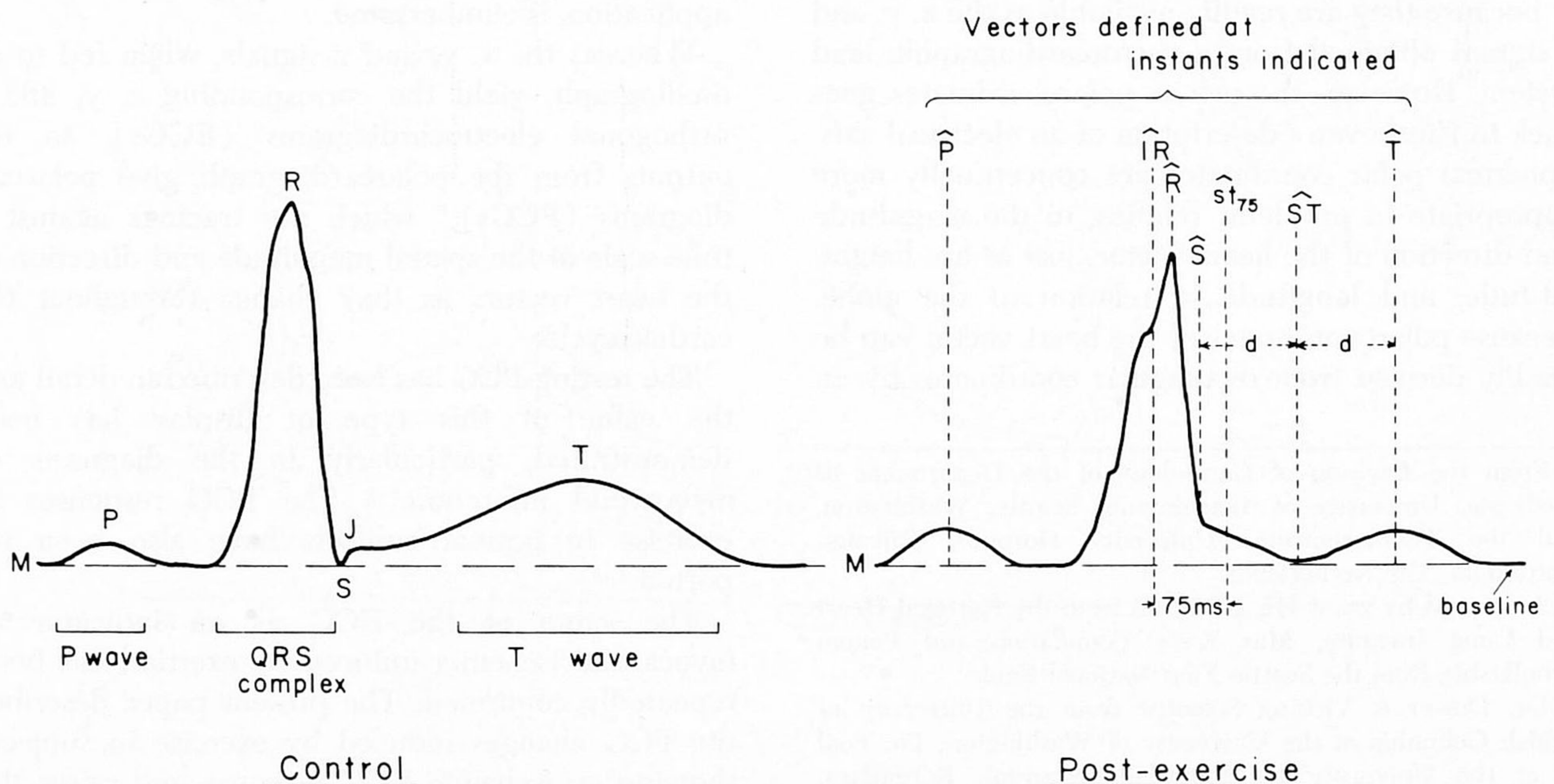


Figure 1

Drawings of the spatial magnitude tracings typical of a postexercise ischemic response. Unlike the ECG, which it otherwise resembles, the M tracing does not deflect below the baseline—since magnitudes cannot be less than zero. $M = \sqrt{x^2 + y^2 + z^2}$. Apparent negative values represent errors in baseline reference. These are eliminated by baseline clamping prior to each QRS complex. Ischemic features are elevation of the point S and downsloping of the ST-segment, M_{st} . \hat{P} , \hat{R} , and \hat{T} are the maximum P, QRS, and T vectors, respectively. \hat{IR} occurs at a time which divides the QRS complex of the M tracing into two equal areas. \hat{S} marks the end of the QRS complex. \hat{ST} occurs midway between \hat{R} and \hat{T} , and \hat{st}_{75} occurs 75 msec after \hat{IR} .

Definitions

- M tracing*: the tracing of the spatial magnitudes of the heart vectors (fig. 1).
- M_{QRS} : the QRS complex in the M tracing.*
- M_{ST} : the ST-segment in the M tracing.
- MR : the spatial magnitude of the maximum QRS vector, \hat{R} .
- MT : the spatial magnitude of the maximum T vector, \hat{T} .
- MS : the spatial magnitude of \hat{S} , the vector at the end of M_{QRS} (MS is frequently zero).
- \hat{S} : the vector occurring at the midpoint in time between \hat{S} and \hat{T} .
- \hat{IR} : the vector occurring at the instant when the area under M_{QRS} is half its final value.

- \hat{st}_{75} : the vector occurring 75 msec after \hat{IR} .
- α -longitude tracing: the tracing of the directions in the frontal plane of the heart vectors (fig. 2).
- PA-latitude tracing*: the tracing of the postero-anterior directions of the heart vectors (fig. 2).
- θ : the angle subtended in space between \hat{st}_{75} and a defined reference direction, e.g., 50A, +30 at rest, or 50A, +20 after exercise.
- fm tracing*: the tracing of the magnitudes of the heart vectors in the frontal plane (fig. 2).

Results

As in the ECG, the ischemic response to exercise in the PCG appeared to be greatest for ST events. Although obvious QRS changes in the M tracings were seen in several subjects, these did not correlate with ischemia, since they were common in the normal young men (fig. 2).

Ischemic Vectors

Typical ischemic changes in the M tracing are shown schematically in figure 1. There is an increase in MS , the spatial magnitude at the

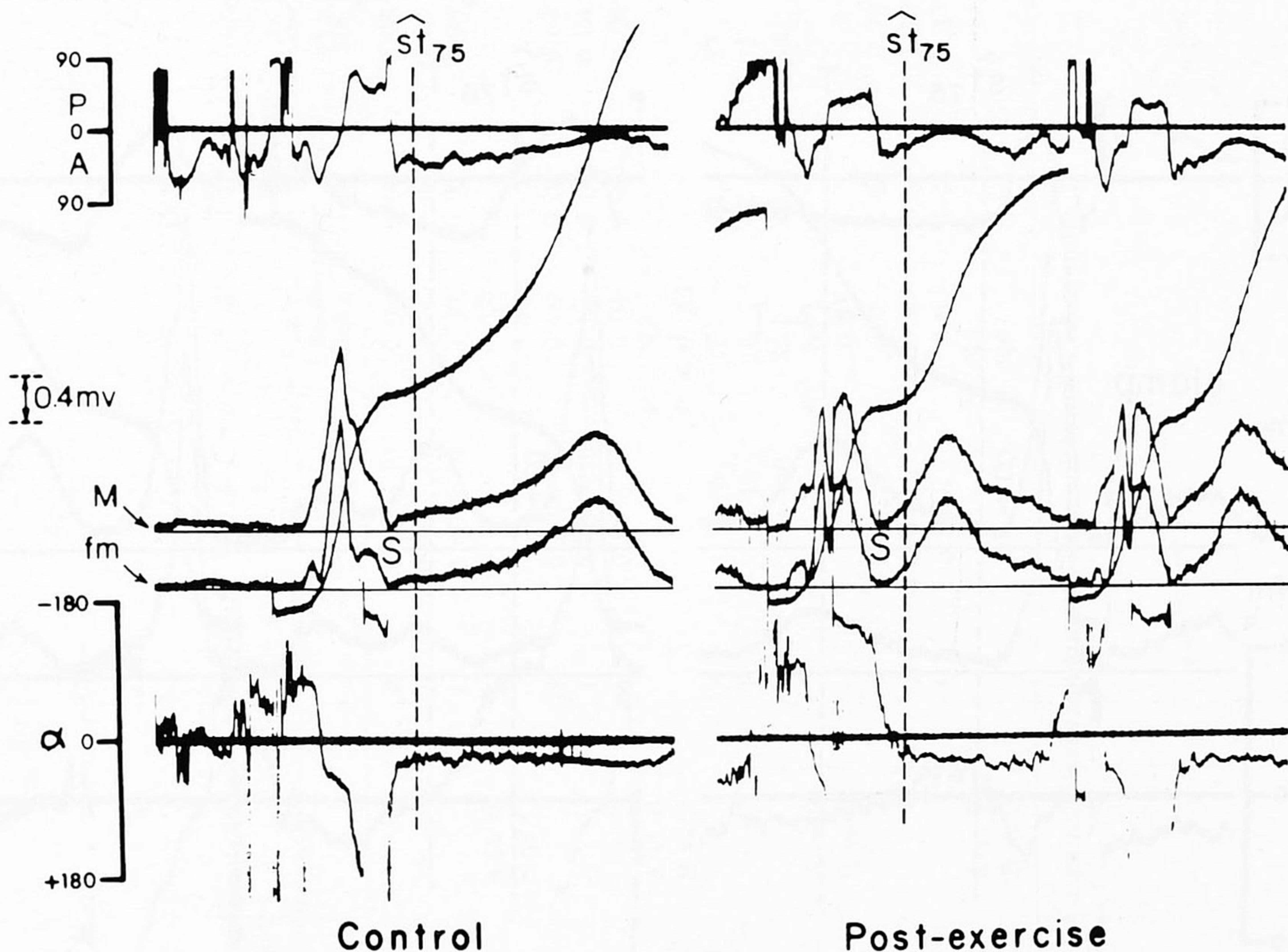


Figure 2

PCG in a normal young man, aged 22, provides an example of a marked change in the configuration of M_{QRS} and fm_{QRS} , the QRS complexes in the magnitude tracings (M and fm). Changes were frequently seen both in normal and in ischemic subjects, but in this case the change is so marked as to suggest transient right bundle branch block; however, the QRS duration increased by only 5 msec (5%). Note that MS , the spatial magnitude of \hat{S} , hardly increases following exercise and that the directions of \hat{st}_{75} and \hat{T} remain much the same. For orientation of PA (latitude) and α (longitude) see figure 6.

termination of the QRS complex, and downsloping of M_{ST} . Figures 3-5 show PCGs in typical cases. In addition to the changes in the M tracing, there was a rightward direction of early ST-vectors revealed by the α -longitude tracing approaching 180. The vector \hat{st}_{75} provides a typical sampling of ischemic vectors. At 75 msec after \hat{IR} —which is approximately at the middle of the QRS complex— \hat{st}_{75} is neither so early as to involve terminal QRS vectors nor so late as to involve the T wave, even at maximal heart rates.

Ischemia tends to direct \hat{st}_{75} in a direction opposite to that recorded in normal subjects. Before exercise, the direction of \hat{st}_{75} in the normal young men appears to be centered over lat 50A, long +30 (fig. 6, top panel). Immediately after maximal exercise, the center is scarcely different—lat 50A, long +20. In the normal middle-aged men the directions of \hat{st}_{75} were much the same, although more scattered (fig. 6, middle panel). In those ischemic subjects whose resting ECGs were normal, the resting \hat{st}_{75} directions corresponded more or less

to those in the normal subjects. However, in all the ischemic subjects there was a migration of \hat{st}_{75} toward the opposite side of the reference globe with exercise (fig. 6, bottom panel). This migration resulted in a rightward direction of these vectors; in the ECG this would correspond to depression of the ST-segments in leads I, V_5 and V_6 .

If we take 50A, +30, and 50A, +20 as typical normal directions of \hat{st}_{75} at rest and after exercise, respectively, we can express the deviation from normal by the spatial angle θ , obtained from a table¹ for determining the spatial angle between two vectors from their latitudes and longitudes.

Thus we have MS and θ as measures of the ischemic response. But note that when $M_{st_{75}}$ is zero the direction of \hat{st}_{75} is undefined: hence θ is undefined. To avoid difficulties with the direction of \hat{st}_{75} when its magnitude is small, θ is considered to be zero if $M_{st_{75}} \leq 0.04$ mV.

The values of the PCG measures of ischemia are summarized in table 1, which suggests that both MS and θ might be promising discriminants between

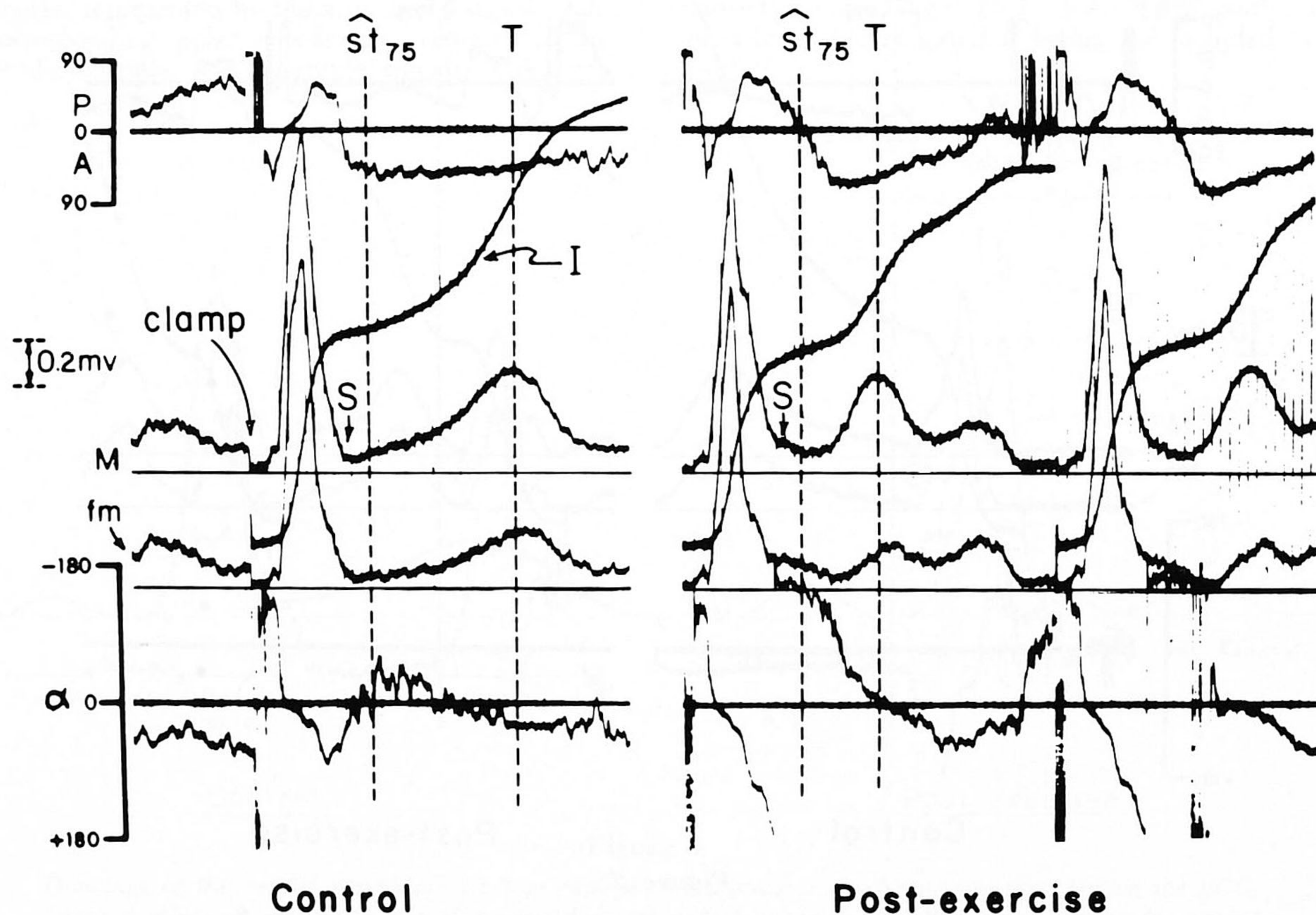


Figure 3

PCG showing typical ischemic response to exercise. Note increase in MS and change in longitude (α) of \hat{st}_{75} from approximately zero in the control α longitude tracing to close to -180 in the postexercise tracing. The I tracing is the integral of the M tracing. The integrator is automatically reset just before the QRS complex, when the baseline is clamped to establish zero. The I tracing is used to determine IR (see legend to figure 1).

Table 1

Polarcardiographic Measures of Ischemia in Three Groups of Men: (A) 29 Young Normal Subjects, (B) 27 Middle-Aged Normal Subjects, (C) 26 Ischemic Subjects

Measure	Units	Group	Before exercise			After exercise			P*			
			Range	Median	Mean	SD	Range	Median		Mean	SD	
MS	mV	A	0.00-0.10	0.02	0.03	0.03	0.03	0.00-0.08	0.02	0.04	0.04	NS
MS	mV	B	0.00-0.12	0.04	0.05	0.03	0.03	0.00-0.10	0.06	0.05	0.03	NS
MS	mV	C	0.00-0.10	0.00	0.02	0.03	0.03	0.08-0.40	0.18	0.20	0.09	<0.001
				P < 0.05						P NS		
				P < 0.01						P < 0.001		
Mst ₇₆	mV	A	0.00-0.16	0.08	0.09	0.03	0.03	0.04-0.32	0.12	0.15	0.07	< 0.001
Mst ₇₅	mV	B	0.00-0.16	0.04	0.09	0.04	0.04	0.02-0.25	0.12	0.11	0.05	NS
Mst ₇₆	mV	C	0.00-0.12	0.04	0.05	0.04	0.04	0.08-0.40	0.17	0.19	0.09	< 0.001
				P NS						P < 0.05		
				P < 0.001						P < 0.001		
θ	°	A	6-68	17	22	17	17	0-60	26	27	16	NS
θ	°	B	0-43	16	18	11	11	8-115	50	48	26	< 0.001
θ	°	C	0-160	35	46	44	44	66-166	128	126	26	0.001
				P NS						P < 0.001		
				P < 0.01						P < 0.001		
MS · θ	mV°	A	0.0-2.7	0.4	0.57	0.70	0.70	0.0-4.8	0.5	1.28	1.57	< 0.05
MS · θ	mV°	B	0.0-5.2	0.6	0.87	1.12	1.12	0.0-6.9	2.4	2.53	2.20	< 0.001
MS · θ	mV°	C	0.0-16	0.0	1.41	3.43	3.43	10.6-51.2	22.0	25.2	11.80	< 0.001
				P NS						P < 0.05		
				P NS						P < 0.001		

*P expresses value for unpaired t test comparing measure before and after exercise.

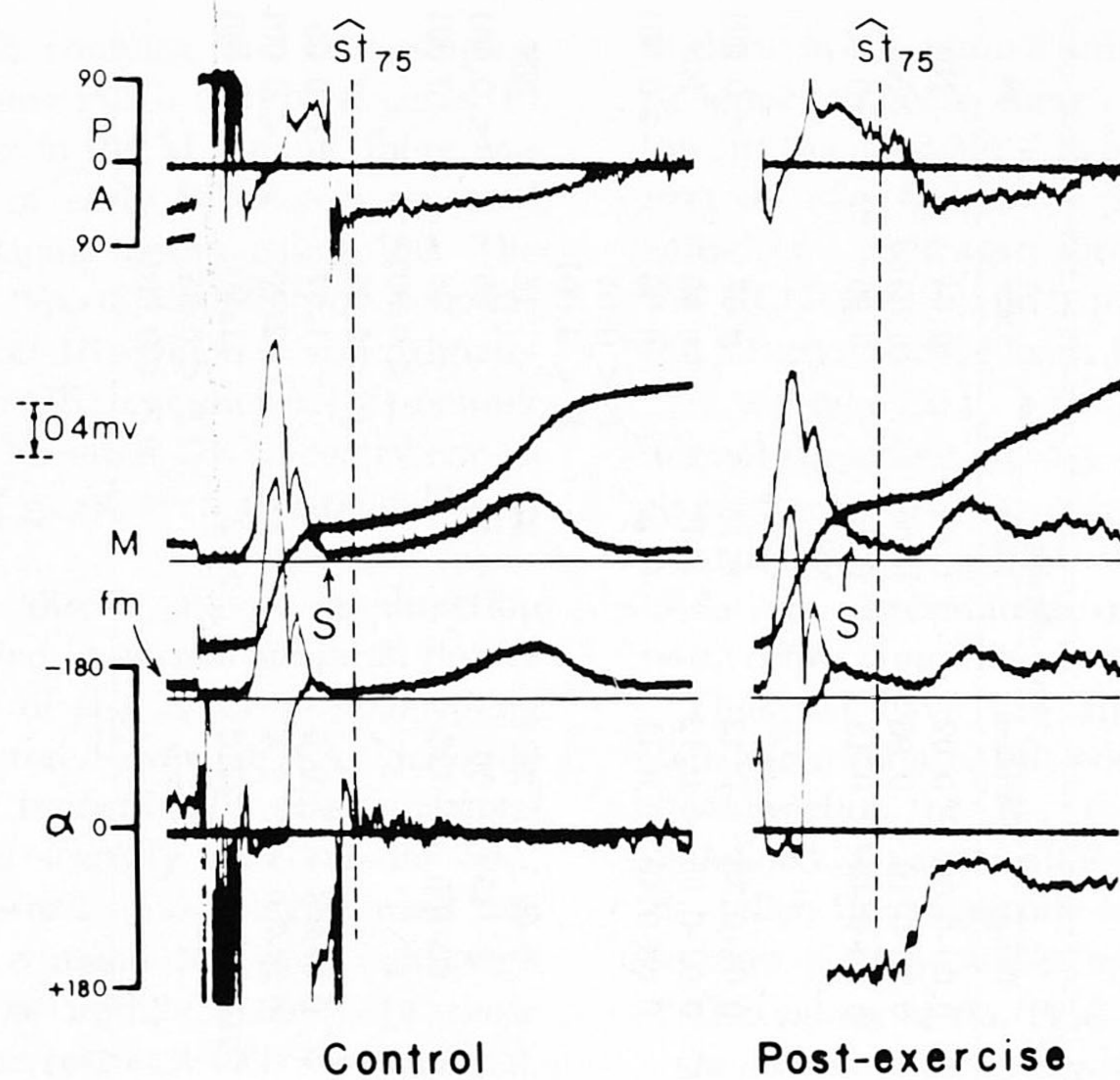


Figure 4

PCG showing typical ischemic response to exercise. Note increase in MS and downsloping MST. The configuration of M_{QRS} is also changed, but this is not diagnostic. The latitude (top tracing) of \hat{st}_{75} changes from 55A to 20P; the longitude changes from -10 to $+165$; the corresponding values of θ are 25 at rest and 139 after exercise.

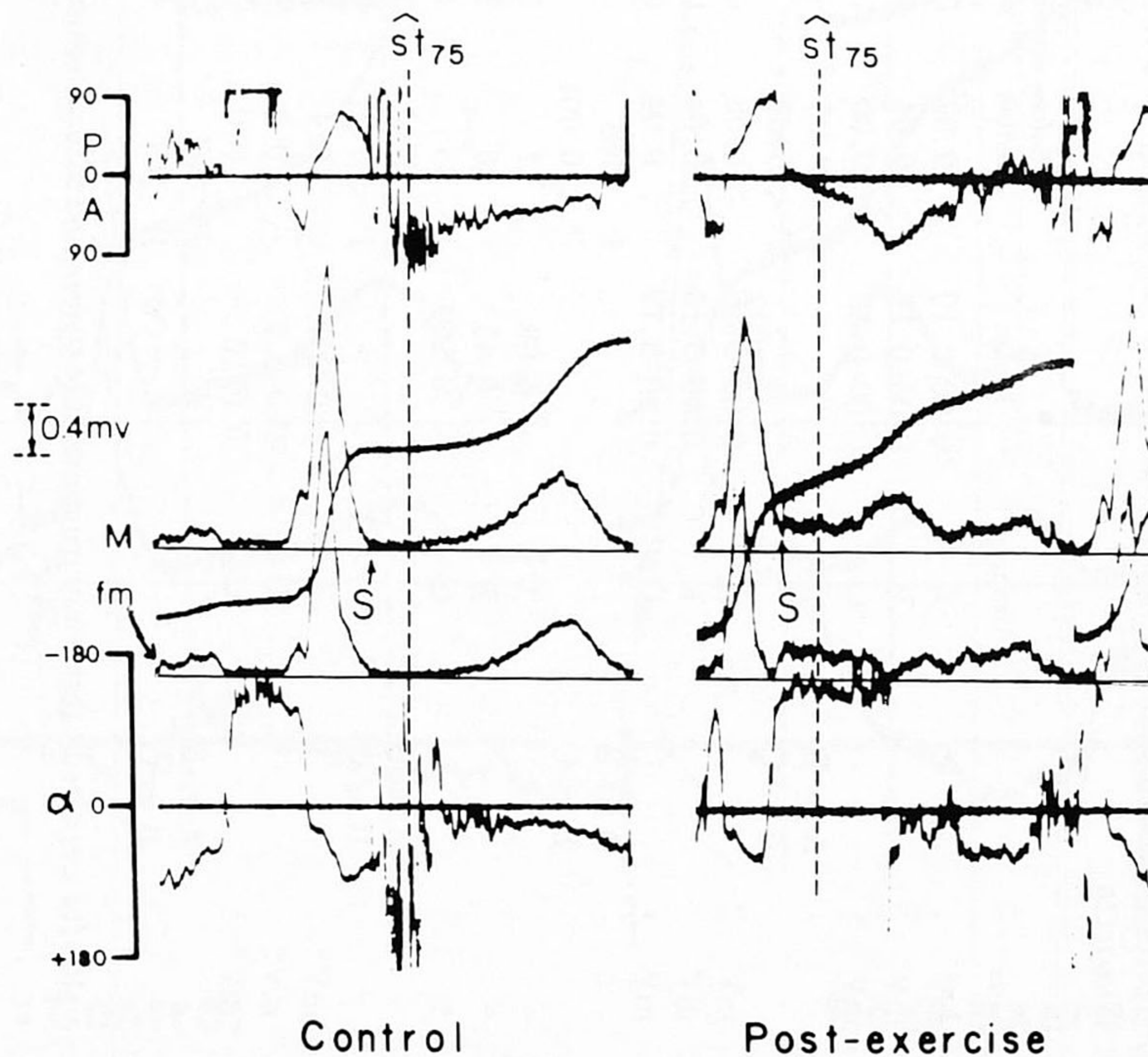


Figure 5

PCG showing typical ischemic response to exercise. Note increasing MS and downsloping or flat MST. The direction of \hat{st}_{75} is indeterminate in the control tracings because Mst_{75} is zero. After exercise, however, the direction of \hat{st}_{75} is 15A, -170 , giving a value for θ of 114.

Table 2

Mean Spatial Magnitudes of Maximum T Vectors

	Before exercise			After exercise			
	Young men (a)	Middle-aged men (b)	Ischemic men (c)	Young men (d)	Middle-aged men (e)	Ischemic men (f)	
MT:	Mean (mV)	0.55	0.61	0.38	0.66	0.61	0.33
	SD	0.18	0.17	0.15	0.18	0.15	0.14
		(a) vs (b)	(a) vs (d)	(b) vs (c)	(d) vs (e)	(e) vs (f)	
		NS	NS	<0.001	NS	<0.001	

N.B. No significant difference was found between young and middle-aged groups before and after exercise, in contrast to findings in the ischemic group.

normal and ischemic subjects, and that their product, $MS \cdot \theta$, might be even better. Note that in the normals $MS \cdot \theta$ after exercise ranged from 0 to 6.9, whereas in the ischemic subjects it ranged from 10.6 to 51.2, i.e., there was no overlap. On the other hand, there was some overlap in the extreme values of MS and θ of the normal and of the ischemic subjects.

Exercise produced little change in the directions of the maximum T vector, \hat{T} , in the normal young men and in the normal middle-aged men (fig. 7). However, in the ischemic subjects considerable changes were noted in many cases. In general, for all three groups the postexercise directional changes of \hat{T} were less than those of \hat{st}_{75} .

Changes in MT, the spatial magnitude of \hat{T} , are summarized in table 2. MT was significantly smaller in the ischemic subjects than in the normal, both at rest and after exercise. The changes in MT did not appear to be diagnostically useful.

Discussion

The relevance of the ST-segment to ischemia observed in the ECG is now documented in the PCG. The question arises as to whether or not polarcardiography makes the detection or recognition of ischemic vectors easier than, say, a simple bipolar lead such as CB_5 , employed in this study.

Any single ECG lead can have its axis perpendicular to some ischemic vectors and therefore not respond to them. The CB_5 lead may be an excellent choice because its axis is roughly parallel to the general direction of the ischemic vectors. This may be judged from the fact that the coordinates of the conventional V_5 lead vector in image space give it a direction close to 0,0, i.e., the center of the global plot used in this paper. Nevertheless, cases have occurred in which CB_5 has failed to show ST-segment depression, visible in leads III and aV_F synthesized from the Frank x, y, and z signals.

Obviously, the use of the orthogonal x, y, and z leads avoids this problem because any ischemic vector must reveal itself in at least one of them, and consequently, in the M tracing and at least one of the tracings which indicate direction.

Apart from being susceptible to ischemic vectors, regardless of their direction, the M tracing offers another advantage which is unique: there is no exact equivalent, in any ECG display, to the observation that MS is above a certain threshold value. This is because, in the ECG, the end of the QRS complex is not as clearly identifiable. (Note that the J point in the ECG does not correspond to S in the M tracing.) This advantage of the M tracing may be important because MS seems to be a sensitive indicator of ischemia. It is also much easier to measure than is a depressed J point.

The choice of \hat{st}_{75} as a typical vector was suggested by an observation of Bruce et al. that in order to reveal ischemia the best single point at which to sample the ST-segment of the postexercise ECG was 50 msec after the QRS complex.¹⁰ Translating this to the M tracing, we find that a roughly corresponding point in time would be 75 msec after $\hat{I}R$. Alternatives to \hat{st}_{75} which spring to mind are \hat{S} and \hat{ST} . Although the magnitudes of \hat{S} and MS were better discriminators than was \hat{st}_{75} , its direction was less promising, since \hat{S} represents a transition between QRS and ST events. In normal subjects, \hat{S} directions are not well defined, because MS is commonly zero. On the other hand, \hat{ST} , occurring halfway between \hat{S} and \hat{T} , was attractive inasmuch as its time of sampling was automatically adjustable for changes in the Q-T interval which attend increase in heart rate. An objection to \hat{ST} was that it tended to be rather late, thereby expressing the T wave more than the ST-segment. A solution would be to take \hat{ST} at some fraction less than half of the way from \hat{S} to \hat{T} . However, there

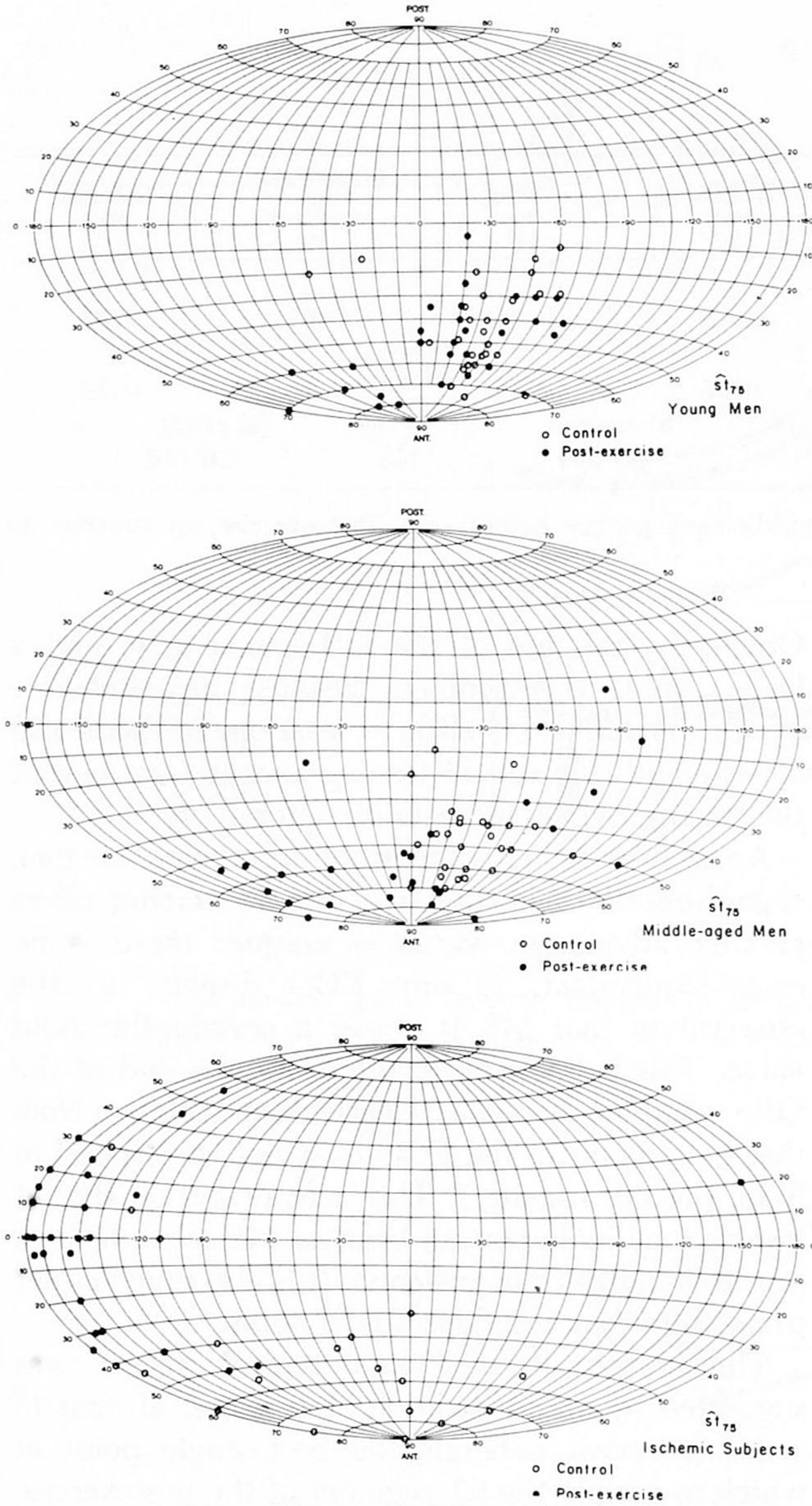


Figure 6 (above)

Plots of the directions of \hat{st}_{75} before and after exercise in 29 normal young men (first panel), 27 normal middle-aged men (second panel), and 26 men showing an ischemic ECG response to exercise (third panel). The centers of the spherical distributions of the directions in the young men are 50A, +30 in the control and 50A, +20 in the post exercise plots. Orientation of global projections with respect to the body are shown below.¹

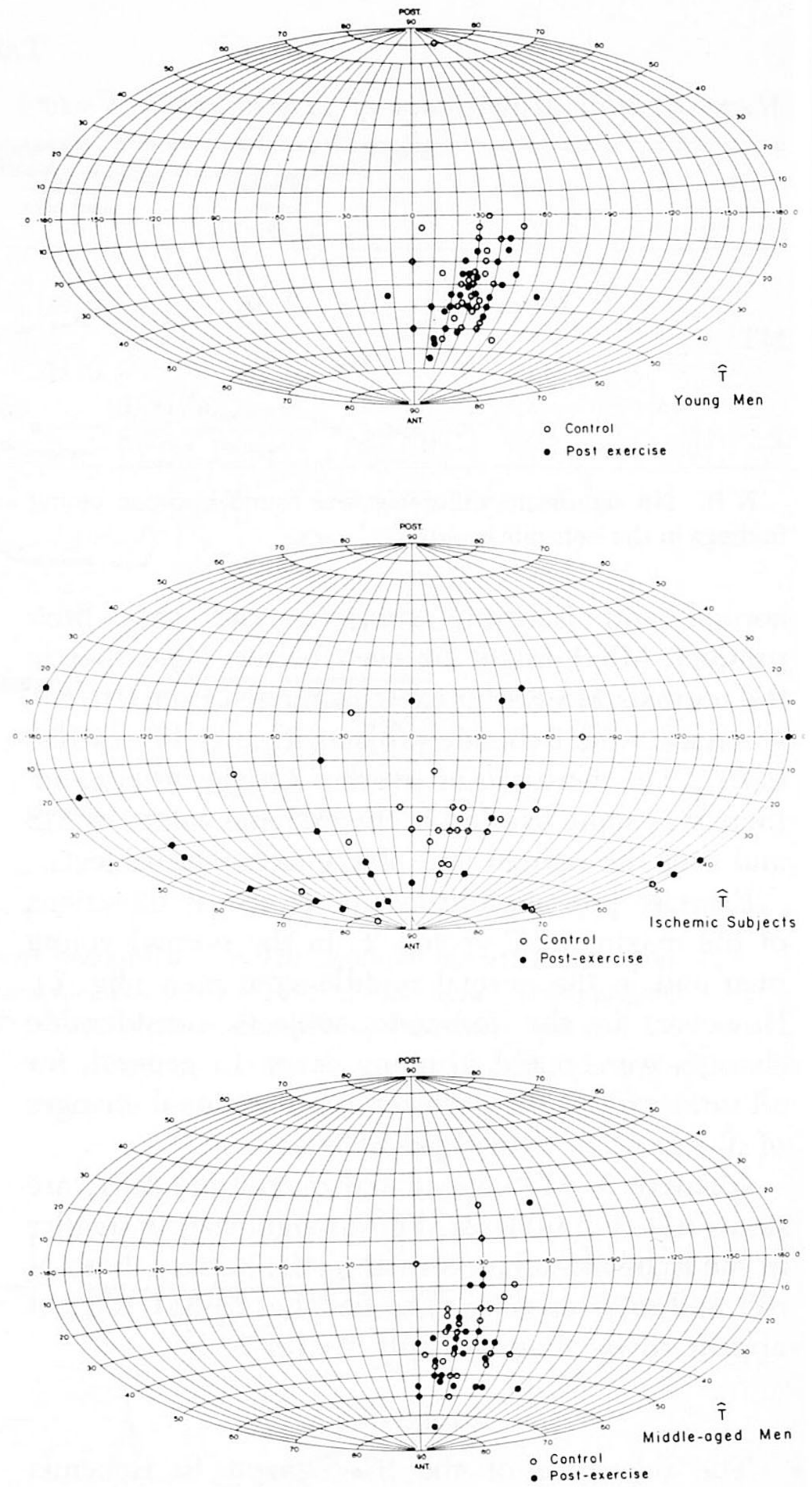
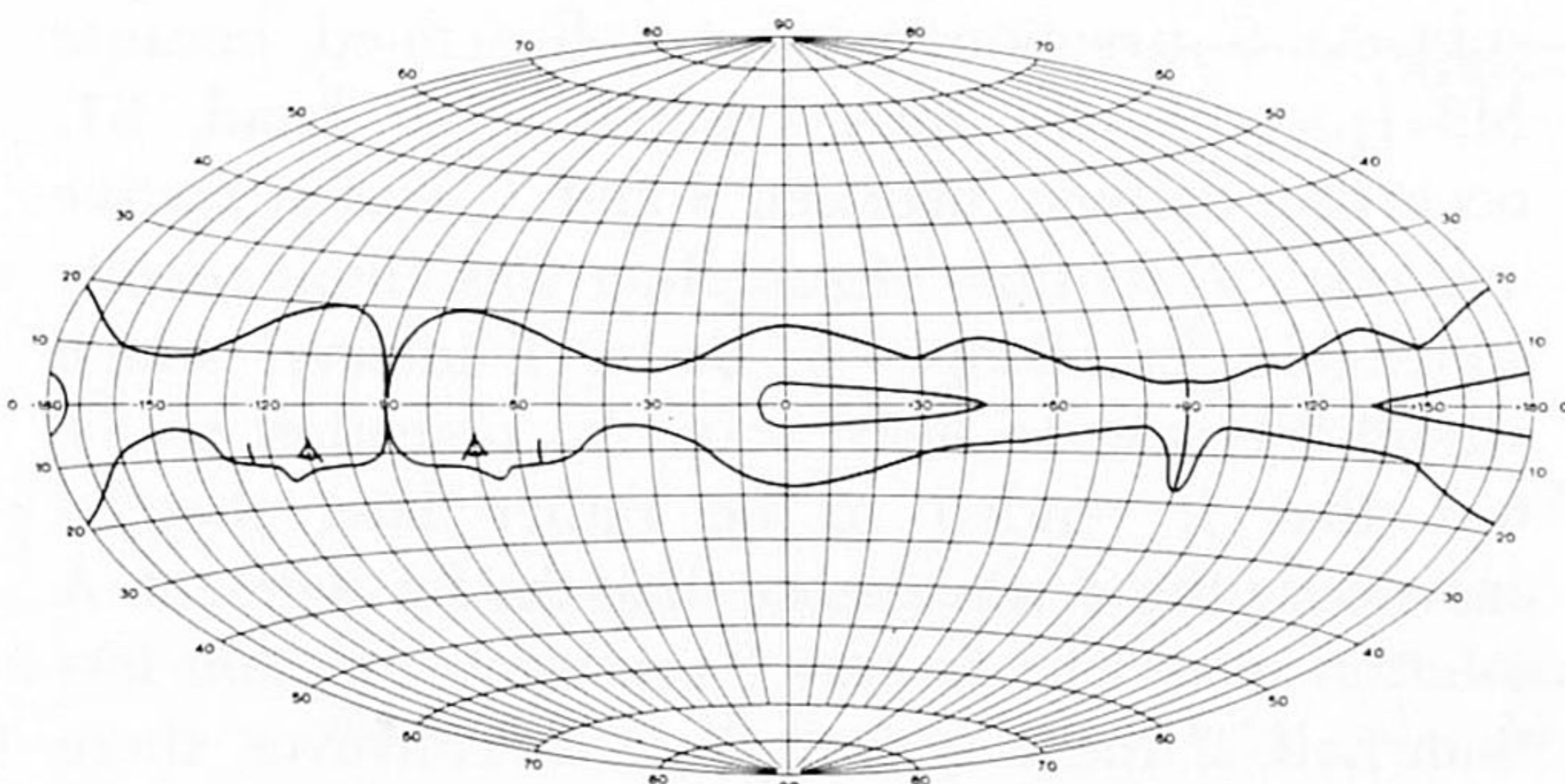
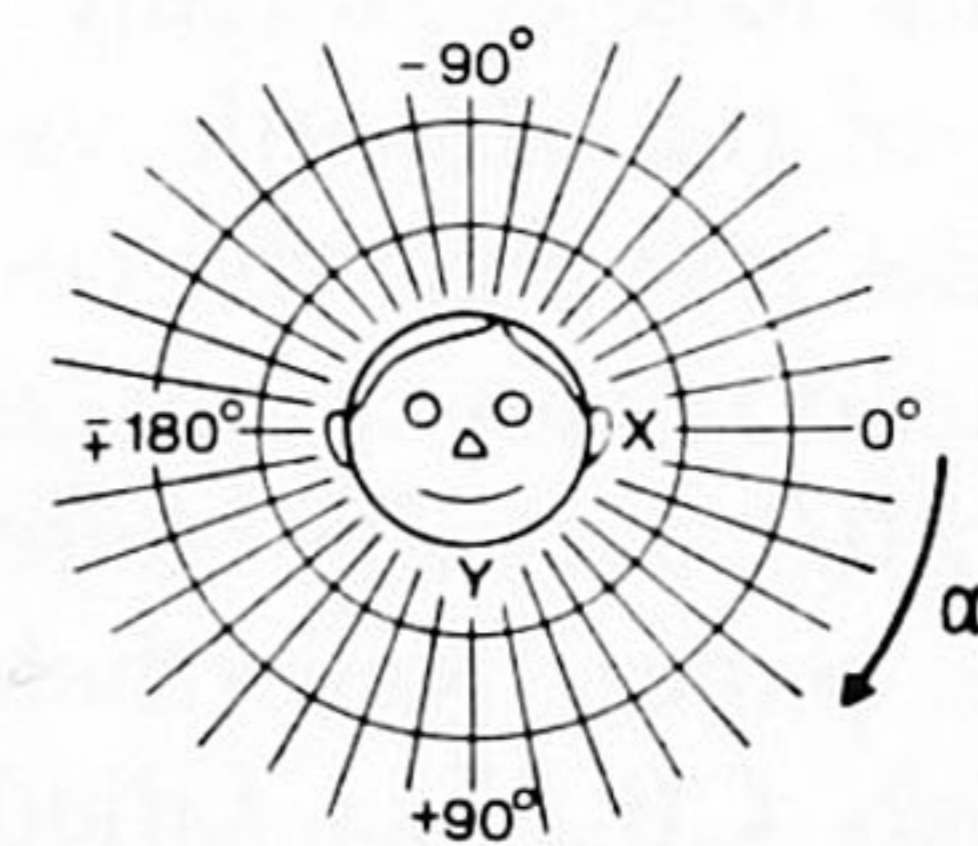


Figure 7 (above)

Directions of \hat{T} before and after exercise; figures represent the same subjects as in figure 6. In the normal subjects (first and second panel) there is less scatter of \hat{T} directions than of those of \hat{st}_{75} . Those ischemic subjects who had normal ECGs at rest had directions of resting \hat{T} roughly corresponding to those in the normal subjects (third panel).



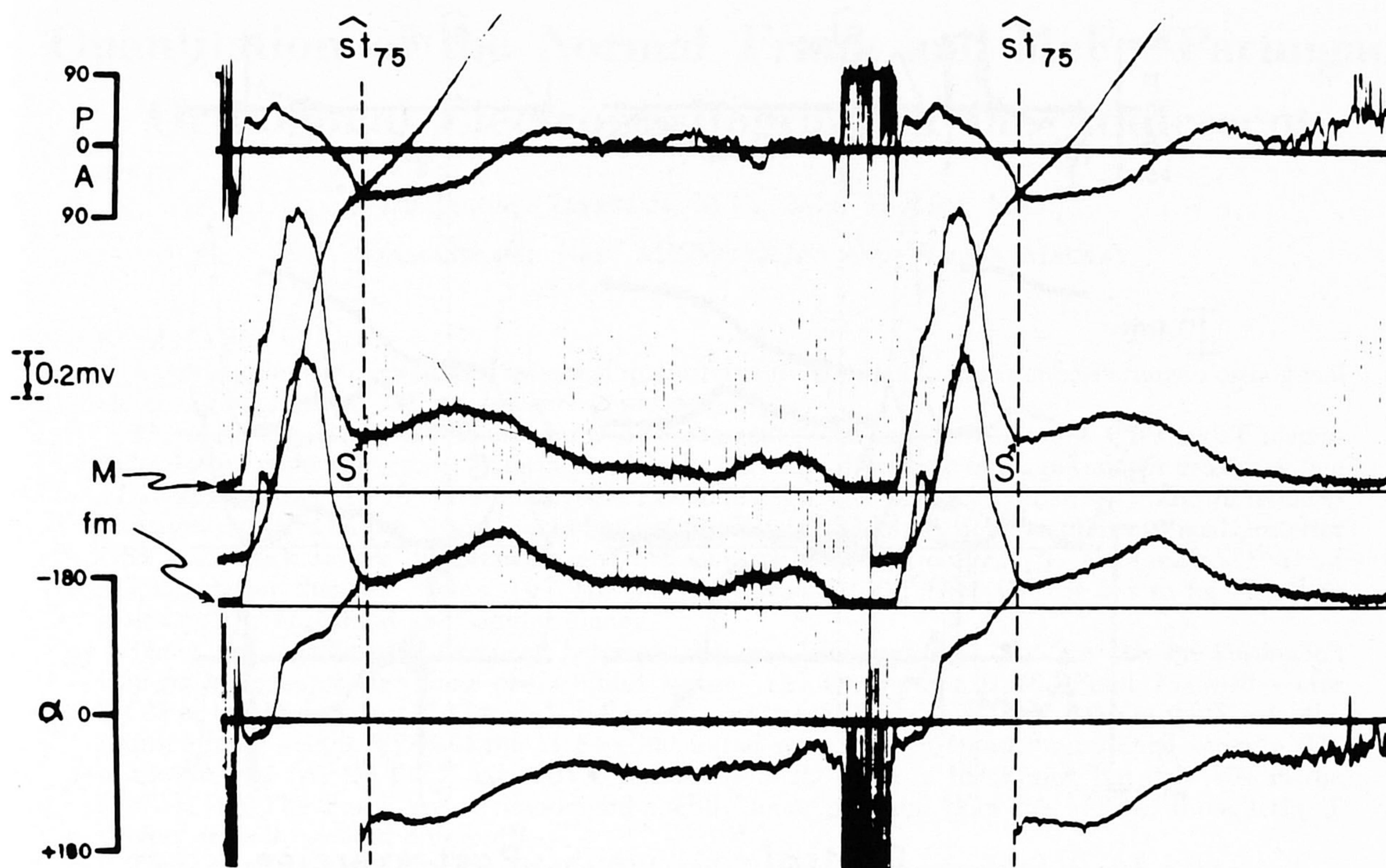


Figure 8

PCG in recent myocardial infarction. S is displaced upward by the current of injury. In this example \hat{st}_{75} lies close to \hat{S} because the QRS duration is 135 msec.

turned out to be another difficulty with \hat{ST} : in some abnormal tracings, the T wave became ill-defined, so that \hat{T} could not be precisely timed.

The combination of MS and θ in a simple product, although empirical, has conceptual justification. Let us first consider the product Mst_{75} and θ . This represents the length of the arc through which the head of \hat{st}_{75} must swing in order to become aligned with the direction taken to be normal such as $50A$, $+20$. The greater Mst_{75} or θ , the greater the arc. It seems reasonable to look upon the length of this arc as being proportional to the abnormality. For example, if Mst_{75} were very small, a large value of θ would actually represent only a small departure from a normally directed \hat{st}_{75} of the same magnitude. However, multiplying θ , a good discriminant, by Mst_{75} , a poor one, would not enhance diagnostic accuracy. But, whereas Mst_{75} does not happen to change in a typical manner, MS has a consistently small value in normal subjects and a consistently larger value in subjects manifesting an ischemic response. Thus the product $MS\theta$ combines the better magnitude discrimination of MS with the better directional resolution of \hat{st}_{75} to give an unambiguous, easily quantified measure of

ischemic abnormality. The superiority of $MS\theta$ is indicated by the lack of overlap between the ranges of the normal and the ischemic subjects after exercise. (The range for $MS\theta$ was up to 6.9 for the normal and above 10.6 for the ischemic subjects.) The determination of MS and θ can readily be programmed for computer analysis and may provide a valuable criterion for a positive exercise response.

High values of MS and $MS\theta$ are not unique to postexertional ischemia. They may be seen in recent myocardial infarction (fig. 8). In some subjects, MS may decrease with exercise (fig. 9). Probably, this does not signify an ischemic response. It indicates that the values of MS and $MS\theta$ after exercise should be compared with their control values.

It is clear from this study and from many ECG studies that the importance of ST -vectors in the diagnosis of ischemia is far greater than their magnitude. Accurate resolution of these vectors both temporally and with respect to their magnitude and direction appears to be justified. Such resolution is given by the polarcardiographic technique; a vectorcardiographic display does not provide it, nor does the electrocardiogram.

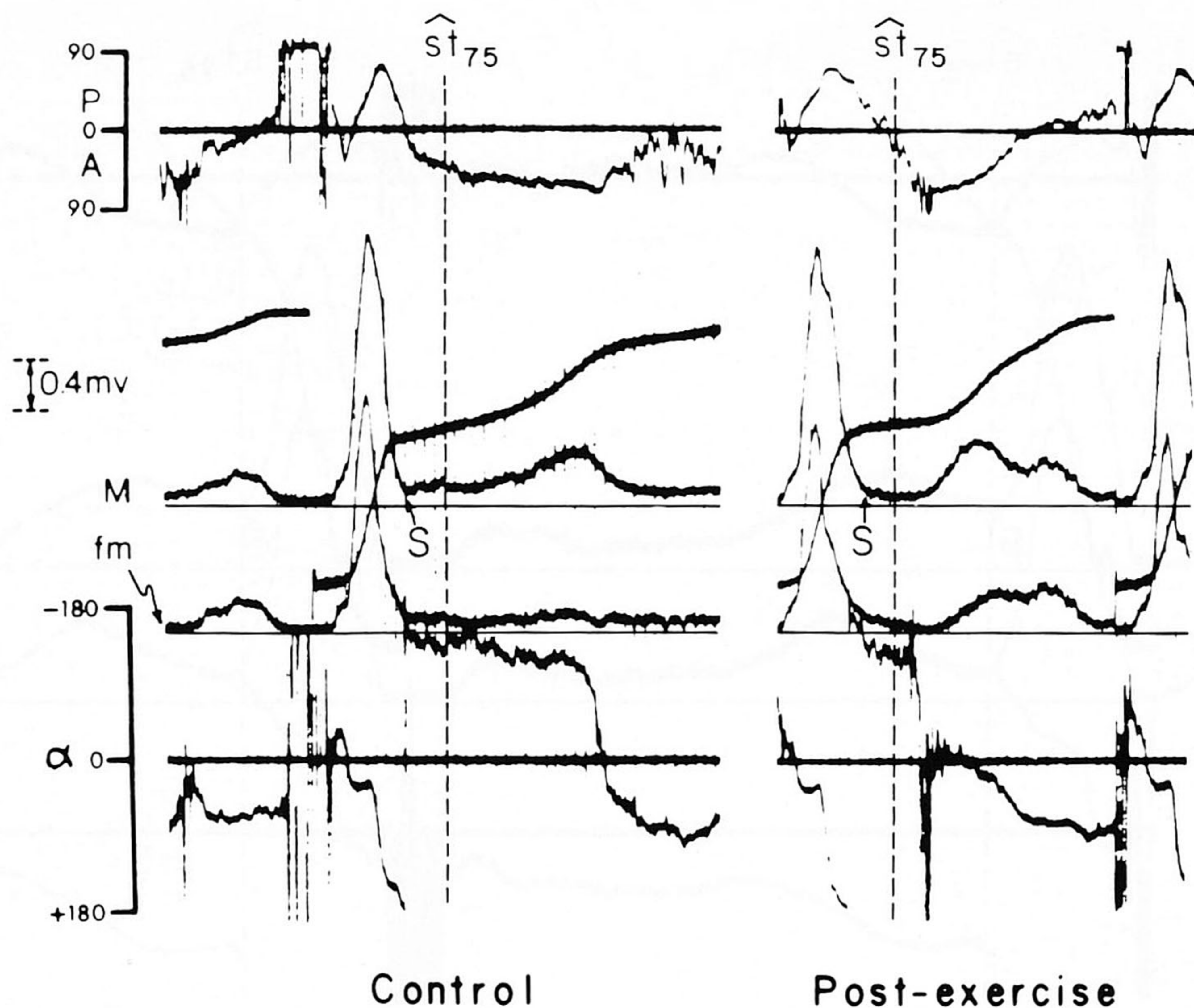


Figure 9

An example of a negative test response for ischemia associated with abnormal ST-segments and T waves at rest in a 45-year-old hypertensive woman (BP 215/135 mm Hg) with Cushing's Syndrome, who was not taking digoxin. The resting ECG showed ST-segment depression with inverted T waves, and changed little with exercise. Note that there is a slight decrease in MS, from 0.10 mV to 0.08 mV, between the control and postexercise PCGs. The directional abnormalities of the ST and T vectors are indicated by the PA latitude and a longitude tracings. MS- θ changed from 8.9 to 8.8.

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