

THE EFFECT OF HYPERBARIC OXYGEN ON  
PERIPHERAL ARTERIAL CIRCULATION

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The effect of hyperbaric oxygen on peripheral circulation was examined on the lower limbs of healthy persons, indirectly, using bilateral impedance rheography. In the experimental group the rheogram was recorded in control conditions before and after exposure to hyperbaric atmosphere, during air breathing, and in different phases of oxygen breathing under a pressure of 2.8 bars. In the control group the rheogram was recorded in the same time intervals while the subjects breathed air under normal atmospheric pressure.

The results show that while the subject breathed hyperbaric oxygen the average pulse frequency, depending on the phase of examination, decreased in average by 13—15%, pulse volume by 6—8%, and volume perfusion by 17—19%. In the control group these parameters varied to a much lesser degree, irregularly and mainly in the opposite direction. The volume perfusion fall in the experimental group was primarily caused by bradycardia and to a lesser extent, by a decrease in pulse volume.

It remains to elucidate whether pulse volume is influenced by vasoconstriction, since the effect of vasoconstriction on brain circulation during hyperbaric oxygen breathing, has been confirmed by numerous researchers.

Oxygen is widely applied in medicine, diving activities and in aviation. In clinical application, oxygen is mostly used under normal pressure and primarily for treating cardiorespiratory insufficiency and shock. Hyperbaric oxygen is the most effective means of treating carbon monoxide intoxication, cyanide poisoning, gas gangrene and all forms of gas embolism, including decompression sickness. Further, oxygen is also used for the denitrogenation of pilots before flying at high altitudes. In underwater activities hyperbaric oxygen is used for some categories of military diving, for decompression and recompression treatment.

The effectiveness of hyperbaric oxygen in clinical application is based on the fact that with the increase of partial pressure of inhaled oxygen, the oxygen dissolved in the blood increases. A better oxygenation of tissues is achieved by this method, while, in decompression sickness and other forms of gas embolism, hyperbaric oxygen also accelerates inert gas elimination.

Together with beneficial, hyperbaric oxygen also produces negative effects. The negative effects on blood circulation are reflected in bradycardia and vasoconstriction. Although extensive data on the effects of hyperbaric oxygen on brain circulation have been published, similar studies about peripheral circulation have not been attempted.

Since hyperbaric oxygen is often used for treating peripheral arterial insufficiency, this study undertakes, by means of the indirect rheographic method, to establish whether hyperbaric oxygen affects lower limb perfusion and in what way.

#### SUBJECTS AND METHOD

Bilateral impedance rheogram was recorded with two groups of healthy persons. In the experimental group, the rheogram was recorded under normal pressure, during air breathing at 2.8 bars, at different stages of breathing hyperbaric oxygen under 2.8 bars and after return to normal pressure. In the control group, the rheogram was recorded only under normal atmospheric pressure, at the same time intervals as in the case of the experimental group exposed to hyperbaric conditions.

The experimental group, consisting of 22 healthy men of an average age of  $24.3 \pm 5.7$  years, had a completely normal rheogram. The control group, comprising 12 healthy men of an average age of  $22.3 \pm 5.0$  years, also had an absolutely normal rheogram.

Both groups were examined using the same method and at the same intervals by means of a bilateral impedance rheograph Beckman BR 160. The rheogram was recorded in a comfortable environment at a room temperature from 22 °C to 24 °C.

With the experimental group, the rheogram was recorded consecutively as follows:

- at atmospheric pressure during air breathing (1st phase)
- after five minutes of air breathing at 2.8 bars (2nd phase)
- after five minutes of oxygen breathing at 2.8 bars (3rd phase)
- at the end of a 10-minute period of oxygen breathing at 2.8 bars (4th phase)
- at 2.8 bars, two minutes after switching off from oxygen (5th phase)
- at atmospheric pressure 10, 20 and 30 minutes after switching off from oxygen (6th, 7th and 8th phase).

The rheogram-curve morphology, heart rate, pulse volume and volume perfusion were analysed.

#### RESULTS

The obtained data were classified according to the groups, phases and parameters.

### Morphology of rheographic curves

By comparing the morphological characteristics of rheographic curves during different stages of recording, no marked morphological changes were observed. Only a deepening of the incisure and gradual regular lowering of the amplitude during compression and air breathing at 2.8 bars and, particularly during oxygen breathing under hyperbaric conditions were evident. After switching to air and returning to normal pressure the amplitude gradually increased (Figure 1).

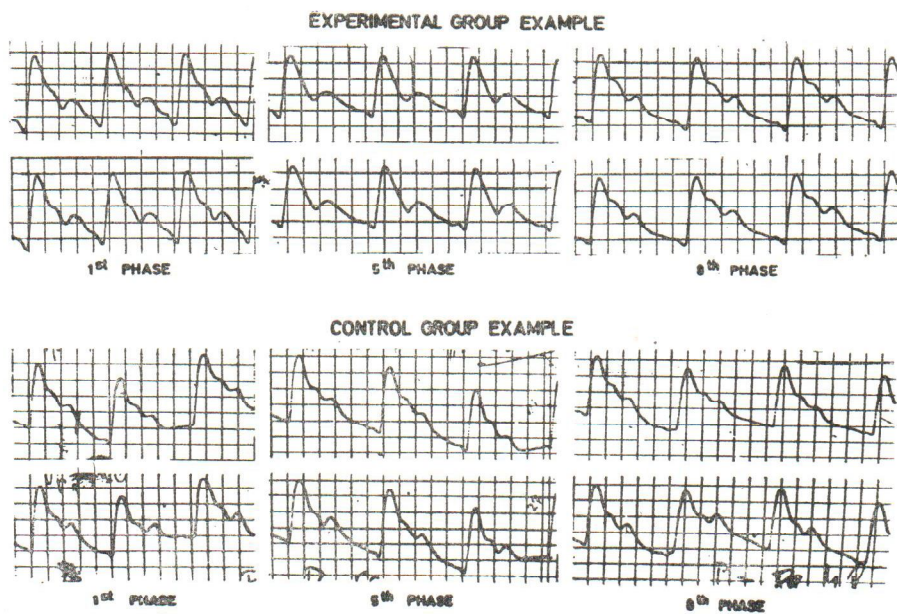


Fig. 1 — Morphological changes in the rheogram

### Pulse volume

The arithmetic means and standard deviations for the experimental and control group are shown in Figure 2. The average pulse volume during air breathing at 2.8 bars decreased by 5.0%. Under hyperbaric conditions and immediately after switching to oxygen, pulse volume further decreased at an average rate of 6.0—8.0%. The respective decrease was the most pronounced at the moment when the subject was switched

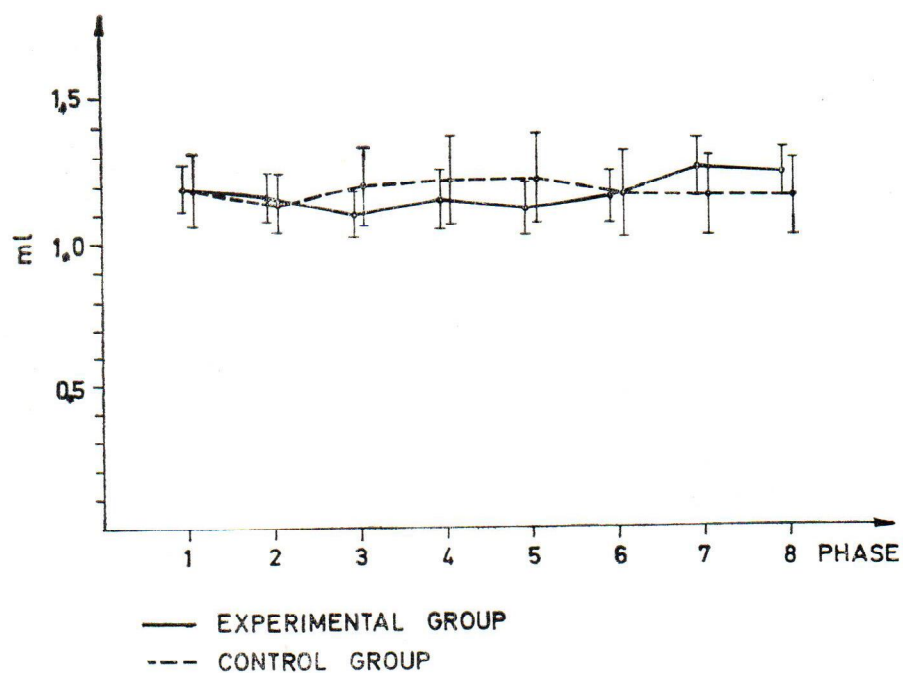


Fig. 2 — Pulse volume oscillation in the control and experimental group (both legs)

from air to oxygen. After switching from oxygen and returning to normal atmospheric conditions, the pulse volume slowly increased and after 20 minutes it was 2.0% higher than the average initial value. The pulse volume in the control group varied irregularly and not as much as in the experimental group.

#### *Volume perfusion*

Depending on the examination phase, this parameter showed a similar but stronger tendency to decrease than the pulse volume. Volume perfusion in the experimental and control group is shown in Figure 3. In the course of compressed air breathing at 2.8 bars, volume perfusion decreased by 8.0% during hyperbaric oxygen breathing. However, this

parameter was on the average by 17.0—19.0% lower than the initial value. After switching from oxygen to air and returning to atmospheric conditions, volume perfusion gradually increased. However, 30 minutes later, volume perfusion was 6.0% lower than the initial value.

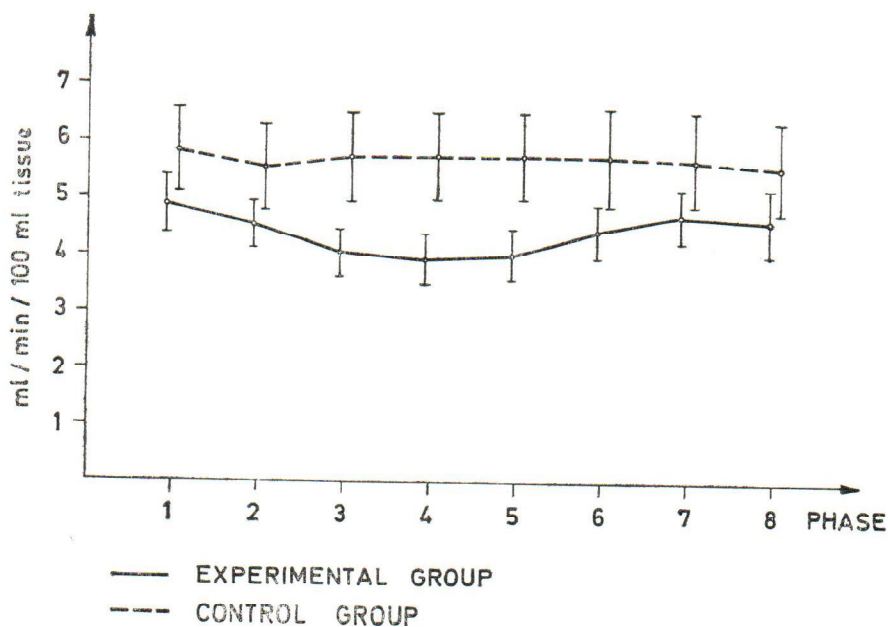


Fig. 3 — Volume perfusion in the control and experimental group (both legs)

In the control group volume perfusion remained practically unchanged, except during the last (8th) phase, when volume perfusion was 5.0% lower than the initial-reference value.

#### Heart rate.

In the experimental group the average heart rate regularly decreased to become more pronounced during hyperbaric oxygen breathing. After switching from oxygen to air and returning to atmospheric conditions, the heart rate gradually increased but did not reach the initial value, not even after 30 minutes (Figure 4). In the control group, the pulse rate practically did not change in relation to the control stage.

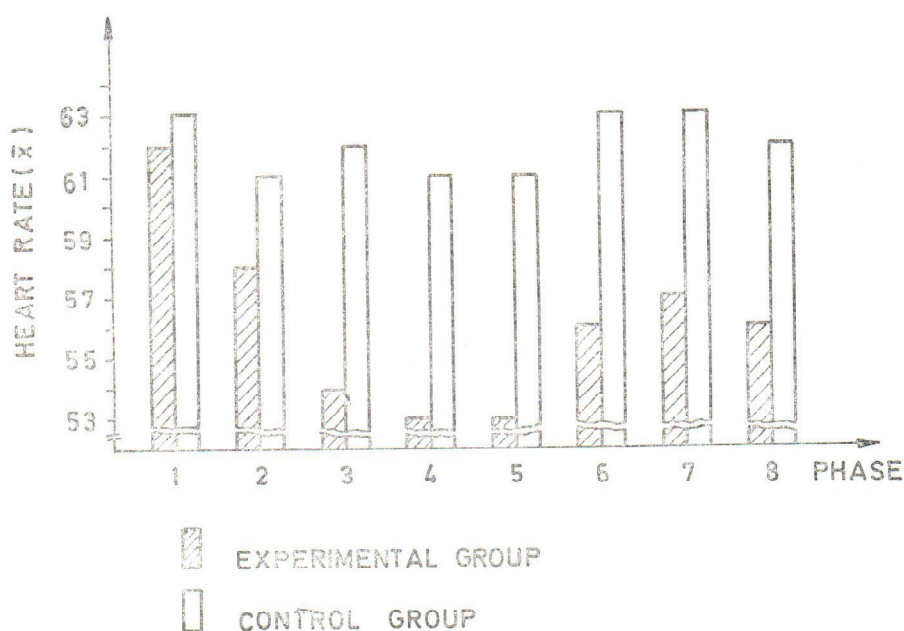


Fig. 4 — Heart rate oscillation in the control and experimental group

#### DISCUSSION

The effects of hyperbaric oxygen on the cardiorespiratory system, metabolism, gas exchange, the production and distribution of blood cells, endocrine and other organic functions are considerable (1—10).

Oxygen at normal and especially at increased pressure definitely and regularly leads to bradycardia (1, 2, 8, 11). Moreover, this phenomenon is also manifested in hyperbaric air and normoxic helium conditions under very high pressures (12—14). The origin of the phenomenon is still speculated about (1, 2, 12, 13, 14). Žironkin and co-workers report that minute volume decreased during exposure to hyperbaric oxygen (8). This phenomenon was also observed by Bean (1) and Anthony (15).

In 1968, Zinoveva recorded a drop in minute volume of 23—49% (16) in subjects exposed to oxygen under 4.0 bars. In 1965 Žironkin and co-workers observed a decrease in blood circulation during exposure to oxygen under pressure from 2.0 to 2.3 bars — 40% in humans and 20—30% in dogs (8). Similarly, Raevich and co-workers found that the

brain circulation slowed down by 51% when the subjects were breathing oxygen at 3.5 bars and by 22% when oxygen was at 2.0 bars (17). In 1940, *Cusick, Benson and Bothby* established that the average calibre of retinal arterioles and veins decreased by 24% and 28%, respectively (12).

According to *Doller and co-workers* the level of vasoconstriction varies in inverse proportion to the initial calibre of blood vessels, while it is directly proportional to the partial pressure of oxygen (18).

*Lambertsen and co-workers* found a significant drop (15%) in the cerebral blood flow during a 60-minute exposure to oxygen at normal pressure. This phenomenon, caused by vasoconstriction, was accentuated during oxygen breathing at 3.5 bars (19).

The data obtained by the rheographic method in our laboratory show that volume perfusion in under-knee segments decreased on the average by 17—19% while the subjects were exposed to oxygen at 2.8 bars. These results are similar, though lower in degree, to the results obtained for brain circulation (19, 5, 6, 17), and retinal blood vessels (12, 18). At the same time, in our research, a drop of pulse volume varied on the average by 6—8% and that of the heart rate by 13—15%. The data show that the decrease in volume perfusion is in the first place caused by bradycardia and in the second, to a lesser degree, by decrease in pulse volume. However, it is not possible to answer whether and to what degree vasoconstriction affects the fall in volume perfusion in under-knee segments during oxygen breathing.

Since we used an indirect rheographic method in our research, this research should be considered as an attempt to point out the problem of oxygen effects on peripheral circulation. As rheography is an indirect method for exploring the lower-limb circulation, the intensity of changes (fall of volume perfusion) should be taken approximately, especially since the triangulating of pulse wave-form is a subjective method.

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#### References

1. *Bean, J. W.*: *Physiol. Rev.*, 25 (1945) 2—69.
2. *Clarck, J. M. and Lambertsen C. J.*: *Pharmacol. Rev.*, 23 (1971) 38—133.
3. *Doležal V.*: *Rev. Med. Aeronaut.*, 233 (1969) 25—219.
4. *Moretti G. C.*: *Ann. Med. Nav.*, 70 (1965) 147—166.
5. *Lambertsen, C. J.*: *Physiological effects of oxygen, Underwater Physiology, NASNRC, Washington D. C., 1963, pp. 152—165.*

6. *Lambertsen, C. J.*: Handb. Physiol., 11 (1963) 1027—1046.
7. *Nichols C. W. and Lambertsen C. J.*: N. Engl. J. Med., 181 (1960) 25—30.
8. *Zironkin, A. G., Panin, A. F., Sorokin, P. A.*: Vlijanije povišenoga parcijalnogo davljenija kisloroda na organizam čeloveka i životnyh. Medicina, Leningrad 1965.
9. *Bradley, M. E., Anthoniesen, N. R. and Linawer, P. C.*: Respiratory and cardiac response in subjects breathing helium-oxygen mixture at pressure from sea level to 19.2 atmospheres. Underwater Physiology IV, Academic Press, New York 1971, pp. 325—357.
10. *Gošović, S.*: Utjecaj temperature okoline na neurotoksično djelovanje kisika. Ph. D. Thesis, Beograd 1975.
11. *Gošović, S. and Radović, A.*: Some cardiorespiratory effects of oxygen toxicity. Underwater Physiology VI, Bethesda 1979, p. 205.
12. *Cusick, P. L., Benson, O. O. Jr., and Bothby, W. M.*: Proc. Staff Meetings Mayo Clin., 15 (1940) 500—502.
13. *Egava, G., Gelend, R., Hamoud, R. E. and Lambertsen, C. J.*: Cardiac, electrical and mechanical functions. In: Work capability and physiological effects in HeO<sub>2</sub> excursion to pressure of 400 — 800 — 1200 — 1600 FWS. Predictive Studies IV, Philadelphia, Institute for Environmental Medicine, University of Pennsylvania, 1978, E13—1—E13—51.
14. *Gošović, S. and Radović, A.*: Frequency and regulation of heart rate during open sea saturation diving. Underwater Physiology VII, Bethesda 1981, p. 257.
15. *Anthony, A. J.*: Dtsch. Med. Wochenschr., 66 (1940) 482.
16. *Zinoveva, N. D.*: Vegetativnye pojavlenija kislorodnoj epilepsii čeloveka v načalnyh statia ejo formirovanija. Giperbaričeskaja epilepsija i narkoz, Nauka, Leningrad 1968, pp. 16—35.
17. *Raevich, M., Dickson, J., Clark, J., Hedden, M. and Lambertsen, C. J.*: Scand. J. Clin. Lab. Invest. Suppl., 102 (1968)
18. *Dollery, C. T., Hill, D. W., Mailer and Ramalho, P. S.*: Lancet, 2 (1964) 291.
19. *Lambertsen, C. J., Stroud, M. W., Gould, R. A., Kough, R. H., Ewinig, J. H. and Schmidt, C. J.*: J. Appl. Physiol., 6 (1953) 358—368.

#### Sažetak

### UTJECAJ HIPERBARIČNOG KISIKA NA PERIFERNU ARTERIJSKU CIRKULACIJU

Utjecaj hiperbaričnog kisika na perifernu cirkulaciju ispitivan je na potkoljениčnom segmentu zdravih osoba, posredno, pomoću bilateralne impedansne reografije. U eksperimentalnoj skupini registracija reograma vršena je u kontrolnim uvjetima — prije i poslije izlaganja hiperbaričnoj atmosferi, za vrijeme udisanja zraka i u različitim fazama udisanja hiperbaričnog kisika pod tlakom od 2,8 bara. U ispitanika kontrolne skupine periferni reogram registriran je u istim vremenskim razmacima dok su udisali zrak normalnog atmosferskog tlaka.

Ispitivanjem je ustanovljeno da se za vrijeme udisanja hiperbaričnog kisika prosječna frekvencija pulsa, ovisno o fazi ispitivanja, u prosjeku snižavala između 13% i 15% pulsni volumen je pao za 6% do 8% a volumna per-



fuzija za 17% do 19%. U kontrolnoj skupini ovi parametri oscilirali su mnogo manje, oscilacije su bile iregularne i uglavnom u suprotnom smjeru. Rezultati ispitivanja upućuju na to da je pad volumne perfuzije u eksperimentalnoj skupini u prvom redu izazvan bradikardijom, a samo u manjoj mjeri padom pulsog volumena. Kod ovog posljednjeg ne može se ocijeniti utjecaj vazokonstrikcije, koju su brojni istraživači ustanovili na cerebralnoj cirkulaciji za vrijeme udisanja hiperbaričnog kisika.

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