Study of Apexphonocardiogram and Carotid Pulsation during Short Term Hypoxia in a Decompression Chamber

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Seven normal subjects were studied to determine the effect of hypoxia at 15,000 ft for 45 minutes. Simultaneous recordings of ECG, apexphonocardiogram and external carotid pulse were taken at ground level, at 15 minutes intervals at altitude and on return to ground level. These were conducted to assess individual's cardiovascular responses during various stages of adjustment on exposure to short duration hypoxia.

A rise in heart rate and fall in LVET was noted immediately on ascent to 15,000 ft. By 30 minutes of stay the heart rate showed a tendency to fall from initial rise while PEP/LVET ratio increased. Heart rate increased again, and PEP/LVET ratio showed a fall by 45 minutes stay at altitude suggesting a cyclic pattern. Possible causes of these changes have been discussed. Further studies have been recommended to establish range of normalcy for assessment of borderline cases of ECG abnormalities in an otherwise healthy individual.

Short term hypoxia has often been used as a stress to determine cardiovascular performance in a subject. Reduction in arterial oxygen tension due to hypoxia initiates a number of reflex compensatory mechanisms, which are not found adequate in clinical or preclinical disease conditions. Though various invasive and non invasive techniques have been used to assess cardiac function under hypoxia, the latter is preferable as it does not alter the basic physiological conditions. Moreover, they are easily acceptable to the subject under study.

Cardiac activity during rest or under stress involves mechanical, electrical as well as acoustic energy. While ECG provides the electrophysiological status of the heart, other parameters must be simultaneously recorded to assess the associated mechanical functions. These can be easily undertaken by non invasive techniques like recording of phonocardiogram (PCG) and external carotid pulse (ECP). All these parameters taken together help in determining the systolic time intervals (STI). Measurement of STIs as introduced by Weissler et al [2,3] is currently being extensively applied for evaluation of left ventricular function.

Among service population and specially in aircrew, maintenance of optimal health and efficiency is essential to meet the various stresses normally encountered. Mild degree of hypoxia is commonly met with during flying and even by the ground crew when they are stationed at high altitude. Thus normal response to hypoxia and physiological compensation are essential for healthy subjects. Besides, preclinical cases of ischaemic heart disease have often been detected from ECG studies under stress of hypoxia (Levy's test) or by exercise (Master's test) or both (Khanna[5], Kirchhoff[6]).

Though ECG is used to determine the normality of integrated heart action and its response to anticipated or increased bodily need for greater minute volume flow of blood (Brooks[2]), it is by no means perfect. The specificity and sensitivity of ECG has still remained controversial (Black Burn[1], Chaterjee et al[3], Hoon[4].) Thus a false negative diagnosis places the aviator at a serious risk while false positive diagnosis is a costly error in terms of loss of a trained pilot for operational flying.

Simultaneous recording of ECG, PCG and ECP gives additional information about the mechanical activity of the heart besides the electrophysiological status, which should contribute in better assessment. With this in mind the present work was conducted to study the left ventricular function with the help of STIs in healthy normal subjects at ground level and under the stress of short term hypoxia at 15,000 ft simulated altitude and also to corroborate whether such findings could be compared with the range of normalcy under resting conditions found by other workers.

The study can be extended to determine borderline cases as abnormal, if they show results which are beyond the accepted range under hypoxia.

Material and Methods

STIs were estimated in seven young, healthy male volunteers in the age group 26-32 years (mean 28.6 yrs). All the measurements were recorded after breakfast, in hypobaric chamber. The subjects were not allowed to smoke and had ingested no drugs during the entire study. The nature of the procedure was explained to them. Most of the subjects had previous experience in decompression chamber. All subjects were examined medically and were found to be clinically normal and fit to undertake decompression chamber test. Recordings were made with subject in a supine position, with head slightly raised by one pillow, during quiet normal breathing.

Simultaneous tracings of the electrocardiograms, phonocardiograms and carotid artery pulsations were recorded using a Grass-model 5D-6 channel direct writing polygraph at a paper speed of 100mm/sec. (Fig. 1&2).

Test Profile :

Subject and observer were exposed to higher altitudes in a hypobaric chamber. After a preliminary ear clearance test, the chamber was raised at the rate 3000 ft/min and was leveled off at 15000 ft for 45 mi The descent was then made at a rate of 3000 ft/m to ground level. Simultaneous tracings of the electro cardiogram, phonocardiogram and external caroti pulse were recorded at :—

(a) ground level prior to ascent (base lin record),

(b) at altitude of 15,000 ft immediately on alt tude ascent and at 15 min intervals (0 min, 15 mi 30 min and 45 min),

(c) immediately on return to ground level an after 15 min.

Care was taken to ensure that subject was breathin normally during the recording.

Measuring Technique :

Electrocardiogram. Standard 12 lead ECG was recor ded at initial ground level and at the altitude o 15,000 ft after stay of 45 min at paper speed o 25 mm/sec. Standard lead II was recorded simulta neously with phonocardiogram and external carotic pulse.

Phonocardiogram. Sanborn Model 62 was used t record phonocardiogram. Output of this was connec ted to the plug in unit model 5 PIK of Grass polygraph for direct recording on paper. Microphone with funnel pick up was placed at the region of apex beat on the precordium with the help of suitable strap.

External carotid pulse. Right carotid arterial pulsa tions were recorded using funnel shaped plastic pick up with internal diameter of 3 cms, and held in posi tion with a suitable strap. It was connected to Gras volumetric pressure transducer PT 5A by a plasti tube less than 16 inches in length and polygraph uni 5 PIK.

Blood Pressure. Blood pressure was recorded manuall by the observer using anaeroid type of sphygmomanc meter. Blood pressure recording was done just prio to the multichannel recording of ECG, PCG and ECP.

Measurement of Parameters :

(a) Total electromechanical systole (QS2), th interval from the onset of ventricular depolarizatio to the first high frequency vibrations of the secon heart sound.

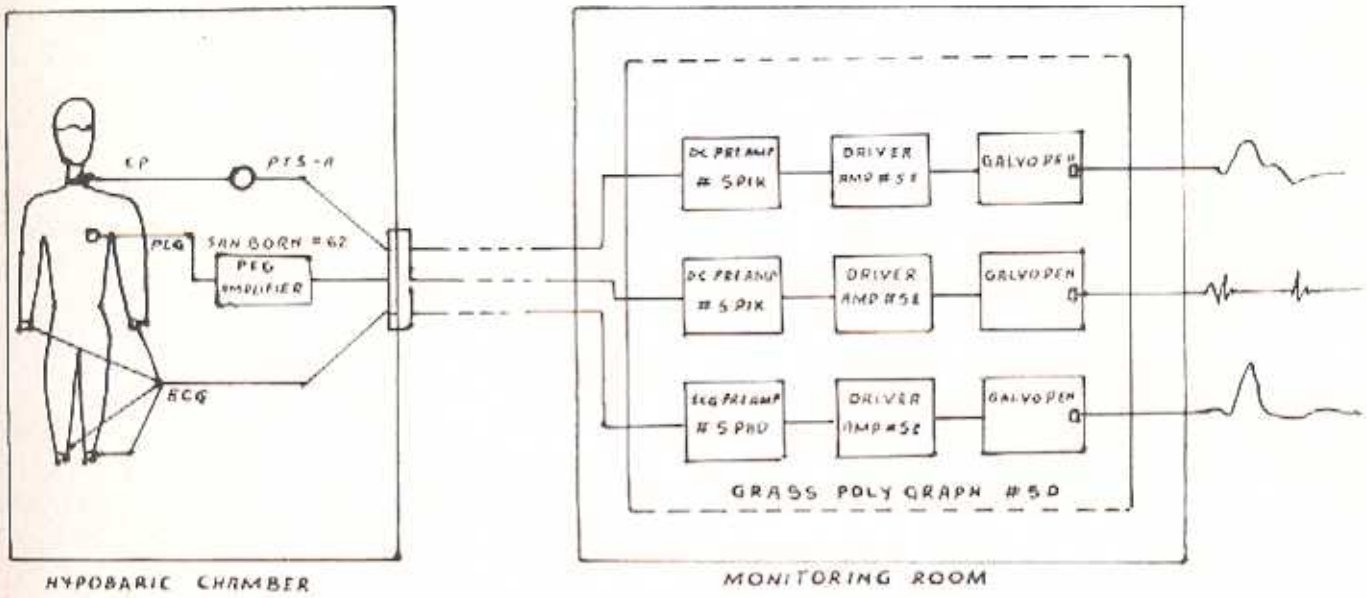

Fig. 1 — Block diagram of instrumentation and recording.

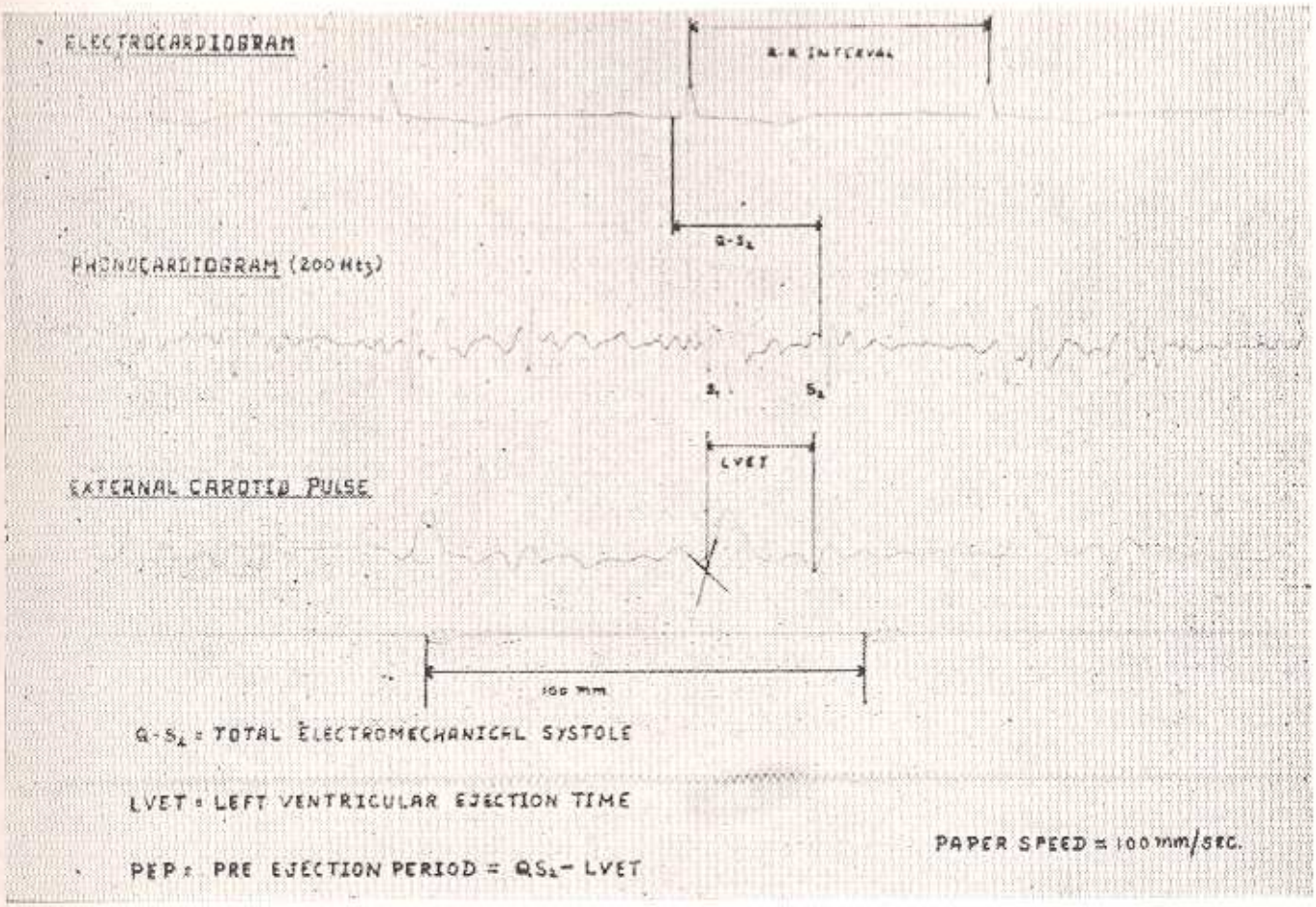

Fig. 2 — Showing simultaneous recordings.

(b) Left ventricular ejection time (LVET) measured from the onset of rapid upstroke of the carotid pulse to the nadir of the dicrotic notch.

(c) Pre-ejection period (PEP), calculated by the equation PEP = QS2 — LVET

The average of each of the above mentioned parameters was calculated from 10 consecutive cardiac cycles. Indices correcting the data for heart rate were calculated using the regression equation suggested by Lindquist et al[7] for resting condition

PEP Index (PEPI) = PEP + 0.44 HR
QS2 Index (QS2I) = QS2 + 1.85 HR
LVET Index (LVETI) = LVET + 1.42 HR

The PEP/LVET ratio was calculated from the appropriate values uncorrected for heart rate.

Significance of difference was evaluated using the paired **'t'** test.

Result and Discussion

Moderate hypoxia of short duration usually leads to physiological compensation accomplished by alterations in cardiovascular dynamics. The responses to reduction in arterial oxygen tension result from an interaction of its direct effects on the heart and vascular bed and its indirect effects mediated through nerves or humoral mechanisms or both. Hypoxia affects the circulatory system in a manner analogous to physical exertion and the physiological basis is similar.

In short duration moderate hypoxia the cardiac output is raised mostly by an increased heart rate. This is also associated with a fall in peripheral vascular resistance. However a differential vasoconstriction exists which helps in maintaining adequate blood supply to the vital organs. These changes are effected through the cardioaccelerator centre, peripheral chemoreceptor activity and sympathetic/parasympathetic interplay.

Lowered arterial oxygen tension, leading to hypocapnia and alkalosis prompt the adrenergic response by stimulating adrenal medulla, either directly or through sympathetic activity or both. Epinephrine and norepinephrine are present to some extent at all times in vascular system. Their levels in most cas reflect the degree of physiological reaction by t organism to a stimulus, being increased during stre and diminished in times of relative calm. Individual response with regard to blood pressure is influenc by the ratio of norepinephrine to epinephrine. Epin phrine produces vasodilatation in skeletal muscle heart and liver causing an overall decrease in tot peripheral resistance. The effects of catecholamine on the mechanical performance of the heart resul mainly from their direct action on individual musc fibre leading to increase in the rate as well as t force of contraction (positive chronotropic and inotr pic effects). As a result, rate of rise of intraventricul pressure is markedly raised, the systolic ejection rat are increased, the systolic ejection period is shorten and systolic emptying becomes more complete.

Rise in heart rate was noted immediately o ascent to 15,000 ft. (11 beats/min) which was foun statistically significant (Table I). By 30 minutes o stay the heart rate showed a tendency to fall fro initial rise though it returned to higher rate by 4 minutes. While QS2 and PEP showed no change LVET reduced at 0 min, 30 min and 45 minutes sta at altitude. PEP/LVET ratio increased significantl after 30 min stay at altitude. Systolic blood pressur was found raised in all subjects at all stages and thi rise was statistically significant. Diastolic blood press ure fell at 30 min and 45 min intervals. These change in blood pressure indicate epinephrine predominan response. The above changes in STIs which were within established normal limits (Table II) were mainly due to alteration in heart rate, because LVETI, PEPI and QS2I showed no significant changes during the exposure to hypoxia. Cardiac output was raised during this period as indicated by maintained mean arterial pressure, while associated with decreased peripheral resistance.

An inverse linear correlation has been demonstrated between PEP/LVET ratio and the ejection fraction (Stroke volume/end diastolic volume). After 30 minutes exposure to hypoxia PEP/LVET ratio has increased significantly ($P < 0.05$), thus showing a decrease in ejection fraction. At the same time LVET has also reduced significantly ($P < 0.01$) showing a decrease in preload or end diastolic volume. Hence it is possible that the fall in ejection fraction was due to

TABLE—I

Significance of Mean Difference in Parameters at 15,000 ft Altitude and on Return to Ground Level (GL) from Baseline (BL) Values

Parameters	BL to 0'	BL to 15'	BL to 30'	BL to 45'	BL to O'GL	BL to 15' GL
1. Heart Rate	10.9*	5.7	3.3	9.9**	−2.4	0.4
2. LVET	−10.1*	− 4.0	−10.6*	−13.7**	−8.2	−0.7
3. LVETI	5.4	3.4	−5.9	7.3	−13.6**	3.7
4. PEP	1.6	−1.0	4.4	−1.1	13.4**	0.8
5. PEPI	6.7	2.0	6.0	3.4	23.6	1.3
6. QS2	−8.3	−5.4	−5.6	−29.1	4.9	0.0
7. QS2I	11.7	7.9	1.1	3.1	0.4	0.9
8. PEP/LVET	3.3	18.0	30.7*	13.3	56.4***	5.7
9. Syst. Press.	4.6*	6.6***	8.6**	9.1**	1.4*	0.3
10. Dias. Press.	1.1	−2.3	−4.3	−1.4*	1.1	−1.1

* $P < 0.05$ ** $P < 0.01$ *** $P < 0.001$

TABLE—II

Systolic Time Intervals in 50 Normal Subjects

STIs	Range	Mean	Standard-deviation
Total electromechanical systole (QS2 interval)	330—440	382	±23
Left ventricular ejectiyn time	230—334	281	±21
Pre-ejection period	78—130	100	±13

Modified from Fabian et al (1972)

decreased stroke volume. The significant rise in heart rate ($P < 0.01$) between 30 and 45 minutes records, a significant fall in diastolic pressure between 30 and 45 minutes showing fall in peripheral resistance and a relative fall in PEP/LVET ratio can be interpreted as the compensatory response of cardiovascular system to the fall in stroke volume which had started after 30 minutes exposure to hypoxia.

A rebound decrease in heart rate was noted on immediate return to ground level. Though LVET and QS2 showed no appreciable change. PEP increased significantly. PEP/LVET ratio also increased significantly (< 0.01) immediately on return to ground level. However, when corrected for heart rate LVETI was and significantly decreased ($P < 0.01$) while PEPI and QS2I showed no changes. These changes can be explained on the basis of sudden reduction of sympathetic tone leading to decreased chronotropic and inotropic action. PEP is known to increase with reduced chronotropic action and LVET decreases with reduced inotropic action. All variations in the systolic time intervals and other parameters were within the established normal range (Table II) and the significant rise of PEP/LVET ratio immediately on return to ground might be due to sudden reduction in force and rate of contraction, as a result of decreased sympathetic tone. After 15 minutes of stay at ground level, LVET, PEP, PEP/LVET ratio, QS2, blood pressure and heart rate returned to prerun baseline values showing complete recovery to original state.

The study showed various stages of compensation in parameters like heart rate, blood pressure and systolic time intervals associated with exposure to short duration mild hypoxia. Though the range of variations of different parameters were found to be within the range of normalcy, some of the changes from baseline values were found significant. The heart rate increased significantly on exposure to hypoxia, showing a normal cardiovascular compensation. The rise in PEP/LVET ratio on return to ground level could be explained by the phase of readjustment when sympathetic drive was being withdrawn, on removal of hypoxia. This rise also leveled to prerun values after 15 minutes of stay at ground showing complete recovery. The alterations in parameters immediately on reaching 15,000 ft. were not being maintained throughout the stay at the altitude. The fall in LVET, rise in PEP/LVET ratio at the end of 30 minutes exposure lead to significant rise in heart rate by 45 minutes, recovering the LEVT and PEP/LVET ratio towards 0 minute values. Whether these changes during compensatory phase of mild hypoxia are cyclic or not can only be assessed by a longer stay at altitude beyond 45 minutes.

Conclusions and Recommendations

(a) Alterations in various parameters, observed in this study demonstrated the cardiovascular adjustments in a healthy subject when exposed to short duration mild hypoxia.

(b) The range of changes in systolic time intervals were found to be within established normal limits.

(c) The alterations in various parameters observed immediately on reaching altitude were not maintained throughout the stay of 45 minutes. A readjustment was seen between 30 and 45 minutes. Further study is recommended with a longer stay at the altitude to observe whether various parameters undergo cyclic change during the phase of compensation of exposure to mild hypoxia.

(d) As the present study was restricted to a small number of subjects, further studies on a larger number can be undertaken to determine the range of normalcy. This may help in identifying abnormal response in borderline cases to assess the electromechanical status of the heart by noninvasive technique.

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