

THE EFFECT OF HYPOXIC HYPOXIA AND LOW BAROMETRIC PRESSURE ON THE HUMAN ELECTROCARDIOGRAM (Vector analysis)

V. N. Alifanov

Central Clinical Hospital (Director I. M. Pishugin)

USSR Civilian Airways, Moscow

(Presented by Active Member AMN SSSR V. V. Parin)

Translated from *Byulleten' Éksperimental'noi Biologii i Meditsiny*, Vol. 50, No. 10, pp. 29-33, October, 1960

Original article submitted July 2, 1959

Most Russian authors who have studied the effect of low partial oxygen pressure on the human electrocardiogram (ECG) [1, 3-7, and others] have observed a reduction in the P and T waves. There is no one opinion agreed upon concerning the other waves. Similar results have been obtained by foreign authors [8, 10, 13, 14 and others]. G. V. Altukhov and V. B. Malkin [1] and other Russian authors have described the effect of low barometric pressure. They found that in a rarefied atmosphere corresponding to a height of 8,000-12,000 m, there is a rotation of the electrical axis of the heart (a change of the angle α) to the left due to the more horizontal position of the heart produced by a more elevated position of the diaphragm.

We have found accounts of only a few experiments in which vector methods of ECG analysis have been applied to traces recorded in hypoxia or at low barometric pressures. Thus, S. Tittel [14], and Gürtler, Poulsen, and Rasmussen [11] found that at low partial oxygen pressures, the integrative depolarization vector of the myocardium (AQRS) is reduced during inspiration, while maintaining its original direction; the repolarization vector (AT) is deviated to the left, but remains unchanged in amplitude; the "ventricular gradient" (G) changes both in direction and magnitude.

H. Becker-Freyseng and M. Stamberger [9] have described similar changes in the AT vector to be caused by exposure to reduced atmospheric pressure at a "height" of 11,500 m when breathing oxygen.

The vector method of ECG analysis is widely used in clinical practice. In attempting to reveal functional myocardial changes in human subjects caused by variations in the external factors already described, in addition to other methods we have used vector analysis of the electrocardiograms recorded at reduced atmospheric pressure.

METHOD

The experiments were carried out in a pressure chamber in which the pressure was reduced to 405 mm mercury,

corresponding to a height of 5,000 m, and the subjects breathed the air of the chamber for 30 min. In investigating the effect of the very lowest pressure, we reduced it to 195-170 mm mercury, corresponding to a height of 10,000-11,000 m; the subjects breathed oxygen for 20-25 min. The ECG was recorded for 15-20 min before "ascending," at the 3-5th and 20-25th min after the particular "height" had been reached, and 3-5 min after "coming down." The three standard leads were used in addition to chest leads CR₂, CR₄, and CR₅. Besides the normal ECG analysis, we also made measurements of the frontal vectors (vectors were constructed on the triple axis system).

In all, 120 investigations were made, 25 in hypoxia, and 95 at reduced pressure.

Healthy male subjects aged 20-30 and showing no ECG anomalies were used. Strictly controlled conditions of work, feeding, and rest were maintained.

RESULTS

At a "height" of 5,000 m, without additional oxygen, a moderate degree of hypoxic hypoxia was well tolerated, and the ECG changes were as follows: the P₁ wave was usually increased, and P₂₋₃ reduced; the precordial vector AP showed some tendency to rotate to the left, and it was usually reduced in magnitude; the P-Q interval and the Q and S waves showed changes which were not consistent. Quite frequently a left type of ECG was found, in which the R₁ wave was somewhat increased and R₂₋₃ reduced; the AQRS vector was displaced to the left. The T₁₋₃ waves were reduced, and the AT vector both reduced and as a rule displaced to the left. The "ventricular gradient" G changed in the same way as did AQRS.

Thus, there was a combined rotation of the precordial and ventricular vectors to the left without them becoming separated, which indicated a changed position of the ECG. Quite frequently there were signs of increased electrical activity of the left ventricle.

A confirmation of these findings was supplied from other axonometric measurements. The R/T ratio showed

no increase, and the R/S ratio changed, but not consistently.

In analyzing the ECG, we have thought it advantageous to introduce the R/P ratio (AQRS/AP), which describes the direction in space of the potentials in the myocardium of the auricles and ventricles. When the hypoxia is well tolerated, the index shows some increase on account of a comparatively small rotation of the AP vector to the left.

When the hypoxia is less well tolerated (Fig. 1), the P_{2-3} waves are relatively increased; the AP vector is quite frequently rotated to the right, and increased. The R_{1-3} waves are reduced, and the AQRS vector, particularly at the end of the experiment, is frequently rotated to the right. These changes indicate an increased load on the right heart, as is confirmed by several x-ray findings [2, 12]. The effects may be attributed to a hypertonia in the lesser circulation occurring in hypoxia. Changes in the T waves under these conditions are similar to those described above, but are frequently primary ("ischemic") in nature. The R/T ratio quite frequently increased, and the R/P (AQRS/AP) index increases on account of rotation of AP to the right and signs of overload of the right auricle, though it may remain unchanged or even decrease if in addition there is an increased load on the right ventricle (rotation of the AQRS vector to the right). There is frequently a reduction in the White index, which indirectly confirms the ideas presented above. The Fogel'son-Chernogorov systolic index showed no essential change.

When a human subject was exposed only to a reduced pressure, being brought to a "height" of 10,000 m and given oxygen, the ECG changes were considerably more complex than would have been expected from published reports. When the low barometric pressure was well tolerated, there was some tendency to a leftward rotation of the AP vector. When the condition of the subjects deteriorated, quite frequently the precordial vector was increased and rotated to the right; this effect was particularly well shown after "descent", when the masking influence of the elevated position of the diaphragm was removed.

When the low pressure is well tolerated a $Q_1 - S_3$ complex occurs which is well known to be typical of the elevated position of the diaphragm. When the pressure is not well tolerated, an $S_1 - Q_3$ complex appears which is typical of a rotation of the heart to the right about its longitudinal axis; however, in the conditions described, this result did not occur frequently, and in our opinion it indicates an increased load on the right heart.

When "high altitude" has no adverse effects, typically there was an increase in the R_1 wave and a reduction in the R_{2-3} waves, i.e., the AQRS vector rotates to the left. In such cases, the amplitude of the vector quite frequently increased, which gave reason to suppose that there was some association of the levogram with the relative increase of load on the right ventricle. When "high altitude" produced adverse effects, the voltage of

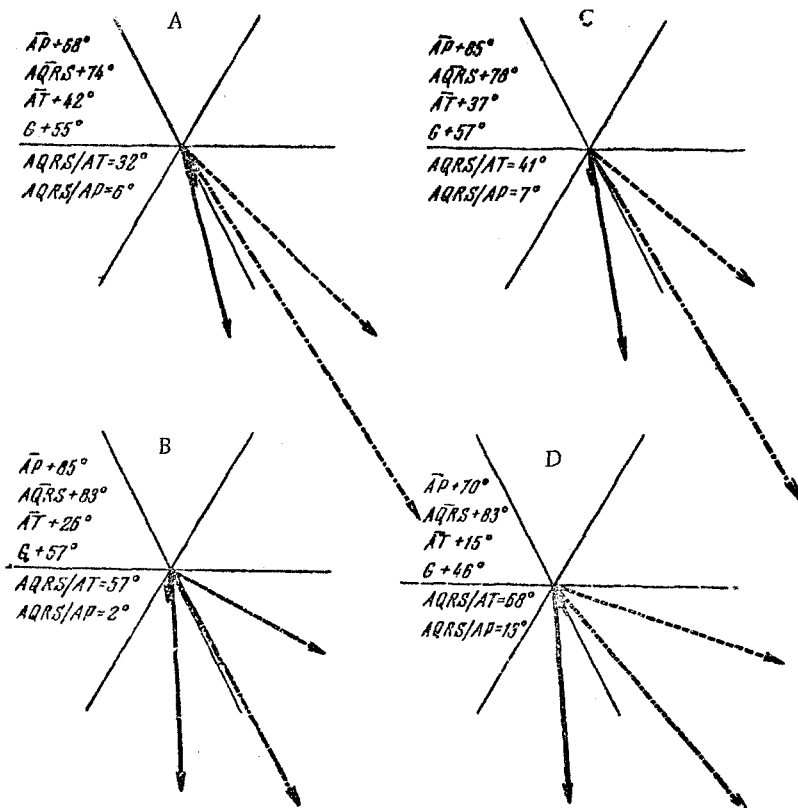


Fig. 1. Frontal vector cardiogram. Subject Ya-va, May 22, 1952. Hypoxia not well tolerated. A) Before "ascent"; B) 3 to 5 minutes at "high altitude"; C) 20 to 25 minutes at "high altitude"; D) after descent. AP - dotted line; AQRS - continuous arrow; AT - broken line; G - broken and dotted line.

the R_{1-3} waves was usually reduced; the AQRS vector quite frequently deviated to the right, particularly at the end of a period at "high altitude" which together with the right rotation of AP indicated that the predominant factor was the increased load on the right heart. Sometimes the ECG changes were typical of partial block of the right branch of the bundle of Hiss.

Changes in the T wave were less typical. The amplitude of the T_{1-3} wave was quite frequently reduced, the AT vector was deviated either to the left or to the right.

The "ventricular gradient" at "high altitude" usually showed a rotation to the left. When "altitude" was reduced, the continuation of the "ventricular gradient" indicated the primary nature of the altered T waves. When the reduced pressure was well tolerated, the R/T ratio was reduced and the R/P (AQRS/AP) ratio quite frequently increased on account of the relative smaller rotation of AP to the left. When the condition of the subject deteriorated, there was a larger number of cases where the AQRS and AT vectors were separated in the frontal plane by more

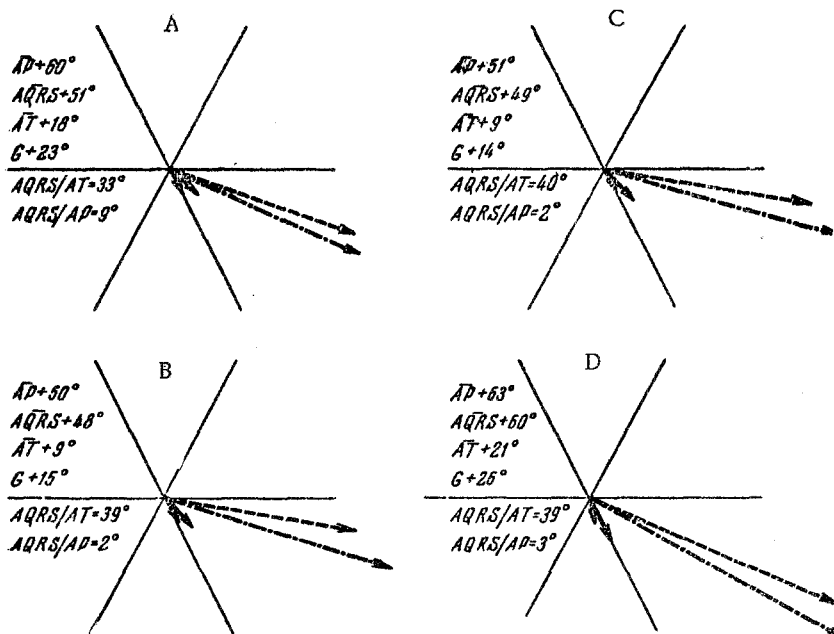


Fig. 2. Frontal vector cardiogram. Subject W-ov, February 12, 1952. Low pressure well tolerated. Indications as in Fig. 1.

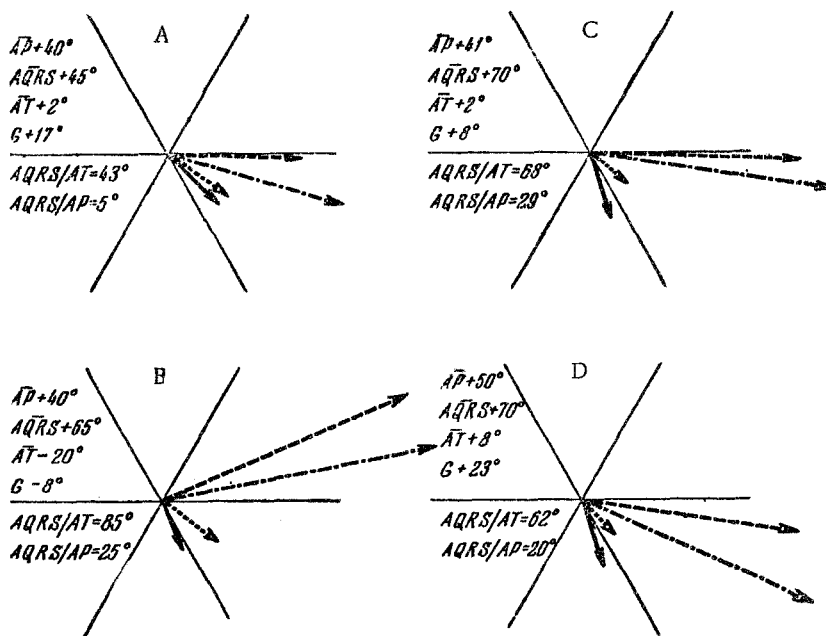


Fig. 3. Frontal vector cardiogram. Subject U-ov, February 14, 1952. Low tolerance to reduced pressure. Indications as in Fig. 1.

than 30°. The AQRS/ AP index was usually increased during the first few minutes at reduced pressure, but the change was due to the right rotation of AP combined with a preponderantly leftward rotation of AQRS. At the end of the period of "high altitude" the R/P ratio might be reduced on account of the additional right rotation of the AQRS vector.

The systolic index rose by 5-8% when the "altitude" was well tolerated; when the condition of the subject deteriorated, the index rose by 12-15%, indicating a disturbance of the ventricular myocardium (Figs. 2 and 3).

Changes of the ECG of the thoracic leads showed that when the reduced pressure was not well tolerated all the waves in lead CR₄ were reduced. In lead CR₂ the P, R, S, and T waves were increased; in lead CR₅ there was a moderate reduction in the S-T interval, i.e., there was a relative increase in the activity of the right heart.

It can be seen therefore that the appearance in the ECG of signs of overload of the right heart together with ischemia of one or other cardiac area (most frequently the left ventricle) seem to us to form a quite constant objective indication of a deterioration of condition following moderate degrees of hypoxia and reduced barometric pressure. Evidently, such changes may be ascribed to hindrance to the pulmonary circulation resulting from the hypertonia of the vessels of the lesser circulation induced by hypoxia, and also to abnormal interoceptive reflex influences brought about by an expansion of the hollow organs of the belly and some mechanical pressure on the lungs due to the elevation of the diaphragm ("high altitude meteorism").

SUMMARY

One hundred and twenty observations were made of the effect of a short stay in an altitude chamber on the ECG of healthy subjects at an effective "altitude" of 5,000 m while breathing either air, which induced hypoxia, or at an equivalent height of 10,000 m while breathing oxygen. In subjects who tolerated the hypoxia there was a combined swing to the left of the AP and AQRS-T vectors without any definite cardiac displacement. When the hypoxia was not well tolerated, after about 20-25 min at the reduced pressure there was a relative swing to the right of the cardiac vectors, indicating an increased load on the right heart. When no adverse effects resulted, the

shift of the cardiac vectors to the left was accompanied by signs of increased left ventricular activity. When the subject's condition deteriorated, an ECG of the S₁-Q₃ type developed, and the AP and often the AQRS vector swung to the right while the R/T and R/P indices rose, which also indicated an increased load on the right heart. Therefore an ECG indicating such an increased right heart load constitutes also an objective sign of a deterioration of the physical condition.

LITERATURE CITED

1. G. V. Altukhov and V. B. Malkin, *Klin. Med.*, 10, 45 (1952).
2. A. A. Gorodetskii, Transactions of the Conference on the Problem of Oxygen Insufficiency of the Organism [in Russian] (Kiev, 1949) p. 27.
3. M. A. Lyass, Transactions of the Central Military Academy of Electrolytic Apparatus, Civil Airfleet (1941) No. 1, p. 57.
4. O. P. Minut-Sorokhtina and N. V. Raeva, *Fiziol. Zhur.* 34, 2, 269 (1948).
5. V. G. Mirolubov and I. A. Chernogorov, *Klin. Med.*, 8, 1163 (1934).
6. N. S. Molchanov, Transactions of the Military Medical Academy [in Russian] (Leningrad, 1941) Vol. 31, p. 106.
7. V. A. Tsygankov, Transactions of the Military Medical Academy [in Russian] (Leningrad, 1940) Vol. 23, p. 77.
8. W. Adamaszek, *Luftfahrmedizin* Vol. 3, p. 125 (1939).
9. H. Becker-Freyseng and M. Stamberger, *Jahrbuch der wissenschaftlichen Gesellschaft für Luftfahrt* (Braunschweig, 1954) p. 184.
10. O. O. Benson, *J. Aviation Med.* Vol. 11, p. 67 (1940).
11. H. Gürtler, H. Poulsen, and K. N. Rasmussen, *Acta med. scandinav.* Vol. 150, p. 331 (1954).
12. H. Kottenhoff, *Luftfahrmedizin* Vol. 2, p. 194 (1938).
13. E. Opitz and O. Tilmann, *Luftfahrmedizin* Vol. 1, p. 153 (1936).
14. S. Tittel, *Luftfahrmedizin* Vol. 4, p. 328 (1940).