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CONCERNING BAROTRAUMA OF THE LUNGS(U) FOREIGN
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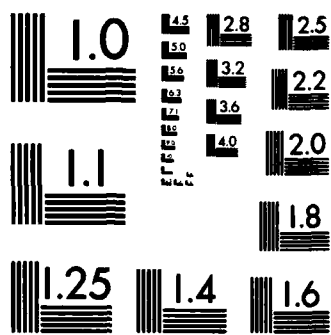
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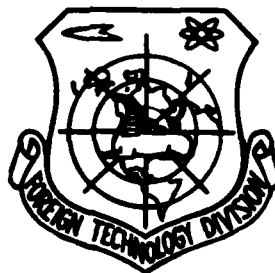


CONCERNING BAROTRAUMA OF THE LUNGS

by

G. Khristov

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CONCERNING BAROTRAUMA OF THE LUNGS

G. Khristov

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Abstract

→ The popularization and development of skin diving has led to the appearance of a new type of trauma of the lungs, caused by rapid change in air pressure in the lungs during sudden ascent of the diver with the breath held. Barotrauma of the lungs may be occasionally observed in anesthesiological practice as well. The paper discusses the etiopathogenesis, clinical manifestations and treatment of the complaint. The author has observed and described three cases of barotrauma of the lungs, one of which ended in death.

It is emphasized that effective treatment of pulmonary barotrauma is feasible only when a barochamber is available, large enough to accommodate the performance of resuscitation and, if necessary, surgical interventions. (Translation, Bulgaria).

The nature of barotrauma of the lungs with its pathophysiological foundations and clinical manifestations has become known only in the past 10-15 years, largely in connection with the rapid development and popularization of skin diving. At present, cases of barotrauma of the lungs are still very rare in Bulgaria, but in countries with well-developed skin diving recreation, like the USA, France, England and others, this is a frequent injury. Each year, the world cases of lung barotrauma exceed 100, while of this number roughly half end in death (Ehm, Seemann, Ulmer, Wandel, et al.). In view of the fact that the injury has been known only recently, there are few doctors, especially surgeons and resuscitation workers, familiar with the treatment of baro-

trauma, and often crude mistakes are made or no action is taken and valuable time is lost.

Actually, as far back as the XIX century there have been reports of cases of death of caisson workers by reason of fast transport to the surface. MacCleag, in 1933, described accidents of scuba divers emerging rapidly and losing consciousness at the surface. The author noted that this was due to shock, without being able to explain the causes. In 1934, Polak and Adams described a case where a diver quickly (2-3 s) emerged from a depth of 4.6 m, lost consciousness on reaching the surface, and died several minutes later.

In the following years, there were many publications and studies on the subject of barotrauma of the lungs (H. Keller, A. Bühlmann, W. Kirker, G. Lauchner, W. Bucklitsch, K. Pfeifer, K. Seemann, A. Wandel, Cl. Moslener, O. Ehm, G. Zaltsman, A. Pechatin, V. Surovikin, V. Fadeyev, D. Dorosiyev, P. Bogdanov, et al.). Today, the mechanism of occurrence of barotrauma of the lungs, the pathophysiology, the pathological-anatomical and clinical manifestations are fully known.

From physiology it is known that the human lung (the alveoli) can withstand a maximum inward-outward pressure from 120 to 200 g per cm^2 (i.e., approximately 1/4 of an atmosphere). If, for any reason, this pressure is exceeded, barotrauma results. This may occur, e.g., by sudden rise in pressure of anesthesiology apparatus when the safety valve is locked, damage to DP, Bird, Dreger and other type respirators, when using skin diving oxygen tank if a strong flow of oxygen is released into the breathing pouch, in event of damage to the automatic working of skin diving equipment with compressed air, as a result of continual supply of air with pressure too high, and so on. Barotrauma in such cases is ordinarily rare and minor, the victims usually experiencing only

slight pains in the chest and isolated bloody expectoration, but there can also be lethal outcome. Fadeyev and Surovikin describe several such barotrauma cases involving a skin diving oxygen tank with lethal outcome.¹

Severe barotrauma of the lungs has also been described in anesthesiology, albeit infrequently, usually ending in death. This occurs during explosion of the anesthesiology mixture (usually oxygen-ether) in the anesthesiology equipment or in the lungs of the patient (incorrect use of an electric cauter, explosion from electric spark while using ungrounded anesthesiology equipment, especially when the hoses and the tank are not made of antistatic material (e.g., rubber). However, severe barotrauma of the lungs is much more common in skin diving, when the diver surfaces quickly while holding his breath, i.e., when he does not exhale the air in his lungs while surfacing. This may happen when the diver does not know the rules for free or emergency surfacing or if, because of fear, panic under water, or distraction, he forgets or does not observe such rules, or in event of coughing or spasm of the vocal cords during the emergency surfacing, and so on.

Skin diving barotrauma is caused as follows: present skin diving equipment with compressed air is provided with an automatic respirator (Fig. 1), which continually furnishes the diver with air at the same pressure as the surroundings (i.e., the water). Thus, e.g., at a depth of 10 m the diver is breathing air under a pressure of 2 atm, at 20 m, 3 atm, at 30 m, 4 atm, and so on. According to the Boyle-Mariotte law, the product of the volume and pressure of the supplied gas at constant temperature is a constant ($P \cdot V = \text{const}$). Consequently, the volume of

¹In view of the frequent accidents and risks (barotrauma, oxygen intoxication, CO₂ narcosis, etc.), skin diving oxygen apparatus is prohibited for recreational purposes.

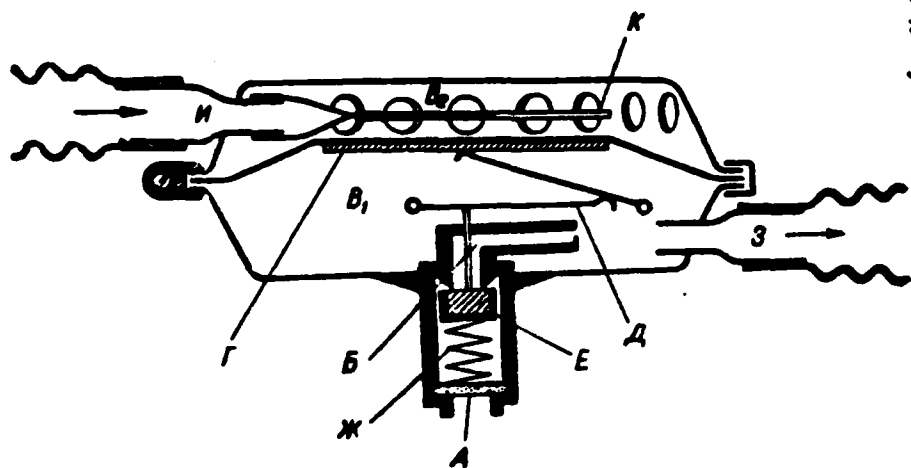


Fig. 1. Automatic respirator for skin diving equipment with compressed air: A - filter, B - nozzle, B₁ - dry chamber, B₂ - wet chamber (in communication with the surrounding water), Г - elastic membrane separating the two chambers, Д - lever system transmitting pressure from the valve-opening membrane, E - inhalation valve, Ж - valve spring, 3 - inhalation hose, H - exhalation hose, K - exhalation valve in form of a clip

the supplied gas changes in inverse proportion to the pressure: doubling the pressure reduces the volume by half, threefold increase in pressure reduces the volume a further 1/3, while a fourfold increase in pressure reduces it a further 1/4 (Fig. 2). Conversely, if the surrounding pressure decreases, the gas expands proportionately. From the above law and the illustration it is clear that the volume of the gas undergoes the greatest changes near the surface of the water. For example, when surfacing from a depth of 10 m the air inhaled at that depth under a pressure of 2 atm will expand twofold upon reaching the surface (1 atm), from 5 liters to 10 liters. Surfacing from 20 m, the air will expand threefold (from 5 to 15 liters), and so forth.

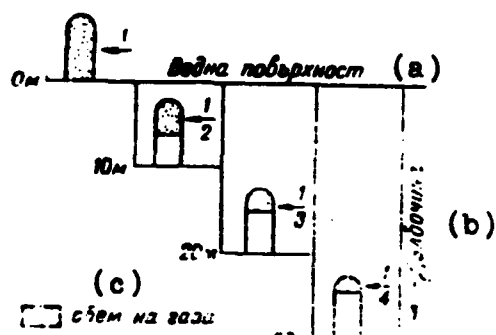


Fig. 2. Changes in gas volume as function of the pressure at various depths.

Key: (a) water surface;
(b) depth; (c) gas volume.

Whereas the air in the lungs under a pressure of 2 atm at a depth of 10 meters causes no damage to the alveoli, since the pressure is equal to the surroundings (the water forcing against all parts of the body), in the case of holding the breath while rising to the surface, where the surrounding pressure is equal to 1 atm, there is a pressure difference of 1 atm between outside and inside, at the expense of the air in the lungs. This pressure is more than enough to tear apart the alveoli, and for the expanding air to pass into the pulmonary capillaries, and from there to the left atrium, left chamber and the major blood circulation, producing a multitude of air embolisms in the brain, heart muscle, liver, kidneys, etc. Naturally, the lighter the air, the more it tends to rise to the brain. Unlike caisson sickness, where the bubbles are formed in the blood itself and the blood vessels, while the bubbles themselves are small, in the case of barotrauma of the lungs the air rushes into the vascular system from the lungs under large pressure, the bubbles are large, and they block a larger caliber of vessel. The injury is much more severe, and the outcome is more often fatal. The air, besides entering the blood vessels from the ruptured alveoli,

can also get under the pleura, into the mediastinum (pneumo-mediastinum) or even under the skin (subcutaneous emphysema). Frequently large emphysematous blisters rapidly build up in the lungs, and if the visceral pleura is ruptured there will be pneumothorax (unilateral or double).

In addition to this type of skin diving barotrauma, albeit much more rare, another type of barotrauma of the lungs is observed, occurring by the totally opposite mechanism, i.e., sharp drop in pressure in the lungs (BAROTRAUMA DUE TO RAREFACTION OR THE VALVE EFFECT). This occurs when, because of damage to the equipment or when it is emptied below 30 atm, the diver forcefully sucks air from the mouthpiece. This creates a valve effect in the lungs, as the alveolar capillaries and the vessels in the bronchial mucosa are engorged with blood, also assisted by the fact that the blood in the surrounding tissues and other organs is under elevated pressure, equal to the water pressure of the corresponding depth (A. Pechatin, V. Surovikin, A. Fadeyev, et al.). In such cases, the blood rushes to the lungs and reaches up to 15% of the volume of the total blood mass, instead of the normal 5%. The blood pressure in a. pulmonalis, which is normally around 25-30 mm Hg, rises to 180-200 mm Hg. When a pressure difference above 100-150 mm Hg occurs, the blood vessels burst and there is hemorrhaging from the lungs. This is usually a mild form of barotrauma, accompanied by 1-2 days of bloody expectoration and pain in the chest, which go away by themselves. But if the air pressure in the lungs increases excessively, immediately or soon after the rupturing of the pulmonary capillaries (coughing fit, deblocking of the automatic skin diving equipment with sudden supply of air at a pressure greater than the surroundings), the air may pass back from the alveoli through the ruptured pulmonary capillaries into the heart and the major blood circulation, producing air embolisms which, albeit infrequently, may be equally deadly.

THE CLINICAL MANIFESTATIONS OF BAROTRAUMA PRODUCED BY ELEVATED PRESSURE are rather dramatic and always occur after the diver has surfaced. In the milder cases, there is acute pain and pangs in the chest cavity and behind the sternum, shortness of breath, coughing with frothy bloody expectoration, dizziness, disturbed balance, visual problems, shaking of the limbs, itching, paralysis, disorders in the pelvic reservoirs (most often, incontinence), etc. There is cyanosis, most pronounced in the face, neck and upper limbs with swelling of the cervical veins (pressure on the v. cava cranialis from the pneumomediastinum), facial edema, conjunctivitis, etc. The blood pressure drops and the pulse races, usually because of decrease in the diastolic filling of the heart as a result of pressure on the empty veins by the pneumomediastinum, as well as resulting from embolisms in the medulla oblongata and the circulatory center.

Percussion reveals a tympanic tone in areas with deadening (hemorrhage). Auscultation reveals moist crepitant, fine and medium rale, occasionally even gurgling breathing. In severe cases with massive cranial embolism, there is always loss of consciousness, pronounced cyanosis, deep snoring respiration or most often a sudden stopping of the respiration and heart activity with onset of clinical death, quickly becoming definitive unless quick measures are taken.

The only safe method of TREATMENT OF BAROTRAUMA OF THE LUNGS, especially when cranial embolism is present, is to place the victim in a barochamber and rapidly recompress.

In the milder cases of barotrauma, when the victim is fully conscious and may take care of himself, he gets into the barochamber by himself. If there is frothy bloody expectoration, hemostatics are prescribed (calcium, vitamin C, kladen, reptilaza, strifnon, etc.); if there is strong painful coughing, drops of

codeine or dionine are given, which requires much care in event of expectoration of rather large quantities of blood, to prevent its retention in the lungs.

If no barochamber is available¹ on the scene of the accident, and if the diver is well enough to achieve recompression, he must return instantly with new equipment and one or two assistants again under water, to an even greater depth than that from which he surfaced. After remaining a certain time at the required depth, it is necessary to surface slowly, in the company of the assistants, observing the decompression chart with appropriate stopping at designated depths (decompression thresholds).

Case 1. K. M., 27 years old, engineer, nationality Polish. In the summer of 1966 in the waters off Stomopolu, during a joint dive of our skin diving group and a Polish group to a depth of 45 meters, in the middle of the distance (around 20 meters) suddenly K. M. bolted to the surface without signaling. The leader of the group noticed this and, foreseeing danger of barotrauma, gave pursuit at full speed, reaching him at around 10 m below the surface, and attempted to restrain him. K. M. calmed down for a minute and took air from the offered mouthpiece of the leader, but at the same time also took in a little water. Because of this, K. M. again panicked and bolted to the surface. Despite the efforts of the group leader and the divers rushing to help, K. M. managed to surface; even so, while struggling with the other divers, he released a portion of his air.

Upon reaching the surface, K. M. complained of difficulty breathing and pain in the chest. Coughing eliminated a little frothlike bloody expectoration. But his general condition was

¹For lack of a barochamber, the navy may employ a compartment of a submarine, where the pressure may be raised as needed.

satisfactory. Since there was no barochamber on board, the victim was again taken under to a depth of 40 m with new and safe equipment and two assistant physician-divers. After a 10 min stay at this depth, gradually and observing the stages of decompression (approximately 15 min) the victim was returned to the surface (total stay 25 min).

Aboard ship, the victim was placed with his body at an inclination to his head. He was given calcium with vitamin C and kladen, as well as drops of codeine. At the skin diver enclosure, K. M. remained in bed an additional three days, continuing to produce a little bloody expectoration. Chlornitro-mycin was given as a caution. The symptoms of barotrauma disappeared gradually in around 5-6 days.

All victims of barotrauma must be regarded as serious cases, regardless of the patient's well-being. Often a victim, feeling well and having almost no complaints, or complaining only of slight pain behind the sternum and slight bloody expectoration, may suddenly lose consciousness and expire.

As soon as the victim emerges from the water, be it only the slightest symptoms of barotrauma or only the suspicion thereof, he should quickly assume a head downward position until transferred to a barochamber. If there is no barochamber at the site of the accident and if it is not possible to return under water with an assistant, the victim should be transported at maximum speed to the nearest barochamber and be treated there. In addition to placing the victim with the head in a low position, several authors (Dorosiyeu, Fadeyev, and others) also recommend it be turned to the left side. Besides protecting the victim against new cranial embolisms, these positions also help facilitate the elimination of blood and froth from the respiratory pathways and simultaneously protect against aspiration of the

stomach contents in event of vomiting. In event of loss of consciousness, the sideways position of the victim also protects against blocking of the air pathways as a result of the tongue dropping down onto the rima glottidis.

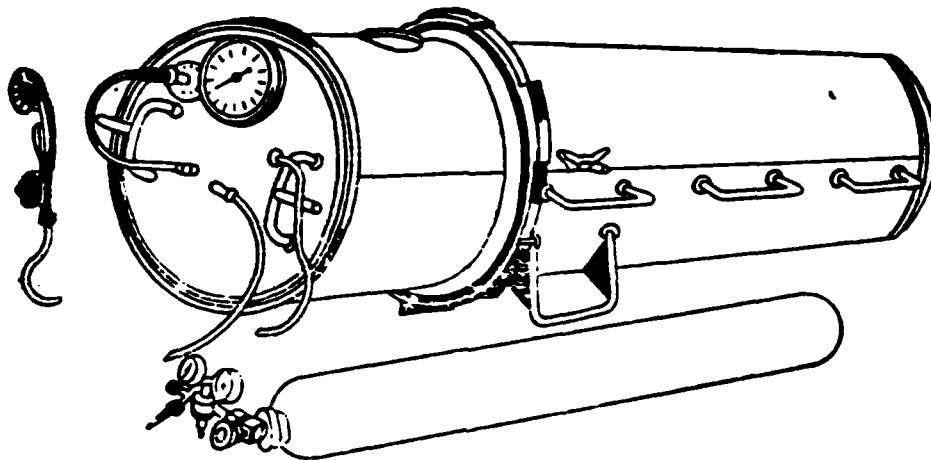


Fig. 3. Small one-man barochamber.

In severe cases of lung barotrauma, large barometers are to be preferred, admitting both the victim and 1-2 doctors, nurses, etc. to carry out the appropriate manipulations - artificial respiration, heart massage, if necessary surgical intervention to correct pneumothorax, suturing of ruptured lungs, direct heart massage and so on.

In the barochamber, the victim is quickly put under pressure to dissolve the air bubbles in the blood vessels, followed by slow decompression in the appropriate procedure (Table 1).

The choice of the compression and decompression regime is suited to the condition of the patient and the time of disappearance of symptoms during the pressure increase. Treatment of barotrauma is done most often by regime III (see table), but in exceptional cases it is also possible to use regime II, if all

Table 1. Therapeutic recompression for barotrauma of the lungs.

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that is available is a small decompression chamber, where the pressure can only be increased to 5 atmospheres (60 m). If, upon raising the pressure to 7 atm, the symptoms of barotrauma do not disappear after 15 minutes, the pressure is increased to 9 atm. The patient remains at that pressure for 20 min, and then the recompression is done by regime IV. If, in the process of lowering the pressure from 7 or 9 atm the symptoms of gas embolism recur, the pressure must be increased to 10 atm and the subsequent decompression done according to regime V.

At the halt with pressure in the chamber under 18 m H₂O (see table), the patient may be given pure oxygen to breathe. The oxygen is given at the end of the halt for 20-30 min. In this case, the halt is shortened by a factor 1.5.

The lowering of the pressure between halts is done for 1 min, this time being added into the total decompression time. If the victim is unconscious, it is advisable to perform a precautionary paracentesis of the eardrums, preferably by persons inside the barochamber, in order to save valuable time.

Successful recompression in serious cases of barotrauma may only be expected with a barochamber available at the scene.

If the breathing of the victim has stopped or is insufficient, an intubation is performed. However, the aeration must be done with minimum pressure (8-10 mm Hg), to prevent forcing additional air into the circulatory system. An intermittent positive-negative respiration is advisable, if the appropriate equipment is available (DP, respirators of Medy, Dreger, Bird and others), as the negative pressure may be increased to 10-15 mm Hg. It is best to use oxygen for the respiration. If a valve pneumothorax is present, intubation and respiration must be done with caution, due to the danger of increasing the

pneumothorax. Preliminary puncturing of the pneumothorax with a broad-tipped needle or emplacement of a temporary aspiration catheter across a thoracocentesis.

If need be, the patient is administered analeptics and transfusions of blood replacement fluids, blood, hemostatics (if required), vitamins, etc.

If the heart activity stops, indirect heart massage is performed. In most of the serious cases, owing to the presence of severe mediastinal emphysema and so forth, a thoracotomy is done to open the mediastinal pleura, thereby reducing the tension in the mediastinum and venous congestion, and direct heart massage is performed. After the situation is in control and the circulation is stabilized, the large emphysematous blisters are resected or sutured, and a drain for permanent aspiration is placed in the pharyngeal cavity. The mediastinal pleura is left open or loosely stitched so as to evacuate the air from the mediastinum and the interstitium of the lungs. This also helps eliminate more quickly the cervical and subcutaneous emphysema. The treatment of subcutaneous emphysema with lenslike cutting of the skin is senseless, and best left untried.

None of the aforementioned manipulations and surgical interventions should be done at the expense of quick delivery of the victim to the barochamber. On the contrary, they should be done in the barochamber itself, or in an emergency, if there is no barochamber at the site of the accident, during the transport of the victim to the barochamber.

Case 2. K. N., 17 years old, student. In August 1968, 5 miles southeast of the city of Pomorie, because of damage in his automatic respirator, vacationing K. N. was forced to surface from a depth of 12-13 m in the company of the instructor, both

taking turns breathing from the same equipment. For unknown reasons (most likely, panic, coughing fit or laryngospasm), K. N. refused to take the mouthpiece anymore from the instructor and rushed to the surface. Despite the efforts of the instructor to prevent the rapid surfacing and force him to breathe air, K. N. managed to surface. Upon reaching the surface, he lost consciousness and the air left his lungs with a whistling through the larynx. From the mouth and nose of the victim, a pink froth began to emerge. With the help of the lifeguards, the victim was instantly placed in a boat with his body sloping toward his head. Along the neck and shoulders there was observed a pronounced subcutaneous emphysema, accompanied by moderate cyanosis of the face and swelling of the cervical veins and the veins of the upper limbs. After around 1-2 min, the breathing stopped, and artificial mouth-nose respiration was begun. The victim was taken to the ship, which made for shore, as there was no barochamber on board.

After around 10 min the heart activity stopped and indirect heart massage was begun. Subsequently, on account of increasing venous congestion in the region of the head and neck and the suspicion of a left-side pneumothorax, growing during the artificial respiration, a thoracotomy was performed, which revealed a pneumothorax, acute vesicular emphysema, and a pronounced pneumomediastinum, compressing the v. cava cranialis. The mediastinal pleura and the pericardium were opened and direct heart massage performed. During this, it was noticed that all the coronary arteries were nearly full with small air bubbles, not permitting the passage of blood. Despite a lengthy heart massage (over 1 hour and 30 min), the heart did not beat and the pupils remained fixedly enlarged. Since death was definite, the victim was not taken to a barochamber upon arrival at Burgas.

Autopsy revealed acute double vesicular emphysema, in places with groups of blisters the size of a man's fist, pronounced

mediastinal and subcutaneous emphysema, a multitude of air bubbles, almost entirely filling the cranial and coronary arteries, as well as the most part of the other arteries of the major blood circulation.

In the case of barotrauma from the valve effect, if there is no indication of air embolism, there is no need for recompression with a barochamber, although the patient should be kept in proximity of one, and put into the barochamber at the least suspicion of embolism. To guard against embolism, the patients are given cough suppressants (codeine, dikodal, etc.).

Case 3. Ya. P., 39 years old, doctor. In August 1968 near the city of Pomorie, he descended with a group of vacationing skin divers to a depth of 12 m as a training exercise. Upon reaching the bottom, he noted that his equipment was supplying air with difficulty, and he was forced to suck air with great force for the duration of around 20 minutes. Soon after boarding the ship, he complained of slight pain in the chest. Coughing brought up a bloody frothlike sputum. That decided the diagnosis: barotrauma due to the valve effect. The patient remained 1 day in bed, receiving codeine tablets to control coughing and vitamin C. At the same time, he was prescribed denosulfamide and forbidden to enter the water for 2 days. In this time, the symptoms of barotrauma completely disappeared.

In the case of patients that are treated in a barochamber, due to the possible occurrence of complications the patients should remain in proximity of the chamber for at least 6 hours after decompression. As a precaution, to prevent bronchial pneumonia, antibiotics or pharmaceuticals are prescribed.

If, during the halting stage, or more often after leaving the barochamber, there are signs of cranial edema, the conven-

tional methods are used to treat it: dehydration therapy (manite, hypertonic glyucose, magnesium sulfate, urea, diuretics) and medications for quick recovery of the brain cells (vitamins, aminosine triphosphate, glutaminic acid, lutsidril, etc.).

Occasionally even after barochamber treatment there may remain temporary or permanent defects, paresis, paralysis, Jenkinson's contortions, etc., depending on the size of the blocked vessel, the zone remaining ischemic, how long it took to get the patient to the barochamber and so on.

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