

HYPOBARIC HYPOXIA TRAINING AND THE RELEVANCE OF DETERMINING THE SO-CALLED RESERVE TIME IN POLISH PILOTS – PRELIMINARY REPORT

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Abstract: The paper summarizes the hitherto experience in methodology, safety measures, and effectiveness of training sessions conducted in the low-pressure chamber of the Military Institute of Aviation Medicine (WIML). These training sessions aim to familiarize flight personnel with the symptoms of reduced barometric pressure and acute hypoxic hypoxia (AHH) at a simulated altitude of 7,500 meters above sea level. Particular attention was given to psychomotor tests performed during the determination of the so-called reserve time (RT). In practice, RT is measured from the onset of AHH (disconnection of the trainee from supplementary oxygen and initiation of breathing ambient air in the low-pressure chamber) until arterial blood oxygen saturation reaches 70%, as measured by a fingertip pulse oximeter, or until the earlier occurrence of concerning symptoms of hypoxia. The need for verifying the training methodology, the type and assessment of psychomotor tests, and the individual RT values of pilots was justified in the context of aviation medicine. This includes evaluating the effectiveness of hypobaric hypoxia (HH) training and the relevance of clinical diagnostics in specific cases, ultimately contributing to flight safety. The paper highlights the necessity of addressing the questions posed in the conclusion.

Keywords: altitude hypoxia, reserve time, flight personnel training

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INTRODUCTION

Altitude hypoxia affects the function of all organs and systems [7]. Proper blood supply and oxygenation of the brain and heart are particularly critical for survival. Hypoxia significantly impairs the central nervous system, leading to cognitive dysfunction, short-term memory impairment, reduced attention span, altered perception and recognition, psychomotor slowing, decreased movement precision, prolonged reaction time, color vision disturbances, and a lowered sensory response threshold. Hypoxia symptoms vary among pilots, but one of the earliest signs, according to some sources [1], is impaired situational awareness. Other symptoms may include one or more of the following:

1. Behavioral changes, such as a sense of euphoria.
2. Poor coordination.
3. Discoloration at the fingernail beds (cyanosis).
4. Sweating.
5. Increased breathing rate, headache, sleepiness, or fatigue.
6. Loss or deterioration of vision.
7. Light-headedness or dizzy sensations and listlessness.
8. Tingling or warm sensations.

A pronounced symptom of hypoxia is central cyanosis (of the lips, tongue, and mucous membranes) as well as peripheral cyanosis (of the nail beds, nose tip, and auricles) [13]. However, due to the conditions under which the exposure is conducted (participants wearing oxygen masks), these changes cannot be observed. Although HH symptoms are common in most individuals, it is worth noting that among the 46 reported or potential symptoms described so far, those listed above are included [1], while some studies assess 24 of them [10], and others—such as in our previously applied research methodology and questionnaire—consider only 12 (with the first six being facial warmth, concentration and attention disturbances, visual impairment, drowsiness, fatigue, and sweating). The symptoms of HH identified by different authors do not fully align, differing not only in number but also in their sequence and frequency of occurrence. Altitude hypoxia symptoms may appear rapidly and distinctly (within seconds) or develop gradually and insidiously, depending on the type of oxygen system failure and the severity of hypoxia exposure. Moreover, hypoxia may occur without pronounced physiological reactions or noticeable symptoms [9]. Our observations in this regard require the completion of ongoing retrospective studies.

Altitude hypoxia triggers a sympathetic-circulatory reflex, leading to vasoconstriction, an increase in total peripheral resistance (TPR), and an elevated heart rate. The rise in TPR reduces blood and oxygen supply to most organs. As a result, oxygen consumption decreases, and tissues rely on anaerobic metabolic pathways. The oxygen thus conserved remains available for the heart and brain, where, due to local metabolic factors and stimulation of cholinergic fibers of the vagus nerve, vasodilation occurs. This process enhances coronary and cerebral blood flow, compensating for the reduced oxygen concentration in the blood. Additionally, hypoxic stimulation inhibits the activity of sympathetic fibers innervating arteriovenous anastomoses, leading to increased skin blood flow. A low-resistance blood flow occurs without significant oxygen loss. Sympathetic system activation may lead to cardiac arrhythmias through a mechanism of pathological automaticity. If the heart's response is dominated by the chemoreceptor reflex with vagus nerve activation, bradycardia occurs. In this case, it serves a protective role by reducing myocardial oxygen consumption. Additionally, the decrease in cardiac output prevents a rise in arterial blood pressure, which would otherwise result from a significant increase in total peripheral resistance (TPR). The body's oxygen supply also depends on the degree of hemoglobin oxygen saturation. As the partial pressure of oxygen in alveolar air progressively decreases, hemoglobin saturation declines in a linear manner [6,9]. The human body's response to acute hypoxia is complex in many aspects [3] and remains not fully understood. It has long been suggested that central and peripheral chemoreceptors play distinct roles in the body's response to hypoxia. New theories on hypoxia detection, the interdependence of peripheral and central chemoreceptors, and increased activity of central nervous system neurons involved in respiratory and sympathetic regulatory pathways [4] extend beyond the scope of this review. The same applies to the interaction between peripheral chemoreceptors and arterial baroreceptors that regulate sympathetic activity. The activation of the baroreceptor reflex inhibits the sympathetic response to hypoxia [11].

ACUTE HYPOXIC HYPOXIA TRAINING IN THE LOW-PRESSURE CHAMBER

For several decades, jet fighter and high-altitude transport aircraft pilots have undergone acute hypoxic hypoxia (AHH) training in the low-

pressure chamber (LPC) at the Military Institute of Aviation Medicine (WIML) under conditions simulating an altitude of 7,500 meters above sea level [5,6]. The primary goal of this training is to familiarize flight personnel with the body's physiological response to acute HH. It may occur in emergency situations involving a sudden oxygen deficiency at high altitudes due to factors such as oxygen system depressurization, human error in its operation, insufficient oxygen supply for the planned flight mission, and other unforeseen technical failures. The conditions at this altitude provide a strong physiological stimulus for acute hypoxia in humans.

To evaluate training effectiveness, the so-called "reserve time" (RT) is measured—the time elapsed from the onset of AHH to the loss of useful consciousness and motor function in the participant. In the late 1990s, the Military Institute of Aviation Medicine introduced a method for determining RT under normobaric conditions using a nitrogen-oxygen gas mixture containing 7.1-7.3% oxygen and a sealed GSz-6 helmet [6].

Once hypoxia exposure began, pilots performed a psychomotor test, which involved continuously subtracting one from 1,000. The RT assessment was terminated when any of the following indicators appeared: noticeable hand tremor or inability to write, sudden heart rate acceleration by 20 bpm or more, or (less frequently) a sudden deceleration, cardiac arrhythmia, a drop in blood oxygen saturation to 60%. Throughout the RT assessment, participants were continuously monitored using electrocardiography (ECG). Golec provided RT data for a Polish pilot population and proposed a seven-level scale for hypoxia resistance classification, ranging from insufficient to outstanding [6]. Notably, significant differences in hypoxia resistance among pilots were already observed, along with a markedly lower tolerance for hypobaric hypoxia (in the LPC) compared to normobaric hypoxia. The maximum RT differences between pilots exceeded 5 minutes and 80 seconds in hypobaric conditions and 6 minutes and 30 seconds when breathing a hypoxic gas mixture under normobaric conditions, and the average RT was 7 minutes and 20 seconds and 8 minutes and 10 seconds, respectively. The accuracy of the proposed RT rating scale, measured to the exact second (where an insufficient rating was defined as below 2 minutes and 40 seconds in hypobaria and below 4 minutes in normobaria), raises concerns about its reliability. It is important to note that these tests were conducted under normobaric conditions until participants reached

a blood oxygen saturation level of 60%. Therefore, they cannot be directly compared to results obtained by other researchers [1] or current exposure protocols. Most often, insufficient tolerance to hypoxic hypoxia was caused by an unhealthy lifestyle on the day preceding the training in the LPC or a poorly slept night.

Currently, according to procedures adopted in NATO countries, hypoxia training in the LPC is primarily of a schooling nature, and RT is not assessed in terms of overall health, even if it is short [12]. From the practice of doctors conducting hypoxia training in the LPC at the Military Institute of Aviation Medicine, it is known that the RT values measured in pilots are significantly lower than those given above and do not show such individual differences. This is likely due to the methodology of the studies/training, primarily the higher level of saturation, which constitutes the endpoint of the exposure (70% vs. 60%). Indications for terminating hypoxic hypoxia training in the previously used methodology, as mentioned earlier, included, among others, a drop in blood oxygen saturation to 60%, or difficulty in subtracting from 1,000, as noticed by the supervising physician or instructor. This was considered indisputable evidence of the negative impact of hypoxia on the body that the subject was experiencing. The error threshold in subtraction from 1,000 was not considered when assessing tolerance to hypoxia; the mere fact of it was sufficient. Currently, we are considering changing the test system to computer-assisted ones, similar to those described in the literature [10].

Considering the purely training nature of the conducted hypoxia training, but at the same time bearing in mind the negative impact of hypoxia on brain tissue and the limited ability to control the cardiovascular-respiratory parameters of individuals trained in the LPC, the blood oxygen saturation level has been reduced to a safe minimum. Due to the risk of decompression sickness in the pilots under study, the Australian Air Force conducts TUC tests in the LPC at 3,048 meters and 10% O₂, using appropriately varied mathematical and psychomotor tests; Singh et al., 2010 [10]. However, this seems to be excessive caution. In the last 20 years of conducted exposures to HH at the Military Institute of Aviation Medicine, no cases of altitude decompression sickness have been reported, which clearly indicates that the preventive protection – the nitrogen desaturation period from the body through 45 minutes of breathing pure oxygen – is completely sufficient. For many years at the Military Institute of Aviation Medicine,

the limit for measuring RT/terminating hypoxia training has been an endpoint saturation of 70%. This criterion is commonly used in NATO countries [8,9]. It is applied for safety reasons, as maintaining saturation below this value for a longer period could lead to serious damage to the nervous system.

However, from the available documentation of doctors conducting hypoxia training in the LPC, it follows that limiting hemoglobin oxygen saturation to the 70% threshold is not always sufficient to achieve the intended/symptomatic effect of HH that the trainee should experience and remember. On the other hand, lowering arterial blood saturation below this value could cause short-term memory disturbances, and even dizziness and loss of consciousness. This would contradict the educational value of such training. It is also important to emphasize that the fainting form of acute altitude sickness develops very insidiously and may not be preceded by any specific symptoms or only by short-lived sensations such as: seeing spots before the eyes, tinnitus, or a feeling of warmth [6]. The trainee/subject in the LPC loses their own assessment of poor tolerance to hypoxia, makes mistakes while completing the tests, and loss of consciousness, sometimes preceded by clonic hand tremors, occurs quickly. Therefore, it is mandatory to have a doctor/instructor safeguard the hypoxia training in the LPC. They must prevent loss of consciousness due to brain hypoxia, especially since, in such a situation, the sensations related to it are not registered/remembered by the trainee.

It is also postulated that studies should be conducted/determined to assess whether there is a significant temporal impact on brain function and cognitive performance immediately after exposure to acute HH in the LPC, once hemoglobin oxygen saturation has normalized [9]. The planned use and repetition of appropriate psychological tests, tasks on flight simulators, and psychomotor tests before the exposure to acute HH in the LPC and immediately after its completion will allow us to form our own opinion on this matter, based on the hypoxia training methodology we apply.

It should be remembered that resistance to hypoxic hypoxia varies individually and depends on many factors. It is likely also non-repeatable during subsequent trainings routinely conducted every 5 years for flying personnel in the LPC, as part of aviation medicine courses. One should not expect the effects of high-altitude hypoxia to be constant and equally strong. The degree of individual variability and the repeatability of indi-

vidual RT results in Polish pilots will be shown by ongoing retrospective studies.

The tolerance to hypoxic hypoxia in hypobaric conditions may, theoretically, depend on:

- the age of the subject,
- body mass and build,
- level of physical training,
- smoking,
- spirometry test results,
- the state of autonomic nervous system tone,
- blood morphology – values: RBC, Hb, Ht,
- concentrations of: Fe, glucose, thyroid hormones,
- water-electrolyte balance,
- current or recently experienced inflammatory/infectious conditions,
- body temperature on the day of the test,
- medications/drugs and substances (alcohol, narcotics, doping agents) taken,
- psychophysical exhaustion, sleep deprivation [1].

Studies on tolerance to hypoxia conducted at the Military Institute of Aviation Medicine

Studies conducted at the Military Institute of Aviation Medicine many years ago [5,6] showed that the best tolerance to high-altitude hypoxia/the longest “reserve time,” while breathing a gas mixture with 7.1-7.3% oxygen content, was observed in pilots aged 34-39 years (not younger), and the worst tolerance was found in pilots aged 40-45 years [6]. It should be noted that these studies did not involve the same pilots over time as they aged. Therefore, they do not allow conclusions to be drawn regarding the effect of age on tolerance to HH. Undoubtedly, individuals with an athletic body build, well physically trained, rested, after a good night's sleep, with no water-electrolyte disturbances, normal blood morphology, and normal concentrations of iron, thyroid hormones, glucose, sodium, and potassium tolerate high-altitude hypoxia better [1].

The psychomotor tests currently used at the Military Institute of Aviation Medicine are more difficult to solve than simply subtracting from 1000, but their evaluation takes place only after the training is completed. The tests currently in use are presented in the annexes to this publication.

It seems reasonable for these tests to be completed directly before exposure to hypoxia (before entering the LPC) and compared with the tests completed during the exposure. If the HH is sufficient to have a noticeable/symptomatic impact on the subject, the fact that the subject

has already become familiar with the psychomotor test before the hypoxic training may only help in its interpretation. This is because difficulties in completing the test will be more noticeable. The order in which the individual test tasks are completed should be determined and standardized. As can be seen from previous observations, some pilots leave more difficult tasks (e.g., finding the way out of a maze) for the end, while others try to solve them at the beginning and stop there, which complicates the interpretation of the results. It is important to note that in the first 1-2 minutes of reserve time, everyone is in undoubtedly better psychomotor condition than in the last one or two minutes.

Task forms should take into account the fact that brain function deteriorates rapidly as HH progresses. Therefore, the difficulty of subsequent psychomotor tasks should decrease, and their completion should be possible in fractions of a second. In this case, the mistakes made will make it easier to identify prodromal symptoms of impending loss of consciousness.

It should also be considered whether the questionnaire on subjective symptoms experienced during hypoxia exposure should be limited to those reported by the subjects themselves. The choice among the symptoms/experiences suggested in the questionnaire by the trainers may be suggestive and may not provide certainty that they were intense enough to be remembered, which is the primary goal of the training. If the subject did not require intervention from a doctor/instructor during the hypoxic training in the LPC and did not experience (did not report in the questionnaire) any subjective symptoms/experiences at the indicated reserve time, it should be assumed that the goal of the training was not achieved, and that the intensity of hypoxia was not strong enough in their case, regardless of the result of the completed psychomotor test. If the symptoms reported by the subjects in the questionnaires are not typical for HH, then the psychomotor test completed in the LPC, with numerous omissions (not errors), cannot serve as confirmation that the omissions were a result of hypoxia. A more convincing sign of brain hypoxia are the mistakes made in the psychomotor tests, rather than omissions in completing them. It is also extremely important that completing the test does not take the subject more time under normal atmospheric conditions (just before entering the LPC) than their RT. Otherwise, it would be impossible to differentiate whether the omissions in the test completed in the LPC were due to the effect

of altitude hypoxia or due to insufficient time for completion. In such cases, the results of the completed tests would be unconvincing, or even useless. The atypicality of the reported symptoms/experiences may have an emotional background and result from the fact that the subject knows when the instructor cuts off the oxygen supply, leaving them to breathe the chamber air, and they wait for the consequences of this fact. The planned remote switching of the breathing mixture from oxygen to air in the chamber (managed by the instructor) will create a situation where the subject will not know they are being exposed to hypoxia (at 7,500 m above sea level) and will have to recognize the symptoms of its effects themselves and immediately take appropriate action (switching the regulator to emergency oxygen – by changing the switch to the “EMERGENCY” setting).

Summing up the usefulness of task-based tests in hypobaric hypoxia, it can be stated that errors in counting from 1000, by one or more units, as well as clear hand tremors preventing writing, were/are a safe proof of altitude hypoxia's effects on the subject and at the same time the effectiveness of the hypoxic training. It should be noted, however, that this test does not account for the possible detrimental effect of hypoxia on color discrimination, which should be taken into consideration when using it. It seems that the currently used psychomotor tests require modification aimed at improving the credibility of the effects of hypobaric hypoxia on their results.

It also seems reasonable that an unacceptably low RT in the tested/trained person should be determined by aviation doctors, and its cause diagnosed. Pilots with such a value should be referred for clinical diagnostics, primarily for silent clinical conditions such as ischemic heart disease, arrhythmias, anemia, thyroid disorders, inflammatory conditions, and others that may potentially affect hypoxia tolerance. Such mandatory procedures would also discourage the possible simulation of poor hypoxia tolerance/desire to end training by those uninterested in the required training procedure, something that cannot be ruled out and is difficult to prove. However, the introduction of the above recommendations would require changes in the normative documents regulating the training of flying personnel. The currently applicable “Instruction for the Specialized Training of Flying Personnel of the Polish Armed Forces in Aviation Medicine” does not foresee such procedures.

The body's response to hypobaric hypoxia, as expressed in heart rate (HR), can vary. Due to

changes in altitude, there is a significant gradual increase in HR [14]. However, a sudden and significant acceleration of the heart rate, such as an increase of 20 or more beats per minute, requires the interruption of hypoxic training because it could indicate paroxysmal tachycardia. The instructor conducting the training in the LPC only has access to HR data and cannot register or interpret an electrocardiogram (ECG) recording. An especially interesting response would be a progressively or suddenly occurring, significant decrease in heart rate—a rare occurrence in aviation medicine—which is an absolute indication to interrupt the test and administer oxygen to the subject. If we were to assume that this is simply a reflexive response of the body to hypoxia, it raises the question of why it does not occur in the vast majority of subjects. In such cases, in-depth clinical diagnostics seem necessary, if only to exclude bradycardia caused by ischemia of the heart muscle or the sinoatrial node. Among non-invasive diagnostic tests, a normobaric hypoxic test using a hypoxia generator, combined with simultaneous electrocardiographic and echocardiographic monitoring, could be utilized.

It is likely that insufficient RTs in pilots, their very low resistance to altitude hypoxia, and/or abnormal heart rhythm changes associated with hypoxia, would find their explanation in more comprehensive clinical diagnostics. The results of these diagnostic tests could provide valuable insights into improving hypobaric hypoxia tolerance for flying personnel, mountaineers, mountain rescuers, HALO/HAHO parachutists, and others.

CONCLUSIONS

Mandatory training for military pilots on acute altitude hypoxia is conducted in a low-pressure chamber (KNC) under conditions corresponding to an altitude of 7,500 m above sea level. The goal of this training is to familiarize trainees with the symptoms of acute hypobaric hypoxia, which, if recognized early in actual flight conditions, could save their lives. These trainings have been repeated every five years. The experience gained from these trainings, along with an appropriately long reserve time (RT), allows for decisions to be made

regarding the need for oxygen equipment checks, the restoration of its efficiency, and the immediate descent to a height where the higher partial pressure of oxygen can prevent unconsciousness [2].

After many years of experience with the current method of hypoxic training in the LPC at the Military Institute of Aviation Medicine, the following questions arise:

1. Is the standard limitation of hemoglobin (Hb) oxygen saturation to 70% sufficient for all pilots to achieve the intended goal of hypobaric hypoxia training?
2. Do the psychomotor tests performed during RT measurements in the Institute's LPC, along with the post-training questionnaire regarding subjective symptoms, meet the requirements/expectations?
3. At what RT values and/or accompanying heart rate (HR) changes does a pilot require further clinical diagnostics for aviation-medical purposes?
4. Do five-year intervals between training sessions allow pilots to remember the subjective symptoms experienced during hypobaric hypoxia exposure? Are these symptoms identical to those reported in the questionnaires filled out during training?
5. Are the measured "reserve times," or more specifically, the time periods from the start of disconnection from oxygen and the initiation of breathing air in the LPC until achieving an arterial blood oxygen saturation level of 70%, or the earlier onset of disturbing hypoxia symptoms, repeatable during subsequent training sessions (as the pilot ages)?
6. Are the results of identical psychomotor tests repeatable during subsequent training sessions?
7. Has previous hypobaric hypoxia training in the LPC proven useful in actual flights preceding the current training?

Conducting surveys among the trained personnel will be necessary. Answers to these questions will help consolidate opinions, verify doubts contained in this work, and improve the methodology of hypoxic training in the well-understood interest of aviation medicine.

HYPOXIA WORKSHEET

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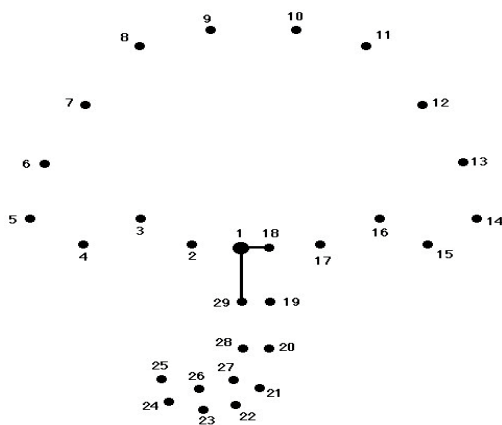
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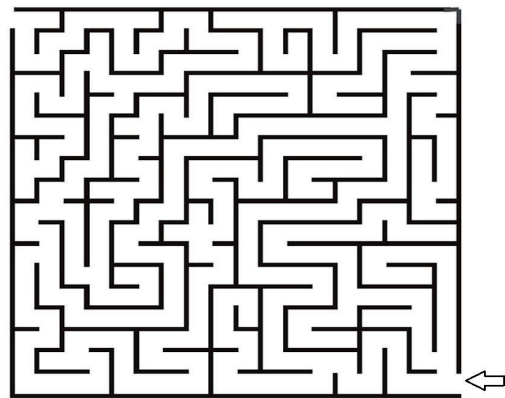
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Connect dots:



Find an exit from the labyrinth:



Expand the shortcut USA:

Write name of this month:

Place the word:

NIVATOIA -

Fulfill hours on clocks:



Do mathematical operations:

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RESERVE TIME:

HYPOXIA WORKSHEET

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Place the word: **NIVATOIA** -DO EXERCISES DUE TO INSTRUCTIONS: **+** 1; **■** 2; **●** 3; **▼** 4; **◆** 5

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Draw suitable figures:

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Do mathematical operations:

$$1 \times (\blacklozenge - \blacklozenge) =$$

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AUTHORS' DECLARATION:

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