Effect of positive acceleration (+g_z) on electrocardiogram of subjects with vasoregulatory abnormality

PURSHOTTAM K. KHANNA, V. BALASUBRAMANIAN, SATISH K. DHAM, KULDIP RAI, AND RAGHUNATH SINGH HOON

From Department of Cardiology, Army Hospital, Delhi Cantt-110010, India

ST-T wave changes in the electrocardiogram detected during routine examination and aggravated by erect posture, hyperventilation, and exercise in apparently healthy young individuals have been termed vasoregulatory abnormalities. No evidence of ischaemic heart disease has been found in such subjects.

Ten young healthy air crew with vasoregulatory abnormalities were subjected to maximal exercise on treadmill and procedure repeated after 120 mg propranolol daily for 3 days. After one week, they were subjected to a stress of positive acceleration (+g_z) in a human centrifuge at 2.5 g and 3.5 g for 15 seconds each at a constant rate of rise of 0.1 g/s and the electrocardiogram was monitored during and in the post-acceleration phase. The procedure was repeated after propranolol 120 mg daily for 3 days. The stress of positive acceleration resulted in pronounced prominence of P waves and inversion of T waves (as has been reported in normal subjects) with minimal ST depression in the electrocardiogram. ST segment depression during exercise, at heart rates corresponding to those achieved during peak centrifuge runs, was significantly more pronounced. The ST, P, and T wave changes were returned to normal after propranolol.

It is concluded that minimal ST segment depression after stress of positive acceleration as compared with conspicuous ST segment depression during exercise at corresponding heart rates, and their normalisation after propranolol, rules out ischaemia as an aetiological factor in subjects with vasoregulatory abnormalities.

It is not uncommon to find 'false positive' electrocardiographic ischaemic responses to exercise in certain subjects (Guzman and De la Cruz, 1972). Similar changes have been reported when the upright posture has been assumed and on hyperventilation (Kemp and Ellestad, 1968; McHenry *et al.*, 1970; Biberman *et al.*, 1971). They were attributed to autonomic influences (Nordenfelt, 1941) and labelled vasoregulatory abnormalities by Friesinger *et al.* (1972). When encountered among asymptomatic healthy individuals, the responsibility of the physician to give a definite diagnosis, especially to air crew, is tremendous. No evidence of ischaemic heart disease has been found in such subjects (Friesinger *et al.*, 1972). It was, therefore, decided to study the effect of the stress of positive acceleration forces and the resulting alterations in cardiovascular haemodynamics on the electrocardiogram of such subjects. This stress, in which the inertial vectors resulting from acceleration are directed parallel to the cardiovascular axis, has been

termed positive *g* or +g_z. Though cardiovascular reaction and electrocardiographic changes resulting from these accelerations in normal subjects have been reported earlier (Cohen and Brown, 1969a), the effect of +g_z acceleration on the electrocardiogram of subjects with vasoregulatory abnormalities has not been reported hitherto, and now forms the basis of this study.

Subjects and methods

Fifty-eight subjects out of a total 800 cases referred to this laboratory for cardiovascular evaluation were found to have vasoregulatory abnormalities as defined by Friesinger *et al.* (1972).

Of these 58, 10 were healthy male air crew, aged 22 to 36 years (mean 30 years), who volunteered to undergo further testing in a human centrifuge. All were found to have an abnormal resting or exercise electrocardiogram during routine medical check-up. They were evaluated clinically and biochemically. An electrocardiogram was recorded at rest and after

Received for publication 31 August 1976

a double Master's two-step exercise test. All of them showed ST-T changes in inferior leads, with or without involvement of lateral chest leads. They were then subjected to a multistage treadmill stress test. The details of the procedure for the treadmill stress test followed in this laboratory have been reported earlier (Balasubramanian *et al.*, 1975).

The electrocardiogram which monitors chest lead V5 was recorded in the supine and standing positions, after the Valsalva manoeuvre and hyperventilation, respectively. This was followed by maximal exercise on an Avionics motor driven treadmill by multistage stress as recommended by Bruce and Hornsten (1969). Heart rate and ST segment depression were monitored by an on-line digital Avionics computer model 2900. The test was repeated after propranolol 120 mg daily (40 mg $\times$ 8 hourly) for 3 days within 60 minutes of the last dose of the drug.

All the subjects were then familiarised with the environment of acceleration in the human centrifuge and were studied after a week of stopping propranolol. The centrifuge has its gondola mounted at a radius of 500 cm from the centre of rotation with the seat inclination 15° beyond 90°. They were then instrumented for electrocardiographic recording from the centrifuge, using a special floating type of electrode which does not come directly in contact with the skin. The contact is established through the conducting jelly. The leads were used as the input for triple electrocardiogram amplifiers model E-33 and recorded on a 6-channel jet recorder (Mingograph). The amplifier system permits selective switching of inputs. To ensure uniformity leads II, aVF, and V5 were recorded in all cases. After instrumentation of the subjects, the electrode connections were tested to ensure impedance values of less than 5000 ohms at the skin to electrode interface with a maximum difference of 1000 ohms between electrodes.

After obtaining a baseline record, the subjects

were exposed to the following centrifuge profiles: (a) peak 2.5 g and 3.5 g for 15 seconds each; (b) constant rate of rise 0.5 g/s; (c) rate of decay 0.1 g/s; and (d) monitoring was continued during the acceleration period and for 3 minutes thereafter. Each subject was observed through a television screen during the entire procedure.

Loss of peripheral vision (greyout), if any, was recorded by the subjective response of the individual to a fixed set of peripheral lights. The procedure was repeated after propranolol 120 mg (40 mg $\times$ 8 hourly) daily for 3 days. The graphs were analysed and the heart rate was determined from successive RR intervals. Changes in P, QRS, T, and ST segment contour were recorded.

Results

Twenty treadmill stress tests and 20 centrifuge runs were completed on the 10 subjects. The response of heart rate and ST segment, as obtained from lead V5, to various manoeuvres and stresses is summarised in Table 1. Fig. 1 shows the ST response to various manoeuvres and exercise before and after propranolol. Minimal ST depression, frank inversion of T waves, and prominence of P waves were noted during the centrifuge run. Fig. 2 shows the ST response, and the contour changes in P and T waves during +gz acceleration before and after propranolol. ST segment changes during +gz stress before propranolol were insignificant and, like P and T wave changes, were completely normalised by propranolol. No intraventricular conduction disturbance or changes in rhythm were noted during any of the centrifuge runs. ST segment response to +gz acceleration was significantly less pronounced ($P < 0.001$) as compared with ST segment response during exercise at corresponding heart rates. None of the subjects complained of fainting or manifested greyout at the gz profiles used.

Table 1 Heart rate and ST segment response in 10 subjects with vasoregulatory abnormality

	Supine	Standing	Valsalva	Hyper-ventilation	Maximal exercise	gz acceleration	
						2.5 g	3.5 g
Heart rate/min	B 85 (68-92) A 60 (54-73)	107 (82-120) 72 (60-87)	118 (92-144) 69 (54-85)	131 (82-173) 82 (64-104)	185 (149-202) 130 (102-159)	123 (110-140) 68 (60-83)	125 (100-155) 76 (60-100)
ST segment (mV)	B -0.7 (+0.0 to -1.2) A 0.2 (+0.8 to -0.7)	-1.3 (-0.2 to -2.0) 0.2 (+0.8 to -0.7)	-2.0 (-0.9 to -3.9) 0.5 (+0.8 to -1.0)	-2.6 (-0.9 to -4.2) 0.1 (+0.8 to -1.2)	-4.0 (-0.2 to -7.1) -1.4 (-0.3 to -1.5)	-0.8 (-0.3 to -1.0) 0.2 (+0.0 to 0.8)	-1.0 (-0.5 to -1.5) 0.3 (+0.1 to 0.6)

B, before propranolol; A, after propranolol; mV, millivolts; -, below isoelectric level; +, above isoelectric level. Figures in parentheses represent range.

Discussion

The 10 subjects reported in this study form a small subset of cases who are found to have an abnormal electrocardiogram, particularly on assuming an upright posture, after hyperventilation and the Valsalva manoeuvre. The electrocardiographic

changes are usually confined to leads II, III, aVF, and V4 to V6. In the majority of our cases the changes were initially aggravated by exercise but disappeared or improved as the exercise proceeded. They did not manifest any symptoms even at the height of maximal exercise. Unlike the findings of Friesinger *et al.* (1972), the ST-T changes were also

Fig. 1 Electrocardiogram of a subject with vasoregulatory abnormalities in the supine position, on standing, during hyperventilation, Valsalva manoeuvre, and exercise. Panel on the left shows electrocardiograms before the drug and the one on the right, after propranolol.

Fig. 2 Electrocardiogram of a subject with vasoregulatory abnormalities recorded before centrifuge run—basal (left panel), during centrifuge run at 3.5 g peak, without propranolol (central panel), and after propranolol (right panel).

aggravated by the Valsalva manoeuvre. Therefore, the changes attributed to increased sympathetic tone (Nordenfelt, 1941) may essentially be the result of an unstable autonomic nervous control (Holmgren *et al.*, 1959; Levander-Lindgren, 1964). It has not been possible to rule out categorically an ischaemic aetiology in these cases, but the clinical picture, electrocardiographic changes, normalisation of these under beta-blockade, and normal coronary arteriograms in some of them have been used as evidence against ischaemia (Friesinger *et al.*, 1972). Positive acceleration in normal individuals results in progressive flattening of the T wave going on to frank T inversion and distinct peaking of the P waves. These changes are confined to leads II, III, aVF, and V4 to V6. A distinct increase in the amplitude of the P waves was found in 78 per cent of 18 subjects exposed to 116 centrifuge runs (Cohen and Brown, 1969b) and 100 per cent of 20 subjects exposed to 40 centrifuge runs (Rai and Dham, 1973). T wave changes were, however, noted in 100 per cent and 80 per cent of the subjects in the two series, respectively. Our findings in subjects with vasoregulatory abnormalities are similar to those found in normal subjects by these workers. Electrocardiographic changes during positive acceleration in normal subjects have been attributed to myocardial ischaemia (Gauer, 1950; Gauer and Gienapp, 1950; Zuidema *et al.*, 1956; Gauer and Zuidema, 1961; Zuidema *et al.*, 1956), to descent of the diaphragm (Browne, 1958-1959), and related to heart rate (Bjurstedt *et al.*, 1959). Nordenfelt (1961) was, however, unable to reverse these changes produced by orthostasis with rate slowing carotid massage. Similar P and T wave changes have been produced by administration of sympathomimetic drugs and abolished by beta-blockers (Sjöstrand, 1950).

Positive acceleration results in pronounced haemodynamic alterations which have been reported earlier (Gauer, 1950; Gauer and Gienapp, 1950; Zuidema *et al.*, 1956; Gauer and Zuidema, 1961; Wood *et al.*, 1961). These consist of a fall of venous return to the heart, a decrease in left ventricular end-diastolic pressure, a diminution in the aortic root pressure and left ventricular dp/dt. There is a rise in heart rate, though bradycardia has also been reported (Peterson *et al.*, 1975). Calculated peripheral resistance begins to increase very early and remains high at the peak of *gz* acceleration. It is directly proportional to the level of *gz* acceleration. Most of these values have a tendency to overshoot before return to control levels in the post acceleration phase. Though coronary flow has not been estimated in humans during acceleration, animal experiments have shown that coronary flow increases at low *g* forces but decreases at higher *g*

forces (Shubrooks *et al.*, 1975). These workers have shown an increase in coronary flow and a decrease in coronary resistance in experimental animals subjected to positive acceleration, in spite of a fall in coronary perfusion pressure.

Our data have shown that electrocardiographic changes during positive *gz* acceleration in cases with vasoregulatory abnormality are not essentially different from normals. The P and T waves behave exactly as they do in normal subjects. The ST segment depression which is much more distinct after assumption of the upright posture, hyperventilation, and Valsalva manoeuvre, is much less so during the centrifuge run, and like P and T wave changes is completely normal with beta-blockade.

In addition to haemodynamic alterations produced by acceleration in the +*gz* axis, positive acceleration produces a reduction in the arterial pressure at head level. This in turn results in tachycardia and reflex vasoconstriction (Cohen and Brown, 1969a). Similar changes take place after orthostasis, and the similarity between the findings seen during positive acceleration and orthostasis is worth noting. Since the changes in both situations are normalised by beta-blockade, sympathetic stimulation is probably the common denominator in both the situations (Cohen and Brown, 1969a; Nordenfelt, 1965). Minimal ST deviations seen in our cases during positive acceleration and their normalisation with beta-blockade compared with much more pronounced changes during Valsalva and hyperventilation rules out the possibility of an ischaemic aetiology. The ST segment response is not rate related, because at a similar heart rate after exercise and +*gz* acceleration, the ST response is significantly different (Table 2). It may be argued that significantly less ST depression secondary to

Table 2 Maximal ST segment and heart rate response to +*gz* at 3.5 for 15 sec and ST segment response to treadmill exercise at corresponding heart rates in subjects with vasoregulatory abnormality

Case No.	HR/min	ST <i>gz</i>	STE
1	136	-0.9	-2.6
2	100	-0.5	-1.9
3	125	-1.1	-2.3
4	100	-0.8	-2.0
5	155	-1.0	-4.2
6	125	-1.4	-2.7
7	115	-0.6	-2.0
8	130	-0.8	-2.4
9	132	-1.4	-3.8
10	132	-1.85	-2.6

ST *gz*, ST segment response to 3.5 *g*; STE, ST segment response to exercise; -, below isoelectric level.

+gz acceleration is the result of improvement in coronary flow and fall in coronary resistance as seen in experimental animals (Shubrooks *et al.*, 1975). Beta-blockade, however, results in an increase in coronary vascular resistance (Jorgensen *et al.*, 1973) and would, therefore, result in aggravation of these changes. It seems highly likely that beta-blockade in combination with +gz acceleration would result in an increased incidence of fainting reactions but we were surprised to find that none of the subjects had any such reaction or decrease in tolerance to +gz in the form of greyouts. Cohen and Brown (1969a) exposed their subjects to progressively increasing acceleration profiles and greyouts were noted only beyond +4g. Administration of propranolol did not significantly affect the tolerance to +gz, though 3 out of 10 subjects stated that their greyouts seemed to progress more rapidly after propranolol administration. We did not expose our subjects to more than +3.5g and that may explain the absence of greyouts. As stated above, positive acceleration produces a reduction in the arterial pressure at head level and when combined with beta-blockade, a further fall in systemic arterial pressure may aggravate this. Beta-blockade is also known to increase the alpha-adrenergic activity of the venous capacitance vessels and may thus indirectly induce venoconstriction (Harris *et al.*, 1966). Propranolol, by increasing the diastolic filling time secondary to slowing of the heart, may neutralise the effect of the peripheral venous pooling effects of +gz acceleration. Propranolol also causes significant myocardial depression in man (Sjöstrand, 1950; Lucchesi, 1965; Paley *et al.*, 1965). It is, therefore, possible that the sum total of various haemodynamic alterations caused by +gz acceleration may be neutralised by propranolol. The fact that the ST changes are significantly less at a similar heart rate during +gz acceleration than during various manoeuvres and exercise, is evidence against ischaemia, as the basic aetiology in these cases with grossly abnormal ST segment. This supports the hypothesis put forward by Nordenfelt (1941) and Friesinger *et al.* (1972) that ST-T changes in such subjects are not caused by ischaemia and subjects with these abnormalities can undergo severe physical stress including flying aircraft involving +gz acceleration. Subjecting these asymptomatic individuals to invasive procedures of coronary arteriography may not be justified, but follow-up of these subjects is needed in order to find an answer to this question.

We are grateful to Air Cdre. S. P. Verma, V.S.M., for his technical and administrative help.

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- Requests for reprints to Lt. Col. Purshottam K. Khanna, Department of Cardiology, Army Hospital, Delhi Cantt-110010, India.