Significance of Primary T Wave Aberrations in the Electrocardiogram of Asymptomatic Young Men

Part II. Working capacity and anthropometric data

Jan-Henrik Atterhög, Lars-Göran Ekelund, Gunnar Ericsson and Björn Ahlborg Department of Clinical Physiology, Karolinska Hospital, Stockholm, Sweden

ABSTRACT

Fifty-one healthy men, 18 - 19 years of age, with "organic" T wave aberrations (group T) were compared to 112 controls of the same age with normal electrocardiograms (Group A + B). Group T had increased heart rate and higher systolic and diastolic blood pressures. Their lower physical work capacity could be "explained" in multivariate analysis by their shorter height, smaller heart volumes and lower total hemoglobin (the latter not constituting a significant difference between the groups). Multiple regression analysis indicated that differences in heart rate, systolic blood pressure and blood volumes explained differences between subjects with and without primary T wave aberrations. These variables are all influenced by changes in sympathetic discharge, whereas no variable representing performance or body dimensions contributed to the explanation.

INTRODUCTION

A low T wave without concomitant ST depression, a notch in the T wave which may become inverted, or a break in the trend of the T wave amplitude from V1 through V6 are often accidental findings in the electrocardiograms (ECGs) of young men who are otherwise healthy and without any signs or history of heart disease (1, 9, 13). These T wave changes have been reported to occur in organic heart disease (3, 13, 18) and have, therefore, sometimes led to disablement when found in young apparently healthy people (7, 14, 15).

It was indicated in two earlier studies (1, Atterhög, J-H & Malmberg, P, unpublished) that young men with such T wave aberrations of an "organic" type had increased sympathetic tone and differed from subjects with normal ECGs regarding some anthropometric data. The aim of this study was to further characterize subjects with these T wave abnormalities and elucidate possible causes of the aberrations and their functional significance.

MATERIAL

Subjects 18 - 19 years of age with T wave aberrations and a random sample of subjects of the same age and with normal ECGs were selected from a military indiction center as described in detail in part I of this study (2). The selection criteria were the T wave abnormalities mentioned in the introduction and reported earlier (2). The ECG aberration was the only finding and both groups were healthy without other signs or history of heart disease. The subjects were divided into the following groups:

Group T subjects with T wave aberrations at rest and during orthostasis, (n = 51).

The normal ECG group A + B, n = 112, which was subdivided into:

Group A: subjects with normal ECGs in the supine position and during orthostasis (n = 84).

Group B: subjects with normal ECGs in the supine position but T wave aberrations during orthostasis (n = 28).

METHODS

The procedure has been described in detail earlier (2) and included a standardised orthostatic test as well as an exercise test (18, 20). The load giving a heart rate (HR) of 170 beats/min (W 170) was used as a measure of the physical work capacity. HR was calculated from a 30 seconds' ECG recording. Blood pressures (BP) were measured by ascultation with a calibrated cuff on the right upper arm by the same person throughout the whole study. A single estimation of the total amount of hemoglobin (THb) was determined by Sjöstrand's alveolar CO method and blood volume (BV) was calculated from a peripheral Hb concentration estimation with correction of body hematocrit by a factor of 0.91 (6). Heart volume (HV) was measured in the supine position (11). Body surface area and body volume were calculated according to Sendroy & Cecchini and Sendroy and Collison (16, 17).

The differences between group means were tested for significance using Student's t-test. The relation between variables was studied with linear regression analysis and in the analysis of significance of differences between regression lines covariance analysis was applied. In the analysis of multivariate relations a sometimes hierarchical stepwise multiple linear regression analysis was used.

RESULTS

Cumulative distribution curves for some anthropometric data. In figures 1 - 4 the cumulative distributions of HR at rest, SBP at rest, HV and W 170 for groups T, A and B are presented. The curves show a normal distribution with a parallel displacement to each other. The distribution curves for group B were generally closer to the group T curves than to those of group A, however.

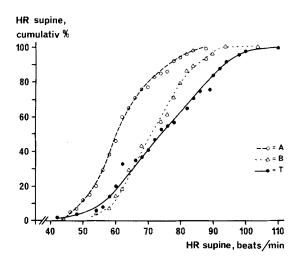


Fig. 1. Cumulative distribution of HR at rest in the groups A (n = 84), B (n = 28) and T (n = 51).

Sixteen percent of group T and 6 % of group B had a HR at rest of 90 beats/min or more, whereas none in group A reached that HR level (Fig. 1).

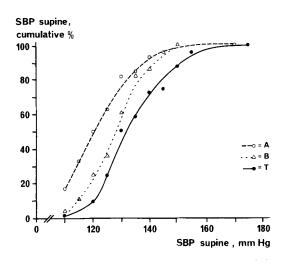


Fig. 2. Cumulative distribution of SBP at rest in the groups A (n = 84), B (n = 28) and T (n = 51).

Twenty percent of group T had a SBP of 145 mm Hg or more compared with 3 % in group A and 6 % in group B (Fig. 2). Twelve percent in group T had a SBP of more than 145 mm Hg and one subject had 175 mm Hg. Only one subject in group A had a SBP more than 145 mm Hg (170 mm Hg). The cumulative distribution curves for height, DBP and BV showed similar trends as those reported above, i.e. the group B curves were closer to those of group T than group A.

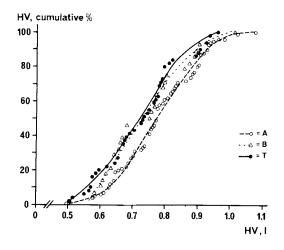


Fig. 3. Cumulative distribution of HV in supine position in the groups A (n = 84), B (n = 28) and T (n = 51).

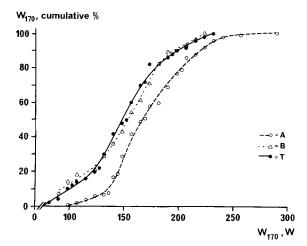


Fig. 4. Cumulative distribution of W 170 in the groups A (n = 84), B (n = 28) and T (n = 51).

Comparison between groups A and B. There were no differences in height, weight, HV, THb, BV or Hb between the two control groups A and B (Table 1). Group B, however, had on the average 10.4 beats/min. higher HR than group A while at rest (Table 2). The increase in HR during orthostasis, as well as the orthostatic HR, was significantly larger (p< 0.01) in group B. SBP in the supine position was also an average of 5.7 mm Hg higher in group B than in

group A. There was no difference between the groups regarding orthostatic SBP or DBP at rest and during orthostasis or regarding SBP fall and DBP increase during orthostasis.

Group B had lower W 170 than group A (Table 3) with a difference of 20.1 W (Table 3). No difference was found between the groups in HR at the end of exercise while the decrease in HR after exercise was slower in group B.

Comparison between group T and group A + B. The subjects in group T were on the average 3.2 cm shorter than the controls (Table 1). There were no differences in weight or THb. Mean HV and BV were smaller in group T, whereas HB concentration was higher.

On the average, mean HR at rest was 9.6 beats higher and SBP and DBP in the supine position was 10.9 and 4.3 mm Hg, respectively, higher in group T. During orthostasis the increase in mean HR in group T was not significantly different from the increase in group A + B whereas SBP decreased more and DBP increased less in group T (p < 0.05 and < 0.01 respectively).

The orthostatic HR and SBP were higher in group T whereas the significance of difference in DBP between all the groups observed while at rest disappeared during orthostasis.

Table 1. Some anthropometric data for the control groups without (A) and with (B) T wave aberrations during orthostasis, for the total control group (A + B) and for the group with T wave aberrations both at rest and during orthostasis (group T).

Variable	Group A mean SD	Group B mean SD	sign. of diff.	Group A+B mean SD	Group mean	T SD	sign. of diff.
Height cm	180.2 6.6	179.3 6.3	n.s.	180.0 6.5	176.8	7.0	p<0.01
Weight kg	68.5 10.1	67.2 9.5	n.s.	68.2 9.9	66.8	8.8	n.s.
HV 1	0.789 0.150 n=84	0.742 0.12 n=28	1 n.s.	0.777 0.12 n=112	5 0.730	0.120	p<0.05
THb g	724.8 90.7 n=67	705.6 79.9 n=25	n.s.	719.6 87.9 n=92	694.5 n=29	111.6	n.s.
BV 1	5.67 0.75 n=67		n.s.	5.63 0.72 n=92	5.25 n=29	0.89	p<0.05
Hb g/l	140.9 9.2 n=67	140.6 7.5 n=25	n.s.	140.8 8.7 n=92	146.0 n=29	9.5	p<0.01

Physical work capacity was lower in group T with a difference to the control group A + B of 17.2 W (Table 3). Both groups finished the exercise test

at the same HR but HR immediately after the test in recumbent position and 10 minutes afterwards was higher in group T. There were no differences between group T and group B regarding W 170 and HR after exercise.

Simple linear regression. W 170 was correlated to height, weight, HV, THb and BV in both control group A + B and group T (Table 4). There were no significant differences found in the correlation coefficients between the groups. The regression lines for W 170 as the dependent variable in Table 4 were all significantly different between group A + B and group T, with a lower physical capacity in group T at a given height, weight, HV, THb or BV, but there were no differences regarding the slopes, however.

The relationship between HV and height, weight, body surface and volume, THb and BV is given in Table 5. There was no correlation between HV and BV in group T. In addition, there were no significant differences in correlation coefficients between the group T and group A + B. HV as a function of THb in group T had a significantly smaller slope than in group A + B. HR at rest was negatively correlated to height in group A + B and to W 170 for both controls and group T (Table 6). HR was numerically higher in group T for a given W 170 (p<0.10). The HR increase during orthostasis was not related to height or physical work capacity in either groups but negatively correlated to HR at rest in the groups B and T and to DBP at rest in control group A.

Table 2. Heart rate, systolic and diastolic blood pressures at rest and during orthostasis for the control groups without (A) and with (B) T wave aberrations during orthostasis, for the total control group A + B and for the group with T wave findings both at rest and during orthostasis (T).

	Group n=84	A	Group n=28	В	Group n=112	A+B	Group n=51	T
HR, supine, beats/min	mean 63.0			SD 11.1	mean 65.6	11.5		SD 15.2
sign. of diff.		p <c< td=""><td>.001</td><td></td><td></td><td>pto</td><td>.001</td><td></td></c<>	.001			pto	.001	
SBP, supine mm HG	124.3		-	9.9	125.7		136.6	13.5
sign. of diff.		рС	.05			p<0	.001	
DBP, supine mm HG	73.2	7.7	76.4	8.4	74.0	8.0	78.3	6.9
sign. of diff.		n.s	•			p<0	.01	
HR, standing beats/min	80.7			10.2	84.6		93.4	16.0
sign. of diff.		p<0	.001			p<0	.001	
SBP, standing mm Hg	124.3	10.8	128.4	9.2	125.3		133.9	12.4
sign, of diff.		n.s	•			p<0	.001	
DBP, standing mm Hg	80.7	8.2	82.9	6.4	81.3	7.8	82.6	7.1
sign. of diff.		n.s				n.s		

Table 3. Physical work capacity (W 170) and heart rate during and after exercise for the control groups without (A) and with (B) T wave aberrations during orthostasis, for the total control group A + B and for the group with T wave aberrations both at rest and during orthostasis (group T).

	Group n=84	A	Group n=28	В	Group n=112	A+B	Group n=51	Т
	mean	SD	mean	SD	mean	SD	mean	SD
W 170 W	174.1	34.7	154.0	36.2	169.1	36.0	151.9	34.6
sign. of diff.		p <0	.01			p<0	.01	
HR at highest exercise load beats/min	168.1			6.6	168.3	7.1	169.2	7.2
sign. of diff.		n.s	• 4			n.s		
HR immediately after exercise in recumbant beats/min	102.6	14.2	112.8	14.3	105.1	14.8	110.7	18.1
sign. of diff.		p ∠ 0	.01			p<0	.05	
HR 10' after exercise in recumbant beats/min		9.1	85.6	10.1	81.3	9.7	86.3	12.5
sign. of diff.		p<0.	01			p <0	.01	

SBP at rest was correlated to HR at rest in both groups (Table 6) and was significantly higher in group T than in group A + B at corresponding HR (p < 0.05) (Fig. 5). The SBP fall during orthostasis was positively correlated to HR, SBP and DBP at rest in groups A and A + B but for groups T and B was positively correlated only to SBP at rest.

DBP at rest was correlated to HR at rest only for group T (Table 6). The DBP increase was not correlated to HR at rest whereas the increase was negatively correlated to basal DBP for all groups.

Table 4. Correlation coefficients (r) and significance of r for physical work capacity (W 170) and some variables in control group A + B and in group T and significance of differences between the regression lines.

Regressions Grou		coup A+B		T	Sign. of diff. between groups A+B and T in		
W 170 = f ()	r	sign. p<	r	sign. p<	intercept p<	slope p	
Height	0.46	0.001	0.39	0.01	0.05		
Weight	0.44	0.001	0.40	0.01	0.01	_	
HV	0.43	0.001	0.54	0.001	0.05	_	
THb	0.56	0.001	0.44	0.05	0.01	_	
BV	0.51	0.001	0.55	0.001	0.05	-	

Table 5. Correlation coefficients (r) and significance of r for heart volume (HV) and some variables in control group A + B and in group T and significance of differences between the regression lines.

Regressions	Group	A+B	Group	T	Sign. of diff groups A+B an	
HV = f ()	r	sign. p<	r	sign. p<	intercept p<	slope p<
Height	0.21	0.05	0.39	0.01	_	-
Weight	0.54	0.001	0.60	0.001	-	-
Body surface	0.48	0.001	0.56	0.001	_	-
Body volume	0.54	0.001	0.60	0.001	-	-
THb	0.67	0.001	0.44	0.05	-	0.05
BV	0.59	0.001	0.34	-	-	

Table 6. Correlation coefficients (r) and significance of r for the functions HR, SBP and DBP at rest, differences of HR, SBP and DBP between supine and erect positions and some variables in the groups A, B, A + B and T.

Regressions	Group n=84		Group n=28	В		A+B	-	T
	r	sign.	r	sign.	r	sign.	r	sign.
HR supine = f ()								
Height	-0.30	0.01	-0.02		-0.23	0.05	-0.09	
W 170	-0.29	0.01	-0.19		- 0.33	0.001	-0.30	0.05
HR increase supine -	erect							
Height	0.10		0.26		0.13		0.26	0.10
W 170	-0.03		0.00		-0.08		-0.09	
HR supine	- 0.19	0.10	-0.58	0.001	-0.18	0.10	-0.31	0.05
DBP supine	-0.29	0.01	-0.25		-0.22	0.05	-0.14	
SBP supine = f ()								
HR supine	0.30	0.01	0.32	0.10	0.36	0.001	0.44	0.001
SBP fall supine -erec	<u>t</u>							
HR supine	0.30	0.01	0.05		0.26	0.01	0.11	
SBP supine			0.43					0.001
DBP supine			0.30			0.05		
DBP supine_= f ()								
HR supine	0.11		0.14		0.14		0.28	0.05
DBP increase supine - erect = f ()								
HR supine	0.11		0.27		0.00		0.09	
DBP supine	-0.43	0.001	-0.65	0.001	-0.48	0.001	-0.37	0.01

Table 7. Partial regression coefficients (b) standard errors of b (SE $_{b}$) and significance of b for the predictors in hierarchical step-wise multiple regression analysis on W 170 for the total material (group T entered by force). The constant, the coefficient of multiple determination (R 2) and the number of degrees of freedom are given for the regression equation.

Predictor		material SE _b	(groups A + t	B and T)
Group T	-8.2	5.8	1.424	n.s.
HV	85.1	28.0	3.040	0.01
THb	0.098	0.036	2.698	0.05
Height	1.06	0.45	2.34	0.05
Constant	-157.5			
R^2	0.40			
Df	115			

The fall in SBP was positively correlated to the increase in DBP during orthostasis in group A + B (p < 0.001) whereas no such correlation was found in group T. Otherwise there were no correlations found in either group A + B or group T between the differences in HR, SBP and CBP when their position was changed from supine to erect.

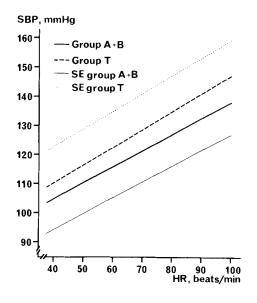


Fig. 5. Regression lines and SE fro SBP_{at} rest = $f(HR_{at}$ rest) in groups A + B (n = 112, continuous line) and T (n = 51, interrupted line). Group A + B: y = 0.36x + 103, Group T: y = 0.40x + 107.

Both THb and BV were correlated to body volume in all groups but the regression lines had a steeper slope in group T.

Step-wise linear multiple regression analysis. The mean difference in W 170 between group A + B and group T can be expressed by what part group T contributes to variations in W 170 in the whole sample consisting of groups A + B and T. With hierarchical step-wise multiple regression analysis the dichotomized variable group T (= 1 if the subject belonged to group T, otherwise = 0)

was entered as the first predictor giving the following equations using computer supplied constants: W 170 = 169 - 17.1 x group T. The t value for the slope (b) was significant (p<0.02). After the other variables HV, THb and height had entered in that order, the significance of b group T had disappeared (Table 7). The other competing variables were weight and HR at rest. I.e. the difference in W 170 between group A + B and group T could be significantly explained by the differences in body dimensions.

The difference in resting mean SBP between group T and control group A + B was analysed in the same manner. SBP = 125.7 + 0.383 x group T. The t-value for b was 5.244 (p<0.001). After control of HR at rest there remained a significant difference regarding SBP (t-value for b was 3.577, p<0.001) i.e. the differences in SBP cannot be entirely explained by differences in HR. The competing variables of height and W 170 did not contribute significantly. The difference in mean BV between the groups remained significant after correction for body volume and HV (t-value for b was 2.023, p<0.05) i.e. the differences in BV cannot be entirely explained by differences in body dimensions. The other competing variables were HR at rest and SBP at rest.

With the aim of studying which of the measured anthropometric and functional variables contributed significantly to the differences between group T and group A + B (besides the T wave abnormality), when the other variables were controlled, a multiple regression analysis was performed on the whole sample A + B + T (n = 163) with the dichotome variable t as a dependent variable (t = 1 if the subject belongs to group T, otherwise = 0).

Table 8. Partial regression coefficients (b), standard errors of b (SE_b) and significance of b for the predictors in step-wise multiple regression analysis on T wave aberrations for the total material. The constant, the coefficient of multiple determination (R^2) and the number of degrees of freedom are given for the regression equation.

	Total ma	aterial (g	roups A + B	and T)				
Predictor	р	SEb	t	р				
SBP at rest	0.011	0.304	3.239	0.005				
BV	-0.111	0.050	2.214	0.05				
HR at rest	0.006	0.003	1.725	n.s.				
Constant -0.842								
	.21							
Df 116								

The subjects with T wave aberrations (group T) differed significantly from the others (group A + B) regarding SBP at rest, BV and HR at rest in order of

influence (Table 8). The other competing variables were THb, height, HV and W 170 which did not contribute to "explain" the T wave aberrations.

DISCUSSION

This study showed signs of increased sympathetic discharge in young asymptomatic men with T wave aberrations of the types reported to occur in organic heart disease. They thus have increased HR and SBP which confirm earlier findings (1, Atterhög, J-H & Malmberg, P, unpublished).

In addition, those in the control group who evidenced T wave aberrations during orthostasis (group B) already had higher HR and SBP at rest and a larger increase in orthostatic HR than the subjects with normal ECGs at rest as well as during orthostasis (group A). Thus, a high sympathetic tone was also shown by group B.

A changed vegetative tone is the probable explanation for both the lack of correlation between HR at rest and body dimensions (height) in either groups T or B, and for the weak correlation between HR at rest and physical work capacity in group T, whereas the corresponding correlations in group A were normal (20). The changed vegetative tone was also evident from the slower decrease in HR after the termination of exercise in groups T and B.

The curves in group B for the cumulative distribution of the measured variables generally show a closer similarity to group T than to group A. This also indicates that the T wave aberrations in group T are not signs of organic lesions. There was not any "skewness" in the cumulative frequencies of HR, HV and W 170 that might indicate the occurrence of any subgroup in group T. This means, for example, that vasoregulatory asthenia (VA) can be excluded as a significant cause of the T wave aberrations in group T because patients with VA generally have substantially increased HR, abnormal HR reaction during orthostasis and a low W 170 (10). The cumulative distributions also exclude the contribution of well trained sportsmen, which may have primary T wave aberrations (8), at least during their most active training periods (5). As a result of their heavy physical training they can be expected to have a low HR at rest, enlarged HV and high W 170 (23) and such a group could not be identified in this material. Likewise, organic heart disease causing enlarged heart volumes and low physical working capacity can be excluded as significant contributor to the changed T waves.

The correlations between W 170 (an indirect measure of stroke volume), HV and THb (a variable reflecting the degree of habitual physical activity) were performed since there normally is a relationship between these variables (20). These relationships are of use in differniating between, for example, a normal

and abnormal cause of the low physical work capacity in a subject. A low physical work capacity is probably normal for that subject if also HV and THb are low.

Thus, group T had a lower W 170 than the controls but this difference could be entirely explained by differences in the body dimensions HV, THb and height and was probably not the result of heart disease. This confirms an earlier finding that the group differences in maximal work capacity between subjects with and without primary T wave aberrations are height dependent (Atterhög, J-H & Malmberg, P, unpublished). We have no explanation why subjects with T wave changes are shorter, which is a recurrent finding.

Group T had a smaller BV than the control groups which could not be explained by differences in body dimensions. Since the Hb concentration was increased in group T and THb was similar it is probable that the decrease in BV in group T can be explained by a plasma volume decrease due to increased sympathetic tone (4, 22).

SBP was significantly higher in group T than in the controls at any given level of HR and thus differences in SBP between the groups could not entirely be explained by differences in HR.

The difference between group T and the rest of the subjects could be explained by multiple regression analysis by differences in SBP at rest, BV and HR at rest, in that order of influence, i.e. variables that can be influenced by changes in the sympathetic tone. On the other hand, no variable representing performance or body dimension such as physical work capacity and HV contributed to the "explaining" of the T wave findings.

The results imply that T wave changes of "organic" type in asymptomatic young men have an origin in changed vegetative tone for the majority of subjects and are not due to organic heart disease.

ACKNOWLEDGEMENTS

Supported by the Research Council of the Swedish Sport Federation and the Karolinska Institute.

REFERENCES

- Atterhög, J.-H., Furberg, B., Malmfors, V. & Kaijser, L.: Prevalence of ECG findings in 18-19 year old Swedish men. Scand J clin Lab Invest Accepted for publication.
- 2. Atterhög, J.-H., Ekelund, L.-G., Eriksson, G. & Ahlborg, B.: Significance of primary T wave aberrations in the electrocardiogram of asymptomatic young men. I Electrocardiographic data. Upsala J Med Sci, preceding article.
- Burch, G.E.: Significance of certain early changes in the T wave in coronary disease. JAMA 165: 1781, 1957.
- 4. Cohn, J.N.: Relationship of plasma volume changes to resistance and capacitance vessel effects of sympathomimetic amines and angiotension in man. Clin Sci 30: 267, 1966.

- Dembo, A.G.: Chronic heart strain in sportsmen. In Heart and Sport. (ed. V.L. Karpman. & G.M. Kukolevsky), p 427, Moscow, 1968.
- 6. Ekelund, L.-G.: Determination of blood volume. Scand J clin Lab Invest (Suppl.) 86: 53, 1965.
- 7. Goldman, M.J.: Normal variants in the electrocardiogram leading to cardiac invalidism. Am Heart J 59: 71, 1960.
- 8. Hanne-Paparo, N., Wendkos, M. & Brunner, D.: T wave abnormalities in the electrocardiograms of top-ranking athletes without demonstrable organic heart disease. Am Heart J 81: 743, 1971.
- 9. Hiss, R., Averill, K. & Lamb, L.: Electrocardiographic findings in 67,375 asymptomatic subjects. VIII Non-specific T wave changes. Am J Cardiol 6: 178, 1960.
- 10. Holmgren, A., Jonsson, B., Levander, M., Linderholm, H., Sjöstrand, T. & Ström, G.: Low physical working capacity in suspected heart cases due to inadequate adjustment of peripheral blood flow (Vasoregulatory Asthenia). Acta Med Scand 158: 413, 1957.
- 11. Kjellberg, S.R., Lönroth, H. & Rudhe, U.: The effects of various factors on the roentgenological determination of the cardiac volume. Acta Radiol 35: 413, 1951.
- 12. Levine, H.D.: Non-specificity of the electrocardiogram associated with coronary artery disease. Am J Med 15: 344, 1953.
- 13. Manning, G.: An electrocardiographic study of 17,000 fit, young, Royal Canadian Air Force aircrew applicants. Am J Cardiol 6:70, 1960.
- 14. Marriott, H.J.L.: Normal electrocardiographic variants simulating ischemic heart disease. JAMA 199: 103, 1967.
- 15. Prinzmetal, M., Goldman, A., Massumi, R.A., Rakita, L., Schwartz, L., Kennamer, R., Kuramoto, K. & Pipberger, H.: Clinical implications of errors in electrocardiographic interpretation. Heart disease of electrocardiographic origin. JAMA 161: 138, 1956.
- 16. Sendroy, J., Jr & Cecchini, L.: Determination of human body surface area from height and weight. J Appl Physiol 7: 1, 1954.
- 17. Sendroy, J., Jr & Collison, H.: Determination of human body volume from height and weight. J Appl Physiol 21: 167, 1966.
- 18. Simonson, E.: Differentiation between normal and abnormal in electrocardiography, C.V. Mosby Company, St. Louis (1961).
- 19. Sjöstrand, T.: Changes in the respiratory organs of workmen at an ore melting works. Acta Med Scand Suppl 196, 1947.
- 20. Sjöstrand, R.: Relationen zwischen Bau und Function des Kreislaufsystems und ihre Veränderungen unter pathologischer Bedingungen. Forum Cardiologicum 3, Boehringer & Soehne. Mannheim Waldhof, Deutschland (1961) (Ger.).
- 21. Wahlund, H.: Determination of physical work capacity. Acta Med Scand Suppl 215, 1948.
- 22. Weil, J.V. & Childsey, C.A.: Plasma volume expansion resulting from interference with adrenergic function in normal man. Circulation <u>37</u>: 54, 1968.
- 23. Astrand, P.-O. & Rodahl, K.: Textbook of work physiology. 2nd Ed., McGraw-Hill Book Company, New York (1977).

Accepted March 26, 1980.

Address for reprints:

J-H. Atterhög Department of Clinical Physiology Karolinska Sjukhuset S-104 01 Stockholm SWEDEN