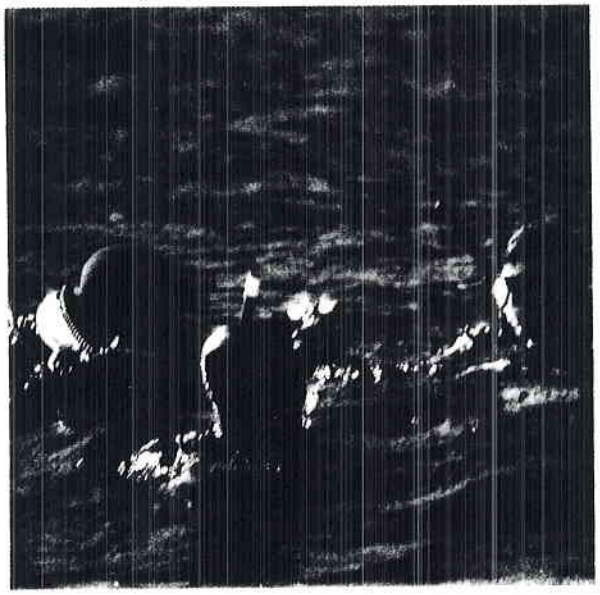
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SCIENTIFIC DIVING METHODOLOGY



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SURFACE SUPPLIED MIXES TECHNIQUE FOR SCIENTIFIC DIVING	COLANTONI, Paolo (Instituto di geologica Marina del C.N.R Bolonge-Italy)	103-104
MARINE FLORA	AYKULU, Güler University of Istanbul School of Aquatic Products	105-107
THE HAND-TOWED NET METHOD FOR DIRECT SAMPLING IN POSIDONIA OCEANICA BEDS	GIOVANNI et all. (Staz. Zool. Napoli)	108-109
A METHOD FOR SEMIQUANTITATIVE SAMPLINGS OF THE SESSILE EPIFAUNA OF THE POSIDONIA OCEANICA BEDS	ANORSEA et all. (Ist. Zoologica Univ. Genova)	110-111
MARITIME RIGHTS	VAN DENBRUCK, Carina (Universty of Brussel)	112-117
MEDICAL ASPECTS OF DIVING DISEASES OF ELEVATED PRESSURE DISORDERS ARISING OF	AYDIN, s. AKTAŞ, ş. (Istanbul Medicine Faculty University of Istanbul, Dept of Underwater and Hyperbaric Medicine)	118-127
DISORDERS ARISING OF ABNORMAL GAS PRESSURES	AKTAŞ, ş. - AYDIN, s. University of Istanbul, Medicine Faculty, Underwater and Hyperbaric Medicine	128-132

DISEASES OF ELEVATED PRESSURE

BAROTRAUMAS

Barotrauma is defined as, the tissue damage resulting from the expansion or contraction of gases in enclosed spaces. There are two types of barotrauma.

1- Barotrauma of ascent

2- Barotrauma of descent

Clinical presentation of Boyle's law is brotrauma. PV=K where K is a constant P= Pressure V= Volume.

Barotauma can occur during all types of diving activites. Most of the divers are well experienced to some degree of barotrauma.

Barotrauma of descent is also called "S q u e e z e". EAR BAROTRAUMA

This is the most common diving related disorder among divers. Subdivision is made according to anatomical sections. External-ear, middle-ear, inner-ear barotrauma. EXTERNAL EAR BAROTRAUMA OF DESCENT (external ear squeeze)

It is caused by a pressure differential between the external ear canal and the middle ear. Mainly, ear plugs, tight fitting hoods or occasionally wax in the external canal exclude water at pressure from entering the canal are the reasons. Therefore external ear canal remains at atmospheric pressure while pressure rises in the middle ear, causing the tympanic membrane to bulge outwards and congestion and haemorrhage of the lining.

A pressure differential of 2 meters of sea sea-water is required before damage occurs. Symptom during dive is the pain which is not releived by equalization.

Clinical symptoms are usually mild. % 3 Hydrogen Peroxide washings and if secondary bacterial infection happens antibiotics should be used.

Diving should not be continued until healing completed. Care must be taken not to use fitting hoods, ear plugs and divers who have excessive cerumen in the canal or external otitis, slould not dive during the healing period.

MIDDLE EAR BAROTRAUMA OF DESCENT (Middle ear squeeze)

This is the most common barotrauma and is caused by the failure or inability of a diver to equalize pressure in the middle ear space through the eustachian tube. The eustachian tube opens when the pressure gradient between the pharynx and the middle ear reaches to 10 mm Hg. Many exprienced divers are doing this by yawning or mowing jaw or swallowing. Most common method is the valsalva manouevre which is called "c l e a r i n g t h e e a r s" by divers.

If the eustachian tubes are blocked during diving, external pressure increases while pressure in the middle ear remains at atmospheric level. Discomfort pain and blocking of the eustachian tube may occur with a descent of 2 meters. If the diver continues to dive deeper mucosal congestion, oedema and haemorrhage within the middle ear cavity occur. Then perforation of the tympanic membrane occurs. Perforation pressure is altering from subject to each subject. After perforation in cold water caloric vertigo may occur but does not persist, disappears after 30 seconds.

PREDISPOSING FACTORS WHICH LEADS TO MIDDLE - EAR SQUEEZE;

Upper respiratory tract infections, allergic or vasomotor rhinitis, hypertrofic adenoid tissue and the inability to perform valsalva.

Diagnosis is made by autoscopic examination.

Treatment includes decongestants, antihistamines and if perforation happens a systemic antibotic should be given. Diver should not return diving until complete healing is achived. Mild squeeze needs about 3,8 days and serious squeeze or perforation needs 2-4 weeks intervals without diving.

Prevention of squeeze could be obtained by frequent clearing during descent, if locking of the ostium happens to ascent a few ftuntil clearing the ear is a good method.

Any diver who is unable to clear ears on the surface should not dive. Also diving in the inverted position makes the eustachian tubes to open more difficult so feet first diving is helpfull.

INNER EAR BAROTRAUMA OF DESCENT

This pathology is frequently occurs during descent with blocked eustachian tube followed by forceful valsalva. In other words it is a complication of middle ear barotrauma.

Two mechanisms are postulated to explain this pathology. In one, because of the relative negative pressure in the middle ear cavity, tympanic membrane bulges inwards and pushes stapes footplate into the inner-ear, therefore displacement of the perilymph forces round-window to bulge outwards, if at this point a forcefull valsalva is performed; a pressure increase in the serebro-spinal fluid occurs leading rupture of the round window into the middle ear.

The other explanation is with the forcefull valsalva an increase in the pressure of the middle ear while leads the tympanic membrane return rapidly to its normal position, the stapes moves outwards and the round window pulled inwards. So the reversed flow of the perilymph may not be sufficiently rapid to avoid damage to the inner-ear membranes.

Symptoms are a sensation of blockade in the affectedear tinnitussudden hearing loss vertigo, ataxia vomiting, nausea and disorientation. Treatment: An Audiogram and electronystagmogram should be taken to document the extent of the damage. Head elevated bed rest, antivertiginous therapy should be iniated. If no improvement obtained in 48 hours, surgical repair should be done.

MIDDLE EAR BAROTRAUMA OF ASCENT

This pathology is caused by the failure of air to vent from the middle ear space during ascent because the eustachian tube is blocked. Symptom is a feeling of a fullness in the middle ear. Pain occurs and if the blockade is very tight sometimes perforation of the ear-drum can happen. Alternobaric vertigo is seen very frequently during this pathology.

Diagnosis: Bulged or perforated eardrum with no haemorrhage or swelling. Treatment is done with oral deconjestents and if rupture happens antibiotics should be given.

DESCENT BAROTRAUMA OF THE SINUSES (squeeze of the sinuses)

Generally frontal and maxiller sinuses are affected. Sinus squeeze occurs when the ostium is obstructed. While the sinus remains at atmospheric pressure, ambient pressure increasese and this negative pressure makes the sinus mucosa to swell and to haemorrhagia. If the pressure differential continues bleeding occurs and after a while pressure in the sinus is equalized.

Symptoms of sinus squeeze include sharp pain above, below or behind eyes during descent which can be releived after blood fill in the cavity. After acsent a bloody discharge comes from the nose.

Treatment: Administration of nasal decongestants, analgesics and antibiotics.

ASCENT BAROTRAUMA OF THE SINUSES

Mostly caused by redundant sinus mucosa, cysts or polyps, obstructing the ostium of the sinus during ascent. Therefore an overpressurezation occurs. Symptomps are sharp pain, numbness or parathesias along the infra-orbital nerve.

Treatment: During ascent while the diver in water, an initial descent to the depth and changing head position and slow ascent rates should be tried if blockade occurs. After the dive nasal decongestants should be given.

DESCENT BAROTRAUMA OF LUNGS (Lung squeeze)

During breath-hold dive or surface-Supplied dive this pathology may happen. If lungs are forced to contract to beyond its residual capacity, intrapulmonary pressure becomes negative and large quantites of blood shifts into the chest, causing capillary congestion and pulmonary haemorrhage.

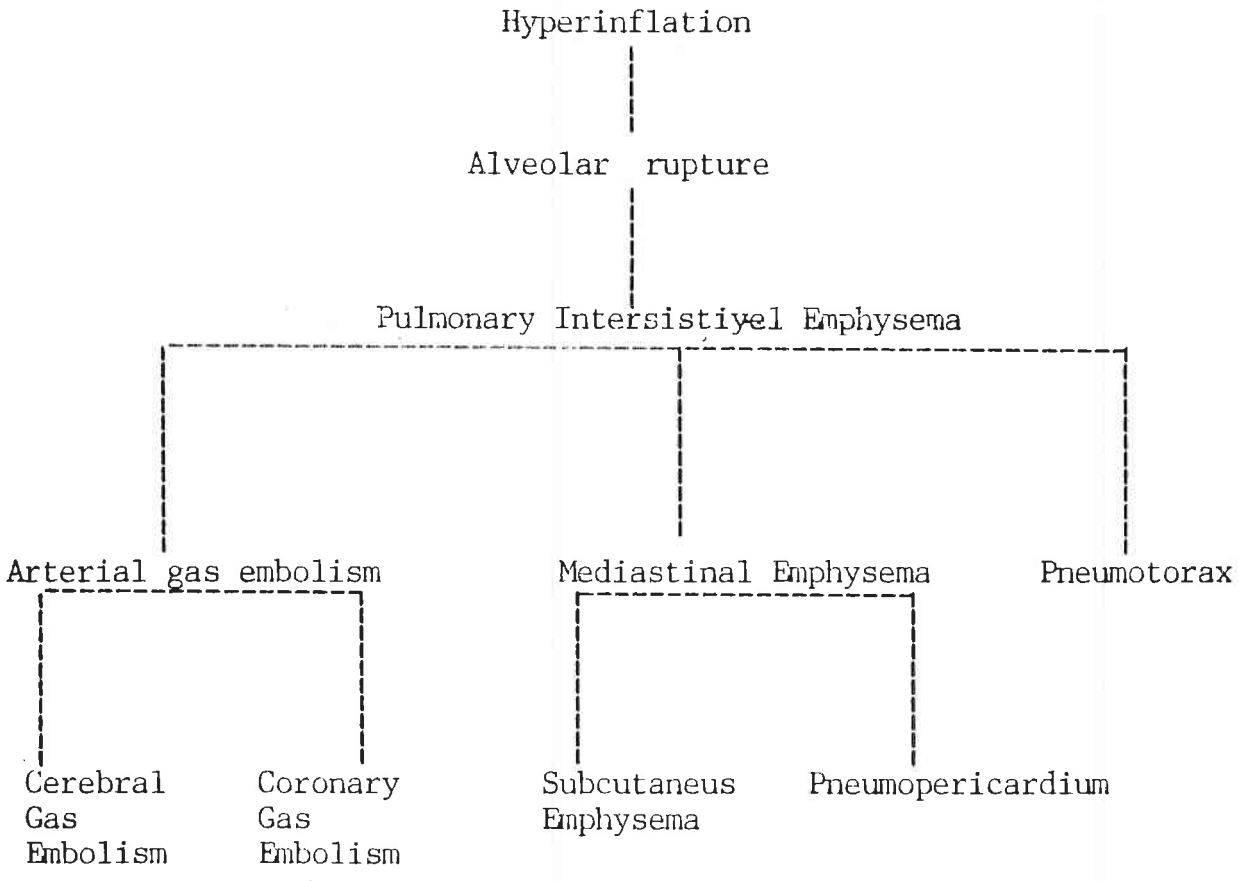
Each individual has a breath-hold dive limit. This limit is determined by the ratio of total lung capacity to residual volume, There fore it is avoidable to prevent this pathology in breath-hold dive by not exceeding the limit.

Symptoms and signs include, duysphea, cyanosis, hemoptysis and chest pain.

Treatment includes resuscitation, aspiration giving oxygen and if necessary PEEP PULMONARY BAROTRAUMA of ASCENT (Pulmonary overinflation): sometimes called burst lung. This pathology may be seen in unconscious ascent, rapid uncontrolled ascent, and divers who do not have proper medical examinations periodically.

Faulty breathing apparatus in head down position can cause this pathology.

Expanding gases in the lungs during ascent may be trapped in the lunge either by voluntary or involuntary breath-hold during ascent. Panic is also one of the main reasons. Also local obstructions trapping the gas intra-pulmonary segments, such as broncholits, pulmonary cysts or viscid pulmonary secretions. Pulmonary barotrauma of ascent can be divided into these sub divisions.



Solid lines indicate probable consequences. Dashed lines indicate possible consequences.

Lungs can not resist to overpressure. Even 70 mm Hg or 100 mm Hg overpressure can burst the lungs

a- PULMONARY TISSUE DAMAGE

A sudden high pitched cry can be heard after the diver surfaces. Dyspnoea, cough and haemoptysis are symptoms of the lung damage. Widespread alveolar rupture may result in respiratory death.

b- SURGICAL (Mediastinel Emphyseme)

After alveolar rupture, gas escapes into the interstitüal pulmonary tissues. This gas passes through loose tissue surrounding the airways and into the Hiler region to medistinium and neck.

Symptoms include voice changes, a fullness in the throat, dysphagia, dyspnoea in severe cases syncope or shock.

X-ray examination well decument this pathology.

c- PNEUMOTORAX :

This is due to the rupture of the viscerel pleura-Air enters in the pleural cavity and expands during ascent. This may be accompanied by haemorrhagie.

Symptoms are pain in the affected site, dyspnoea and tachypnoea. In severe cases shock may appear.

d- AIR EMBOLISM

This is the most serious and dangerous diving related pathology. % 50 of the cases result with death. While ascenting to the surface trapped gases rupture the alveoli and enter into the capilleries then to pulmonary veins. This gas is then distrubuted by left heart into the systemic circulation. These emboli can cause obtruction and infarction. Death usually occurs after cerebral or coronary embolism. Air embolism is frequently accompanying to mediastinal emphyseme.

Symptoms include rapid onset, loss of coinsciousness, vertigo, aphasia, visual disturbances, convulsions, cardiac-type chest pain, skin marbling.

Treatment: Pulmonary tissue damage requires % 100 oxygen breathing to maintain arterial oxygen in adequete levels. Electroyte and fluid balance should be carefully monitored. Surgical emphysema may not need treatment with mild symptoms. % 100 oxygen must be given by mask. If symptoms are severe therapeutic recompression using oxygen is necessary. Treatment of pneumotorax in mild cases: % 100 oxygen given by mask, bed rest and analgesics. Serious cases need underwater suction.

Treatment of air embolism is a real emergency.

-Patient should be recompressed within minutes,

- Steroids (decadron) administration 10-12 mg initially intramuscularly followed by 4-6 mg every six hours.
- I.V fluid infusion.
- Transport of the patient must be head down and lying on his left side.

DECOMPRESSION SICKNESS

This is a disease of protean clinical manifestations which is produced by an excessively rapid lowering of ambient pressure. This causes the inert gas dissolved in the tissues to come out of physical solution and enter the gas phase which causes gas bubbles to from in the tissues and in arterial and venous blood.

- Predisposing factors: Exercise during bottom phase of dive. Main mechanism for is the increase in tribonucleation.
- Injury may play an increasing risk in type I DCS.
- Cold water increases the probabilty of decompression sickness. During decompression diver become cold and vozoconstriction caused by cold may then impair inert gas elimination.
- Obesity: Excessive fat adsorbs large guantities of gas and adiphose tissue is too slow for eliminate adsorbed gas. This effect becomes important in after long deep dives.
- Alcohol drinking before the dive increases the risk because of vasodilation and increased heart rate.
- Fatique: Excessive fatique beginning of the dive makes more suspectible to DCS.
- Age: Divers who are old and begirnners and not fit are under more risk.

TYPE II DECOMPRESSION SICKNESS

Mostly central Nervous System pathologies are seen. Spinal involvement, cerebral involvement, cerebellar involvement, inner-ear involvement, pulmonary involvement are very serious and emergency recompression treatment should be performed. Mechanism is the bubbles which are blocking the capillaries mainly and form edema and infarction.

PULMONARY DECOMPRESSION SICKNESS

Pulmonary capillaries are always involved in the DSC because lungs are filtering the bubbles circulating. But symptoms are severe if the omitted decompression is gross.
These symptoms: substernal pain, cough and dyspnea and respiratory distress. Substernal pain is in burning character and increase with time. Coughing is unpreventable. If immediate recompression treatment not performed it is highly fatal.

SPINAL INVOLVEMENT (Spinal DCS)

This is the most frequent form of type II DCS. Paraplegia is a classic symptom. Bladder paralysis and fecal incontinence often accompany. Blood flow through epidural veins are slow. If central venous pressure rises it gets slower and tends to block very easily by the bubbles. So a venous infarction of spinal cord occurs. Recompression treatment is absolutely a necessity.

INNER EAR DECOMPRESSION SICKNESS

This is also called "Staggers". In these cases cochlea and vestibula are usually involved both. Symptoms are very similar to roud vindow rupture. Vertigo, nausea vomiting, tinnitus and sudden hearing loss. Ataxia and nystagmus are common. This is areal emergency and recompression treatment should begin within one hour.

Most of the type II DCS symptoms occur within the first 3 hours but may elongate till to 24 hours.
Tereatment can be summarized as follows.

1- Recompression Treatmert
2- I.V fluid Treatment (Rheumakrodex, Ringer lactate solutions)
3- Steroids (12-16 mg initially and 4-6 mg for four times)
4- Anticoagulants (Aspirin, Heparin)
5- Rehabilitation
6- % 100 Oxygen breathing on surface

Aseptic necrosis of bone in compressed air workers and divers. Main reason is to expose raised pressures over 2,4 ATA in divers. Etiology is unknown. Particularly long bones are affected. Head neck and shaft of humerus, femur and upper part of tibia.

Lesions are divided into two groubs.

1- Juksta-articular lesions named A type lesions which are concerning the joints of koksa-femoral and Gleno-Humeral joints.

2- Head neck and shaft lesions named B type lesions.

Predisposing factors: Age, years in diving, range of pressure, number of type I decompression sickness.

There is no known treatment. Screening with X-rays in two years period should be done for early diagnosis. Divers who have Juksta-articular lesions should not be allowed to dive any more.