Ear, nose and throat and non-acoustic barotrauma

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Abstract. The organs of the ear, nose and throat (ENT) contain air- or gas-filled cavities, which make them sensitive to pressure changes. There is a specific pathophysiology involved when these structures are exposed to non-acoustic pressure changes, which are usually not traumatic in normals. The concepts of pathophysiology, diagnosis and treatment of these traumas in an emergency setting are reviewed.

Introduction

A blast trauma is the physical damage to the human body caused by a shock wave after an explosion. Typically, blast traumas are subdivided into four possible types of lesions, where only the primary lesions are directly caused by a shock wave.

In general terms, barotrauma is defined as any lesion caused or provoked by a change in pressure differential between various anatomical parts of the organ involved. As membranes and tissue walls possess a certain resistance to pressure, a change in pressure does not always and automatically cause a lesion. Pressure differentials can be caused by external factors (change in environmental pressure, sudden increase in locally applied external pressure) or by internal pressure changes. Barotrauma results in physical disruption of membranes and/or tissue, with anatomical and functional damage as a consequence.

As third window syndromes make the ear particularly sensitive to pressure changes, a separate section of this chapter is dedicated to these syndromes and their typical consequences in scenarios involving even minor pressure changes. The aim of this chapter is to summarize and review expert opinions and literature data on these subjects.

1. Blast trauma

1.1. Physics

The blast is the body’s damage caused by a shock wave after an explosion. It is thus the energy release, which appears after disintegration by a supersonic explosive composition known as a “bang” (e.g., 1-methyl 2,4,6 trinitrobenzene [TNT], Semtex)

This bang generates a quick chemical reaction involving the decomposition of an explosive substance due to a sequential wave shock. This creates a mass of gas, the warmer and more compressed central areas of which are propagated faster than the peripheral areas, thereby creating a frontal pressure or shock wave. The latter transfers the energy to the nearby molecular components. This is known as an “explosive’s brisant power.”

The profile of a wave shock in an open field is ideally of the Friedlander type.

It starts with an abrupt elevation of the static pressure or shock front. Then, the pressure decreases exponentially towards the atmospheric pressure. The time between the bang and the return to the atmospheric pressure is the compression phase (t0). On the contrary, if we achieve distance from the explosion, the wave shock has a negative phase (depressurization), after which there is a progressive return to the atmospheric pressure due to oscillation. Each explosive or weapon has its own pressure wave. The signature of each explosive requires knowledge of the ascent time, the pressure ridge, the time of the positive phase and the pulse.\textsuperscript{1}

A sub-aquatic blast is much more dangerous at the same distance than an air blast because the density of water causes a wider high-speed spread.

The profile of the shock wave depends on the environment in which the detonation appears. In the case of an open space, the wave shock will
be omnidirectional, spherical or hemispherical, whether it appears near to the ground or not. On the opposite, in a closed space, the wave will be confined or directed if channelled by a rigid wall.

The destructive power of each explosive is given by means of an equivalence compared to an explosive reference: TNT.

A cannon effect can be observed if the explosive is maintained in a confined space (e.g., a mine buried in a clayey soil), while we can observe fragmentation if the explosive is placed in a hull, fragments of which will be expelled at a speed greater than that of the initial wave shock.

The presence of a wall is enough to form multiple shock waves due to reflection. Each wave shock then generates bodily injuries.2

1.2. Physiopathology

Two mechanisms of injury should be considered.3

First, the implosion, which means that, in soft body areas, there is initially compression followed by sudden relaxation, causing parietal fractures (e.g., auricular and digestive injuries).

Second, the shock wave in tissues of different density causes the sudden compression of less dense tissues by the denser tissues. The quick return to the initial situation generates injuries in the tissue interface (e.g., pulmonary and laryngeal injuries).

An explosion generates four types of injuries. A primary injury is the result of the direct effect of the shock wave on the organs. A secondary injury is the consequence of the impact from ammunition fragments or environmental rubble. Third, the victim’s body can be injured because of its displacement, or its projection, which would mean a tertiary injury in this case. Finally, a quaternary injury can be observed and covers all others consequences (collapse, thermal effect, dust inhalation etc.).4

The ear is the first organ reached in case of an air blast, followed by damages to the respiratory apparel (lung and larynx) and finally the bowels. The bone or nerve injuries occur in cases of a borne blast (e.g., a projectile striking armour plating).5

1.3. Ear lesions

An eardrum tear occurs at 20 kPa for a wave shock of 3 m/s in a positive phase. The tympanic examination is therefore a priority in cases of blast suspicion. The first affected organ is the middle ear, which can present a tympanic tear and the possible disarticulation of the tympano-ossicular chain, or a haemorrhage resulting in conductive deafness (for more details, see chapter 12). The inner ear can also be affected, with injury to the cochlear membranes, thereby causing perceptive hearing loss.5

The symptomatology is characterized by hearing loss, tinnitus, earache, vertigo and secondary otorrhoea. One must, however, keep in mind that extreme noise exposure also has an impact on the central nervous system, causing neural damage.6 This damage can also influence the intensity and persistence of “aural symptoms”, such as tinnitus, vertigo and poor speech discrimination.

In a recent study of 63 patients, who were examined at the Antwerp University Hospital within one month after the terrorist attack at Brussels International Airport on 22nd March 2016, we found the predominant presenting symptoms to be tinnitus and aural fullness (Table 1). This was the case in both those who presented with drum perforations and one particular individual with haemotympanum, as well as those with normal otoscopy.

1.4. Associated lesions

A clinical finding of tympanic perforation must lead to a lung X-ray or preferably a CT scan of the thorax.

| Table 1 |
| --- | --- | --- |
| **Table 1** Overview of physical findings during otological and audiological examinations vs. presenting symptoms in a population of 63 individuals who presented within one month after being exposed to explosions at the Brussels International Airport departure hall on 22nd March 2016. Complaints of tinnitus and aural fullness predominated, even in those individuals where no objective abnormalities were found. |
| **Hearing loss** | **Symptoms** | **No** | **Yes** | **Total** |
| Normal otoscopy | Tinnitus | 16 | 24 | 40 |
| | Ear pressure | 12 | 19 | 31 |
| | Hyperacusis | 4 | 3 | 7 |
| Abnormal otoscopy | Tinnitus | 3 | 3 | 6 |
| | Ear pressure | 1 | 1 | 2 |
| | Hyperacusis | 0 | 0 | 0 |
| Total tinnitus | 16 | 27 | 43 |
| Total ear pressure | 12 | 20 | 32 |
| Total hyperacusis | 4 | 3 | 7 |
At the pulmonary level, the injuries are explained by the weight difference between the affected lung and a normal lung. The alveolar haemorrhage increases the weight of the lung, thereby causing asphyxia or systemic air embolism. It is generally accepted that it is the impact between the lung and the rib cage that generates injuries. We consider the movement speed of the rib cage to be a good indicator of the injury.7

We first observe laryngeal damages at a speed of 4 m/s, while the pulmonary lesion threshold appears at a speed of 10 m/s.

Among the 63 patients described in Table 1, three individuals presented with a drum perforation and one with a haemotympanum. All patients were examined by fiberoptic indirect laryngoscopy. No one showed evidence of laryngeal damage. Results from the physical examination of the lungs in all patients and the medical imaging of the thorax in 19 patients were normal across the board. Of course, we only examined a selection of victims of this terrorist attack who presented at the emergency room in an outpatient setting.

1.5. Pre-hospital assessment

Blood loss from the ear(s) and obvious hearing loss clearly suggest ear damage. If these signs are present one must think of possible lung damage since the lung is the second organ affected in terms of frequency after the ear. The diagnosis is based on a triad: respiratory distress, hypoxemia and radiological abnormalities. The respiratory signs are: tachypnoea, dyspnoea, cyanosis, haemoptysis and chest pain. Neurologic signs can be seen as a result of cerebral air embolism.8

1.6. Assessment and management at the hospital

Otomicroscopic examination will facilitate the first estimation of ear damage. Audiometry will show any combination, from high-frequency perceptive hearing loss to over-conductive hearing loss and total deafness.

A thoracic CT scan or a standard pulmonary X-ray must be systematic in cases of patients suspected of blast. We can see diffuse opacities appearing after a few hours (max. 24-48 h) and disappearing after seven days. Other injuries can be observed, such as pneumothorax, haemothorax and subcutaneous emphysema.

Furthermore, the upper airway is classically affected by the passage of the shock wave. Laryngeal petechiae are commonly found during fibroscopy, with the presence of these injuries being a sign of pulmonary injury because the laryngeal lesion threshold is just below the pulmonary threshold.

Finally, the digestive system is also affected in cases of primary blast because its lesion threshold is just below the upper airway. The bowel may be torn, leading to pneumoperitoneum or haemoperitoneum.9,10

1.7 Take-home messages

The damage after a blast trauma depends on the power of the explosive, the distance between the victims and the deflagration, and the space where it appears.

The ear and the upper and lower respiratory tract are the most frequently affected.

In order to facilitate a quick screening of the victims, an ear examination is thus the first examination to be undertaken in cases of comminatory explosion.

The lethal lesions appear in the lung and can be seen up to 48 h after the explosion.

2. Barotrauma of the ear

2.1. Introduction

In general terms, barotrauma is defined as any lesion caused or provoked by a change in pressure differential between various anatomical parts of the organ involved. As membranes and tissue walls possess a certain resistance to pressure, a change in pressure does not always and automatically cause a lesion. Pressure differentials can be caused by external factors (change in environmental pressure, sudden increase in locally applied external pressure) or by internal pressure changes. Barotrauma results in the physical disruption of membranes and/or tissue, with anatomical and functional damage as a consequence.

2.2. Relevant anatomy

2.2.1. Eustachian tube (ET) anatomy: bony vs. cartilaginous part, muscle actions in the ET opening

In a normal human adult, the middle ear cavities are connected to the “outside” by means of the ET, a short canal (42-43 mm on average) originating from the antero-inferior wall of the middle ear and
leading to the bony skull and the nasopharyngeal wall towards the nasopharynx (just above the velum palatinum, at the level of the inferior nasal meatus). Here, it raises the mucosa slightly, in turn forming the tubal elevation or torus tubarius. Its wall is composed of a mucosal lining of respiratory epithelium (contiguous with the nasal mucosa), containing columnar ciliated cells, goblet cells and mucus glands. The latero-posterior third of the ET is anatomically in an open state (as it passes through the bone), the anteromedial two thirds of the ET is supported by a cartilaginous “comma-shaped” structure, lying posteromedially to the canal, and the rest of the wall is formed by fibrous tissue. Here, both walls of the ET are in apposition, such that the ET is naturally “closed”. The Ostmann fat pad, lying inferolaterally to the nasopharyngeal orifice of the ET, provides bulk and aids in maintaining a closed state during rest.

The ET lies at an angle of approximately 27° from the horizontal plane, while both parts of the ET are connected at the isthmus. In children, the ET is shorter and generally more horizontal and less angulated.11

The tensor veli palatini muscle originates from the bony skull base and the upper portion of the cartilaginous “comma”, before running downward, then turning medial around the pterygoid spine and fanning out in the soft palate. Other muscles lining the ET are the levator veli palatini muscle, the salpingopharyngeus muscle and the tensor tympani muscle. Contraction of the tensor veli palatini muscle during swallowing or yawning results in the bending of the cartilaginous plate into a more rounded form, thereby opening the ET. Normally, the ET opens frequently (three-five times per minute) ensuring a regular ventilation and pressure maintenance of the middle ear cavities.

2.2.2. Tympanic membrane vs. oval/round window: size/surface difference, connection of round window to perilymphatic fluid, duct and intracerebral pressure changes

External pressures are primarily transmitted to the surface of the eardrum. In the process of sound transmission, the larger surface of the eardrum compared to the oval window (14 times larger) leads to a “sound pressure concentration” of approximately 18:1. As the connection between the eardrum and the stapes is made of articulating ossicles, large but slow (compared to sound vibrations) movements of the eardrum are not found in comparable excursions of the stapes’ footplate.

Two smaller membranes in the middle ear medial wall, the oval window and the round window, separate the perilymphatic ducts (scala tympani and scala vestibuli) of the cochlea from the air-filled middle ear. Intracerebral pressure changes are reflected in the perilymph through the perilymphatic sac and duct, which may lead to pressure on the inner surface of these membranes (fluid side).

The middle ear cavity forms a continuum with the mastoid cells’ air sacs. Whereas the volume of the middle ear cavity itself has been measured at approximately 3.5 mL, the volume of the mastoid cells is highly variable among individuals and can reach volumes of over 10 mL.

2.2.3. Facial nerve anatomy in the middle ear

In its anatomical course, the facial nerve (n. VII) passes along the medial wall of the tympanic cavity. From the cerebellopontine angle, the facial nerve runs through the internal acoustic canal and reaches the middle ear after making a sharp angle, called a “genu”, through the canalis nervi facialis. The tympanic segment of the facial nerve runs medially to the incus, giving rise to the nervus stapedius (a motor branch) and the chorda tympani (a sensory branch to the anterior two thirds of the tongue).
In approximately 11%, there is a bony dehiscence in the intratympanic portion of the facial nerve canal, exposing the nerve to middle ear pressure changes. This proportion is higher in patients with chronic otitis media, cholesteatoma or a history of middle ear surgery, probably due to either erosion or accidental trauma during surgery. This dehiscence may not even show up on high-resolution CT scanning of the middle ear.

The paranasal sinuses are lined with a contiguous respiratory epithelium similar to the nasal mucosa, while ventilation of these cavities is assured by the sinus orifices and orificial canals.

The maxillary sinus orifice or ostium, which is 2.5 mm in diameter on average, lies high up the medial wall of the sinus and opens into the hiatus semilunaris of the lateral nasal cavity. This makes drainage of the maxillary sinus contents dependent on mucociliary clearance. The total volume of the sinus is approximately 10 mL, but considerable inter- and intra-individual variation may occur. The floor of the maxillary sinus, which is approximately level with the floor of the nose, is shaped by the conical protuberances of the roots of the first and second maxillary molar teeth.

The ethmoidal sinuses, which are lying medially to the orbita, form the lateral wall of the upper portion of the nasal cavity. They consist of numerous thin-walled air cells, separated from the orbita and the nasal cavity only by thin bony laminae. Orifices are numerous and their high localization facilitates drainage of their contents.

The sphenoid sinuses, located in the sphenoid bone in the posterior roof of the nasopharynx, are in close relation to the hypophyseal fossa. The orifice of the sphenoid sinuses is located high up the anterior wall, prohibiting easy drainage of its contents when in the upright position.

The frontal sinuses have a very long and narrow vertical drainage canal, opening in the anterior ethmoid sinus and draining into the middle meatus of the nasal passageway via the hiatus semilunaris. There is important anatomical variation, however, in all instances, although, for severe mucosal obstruction, passive drainage of the frontal sinus is possible in the upright position.

2.3. Physics (laws of Henry, Dalton and Boyle)

Barotrauma is caused by pressure changes in the gaseous surroundings or contents of hollow organs, as either a consequence of volume changes or pressure increase in fixed-volume organs. Pressure vs. volume relationships of gases are governed by physical gas laws, of which Boyle’s law is the most important.

2.3.1. Boyle’s law in relation to diving and aviation

Boyle’s law (after Robert Boyle, 1627-1691), which is sometimes referred to as Mariotte’s law (after Edme Mariotte, 1620-1684) or the Boyle-Mariotte law, describes the inverse proportionality of volume and pressure for a given quantity (mass) of a gas. Mathematically, Boyle’s law can be described as:

\[ PV = \text{constant} \]

It states that, as pressure increases, the volume of a gas will decrease in proportion. This, of course, is only applicable in cases involving a container with at least one mobile wall. Conversely, as pressure decreases, the volume will tend to expand, unless the elastic resistance of the container wall opposes this volume increase.

In environmental physiology, the surrounding pressure may be exerted by either gaseous or liquid compounds. When considering human physiology, the “basal” state of things (at sea level) is that we are exposed to an “atmospheric” pressure of 1,013 hPa, which is the force exerted by the usual weight (mass) (1 kg) of a column of 1 cm² of the air constituting Earth’s atmosphere (hence the unit 1 atm, which means “one atmosphere of pressure”).

As elevation increases, the pressure of the overlying atmosphere likewise decreases. However, because of the compressibility of the air, this does not occur in a linear fashion. At an altitude of 6,000 m above sea level, atmospheric pressure is roughly halved to 0.5 atm, although absolute vacuum is only reached at altitudes higher than 300,000 km.

When descending into the water, the surrounding pressure will increase by 1 atm every 10 m, as the weight (mass) of a water column, with a diameter of 1 cm² and a height of 10 m, is 1 kg. Note that there is a slight difference between sea and fresh water (1.026 vs. 1.000 kg/l). The increase in pressure when descending is linear: given that water is not compressible, the added weight of the water column between -10 and -20 m of depth will be the same as the added weight of the water column between 0 and -10 m of depth.

Therefore, plotting the volume changes in relation to depth under water gives a very
pressure exerted on the surface of the body will be transmitted to the entire tissue volume of the body; this is why a solid body is not “crushed” when immersed in (very deep) water, as opposed to a hollow body. Finally, this means that the absolute pressure variations in a human body (e.g., arterial blood pressure) will increase when the surrounding pressure is increased (however, when measuring blood pressure with a sphygmomanometer, which references the surrounding pressure, the measured arterial pressure will not differ).

2.4. Physiology and physiopathology

2.4.1. Mechanism of disease

2.4.1.1. Valsalva and other manoeuvres to equalize pressure in cases involving changes in environmental pressure

When the pressure around the human body changes, this pressure is transmitted (in line with Pascal’s principle) to all body surfaces, fluids and tissues. This causes the tympanic membrane to be pushed inwards, unless the pressure in the middle ear cavities is also increased. This is achieved when the ET opens, allowing air from the nasal cavity (in connection to the outside world, so at the same environmental pressure) can enter the tympanic cavity. As the ET is closed most of the time, several manoeuvres to actively or passively open it have been described.

The best-known such manoeuvre is the Valsalva manoeuvre (after Antonio Valsalva, 1666-1723), which consists of closing one’s mouth, pinching the nostrils to close the nose and blowing through the nose to increase the intranasal pressure. This forceful blocked nasal exhalation results in forcing air through the nasopharyngeal orifice of the ET, thereby “equalizing” the middle ear pressure. This is experienced as a “popping” of the ear drums. A Valsalva manoeuvre typically increases the air pressure of the nasal cavity to a maximum of 700-1,000 daPa (0.1 atm or 50-70 mmHg).

A more physiological way of opening the ET orifice would be by swallowing, yawning or moving the jaws and advancing the mandibula. By doing this, the tensor veli palatini and levator veli palatini muscles elevate the soft palate and change the shape of the cartilaginous portion of the ET, giving it a more rounded appearance and separating the ET walls attached to it. The Toynbee manoeuvre consists of swallowing while
keeping the nostrils closed, which helps because the movement of the tongue when swallowing will slightly increase the nasal cavity air pressure. Although other manoeuvres have been described (Frenzel manoeuvre, Edmonds technique, Lowry technique), they are generally less easily taught than the aforementioned ones.\textsuperscript{17}

Pressure differentials also exist in the paranasal sinuses, when the draining orifice is blocked by mucosal polyposis or by mucosal congestion. As the surrounding ambient pressure is transmitted to the bloodstream, with the mucosal inside the “isolated” sinus effectively subject to increased pressure by its blood supply, Boyle’s law is applicable, such that the air pocket inside the sinus will become smaller. An increase in nasal cavity pressure, such as that achieved with a Valsalva manoeuvre, can “equalize” the intranasal pressure by pushing air through the blocked orifice.

2.4.2. “Locking” of ET in cases of pressure differential: middle ear vs. nasal cavity

When the tissue pressure surrounding the ET increases above the maximum force exerted by the active contraction of the tensor veli palatini muscle, the ET becomes “blocked”, both by compression of the ET walls and by aspiration of the nasal cavity orifice in the direction of the middle ear by the lower pressure in the middle ear. This typically happens at a pressure of approximately 90 mmHg (120 cm water pressure or depth). Then, even a forcefully executed Valsalva manoeuvre will often not be capable of opening the ET.

Tests in a hyperbaric chamber have shown that a rapid pressure increase is more likely to be associated with an ET blockage, as the rapid rise of ambient pressure leads to negative middle ear pressures and reactive serous fluid transudation. This increases the volume (thickness) of the tympanic cavity mucosa, further reducing the patency of the ET. Likewise, forceful Valsalva manoeuvres might exert compression on the medial and lateral “lips” of the ET orifice, formed by the Ostmann fat pad, thereby effectively pushing the orifice into a closed state during the Valsalva manoeuvre.\textsuperscript{18}

2.4.1.3. Excessive Valsalva manoeuvres as the cause of either “blow-out” or “pull-in” inner ear barotrauma

The intranasal pressure, exerted by an excessive Valsalva manoeuvre, will be transmitted to the nasal mucosa, then onto the tissues of the facial massif and the neck base. This results in a direct pressure increase, as well as, by impeding the venous return from the skull vasculature, a rapid and progressive increase in intracranial pressure. By direct pressure transduction via the perilymphatic sac, perilymph fluid pressure will likewise increase. Moreover, it is postulated that this is a first mechanism of rupture of the round window (“explosive mechanism”). This may happen without any clear signs of middle ear barotrauma.

A second mechanism of inner ear barotrauma is related to an excessively forceful Valsalva manoeuvre, which would finally be able to open up the ET, allowing air to forcefully flow into the middle ear cavities. As the eardrum would be forcefully expelled outwards, the ossicular chain would be maximally stretched, resulting either in a luxation of the stapes’ footplate (disrupting the oval window) or in an inward bulging of the round window with subsequent tearing (the “implosive mechanism” of inner ear barotrauma) due to negative pressure transduction all through the scala vestibuli and scala tympani.\textsuperscript{19}

2.4.2. Clinical pictures

2.4.2.1. External ear squeeze

An isolated air space between the eardrum and the “outside world”, for example, by means of a sealed cerumen plug, tightly fitting earplugs or (in divers) a tight neoprene hood, will cause an outward bulging of the eardrum upon descent. Pain and possibly haemorrhage in the eardrum will ensue, which will only increase when the diver performs a Valsalva manoeuvre (as he has been taught to do). This “external ear barotrauma”, which may be diagnosed after removal of the obstructing body, could resemble middle ear barotrauma (see later) or be characterized by bullae on the eardrum. The latter sign may be mistaken for myringitis bullosa or otitis externa.

2.4.2.2. Middle ear barotrauma

The most common complication in relation to air travel, underwater diving and hyperbaric oxygen therapy is a “squeeze” of the middle ear caused by inward bulging of the eardrum, which is in response to the pressure differential between the surroundings (external auditory canal) and the middle ear cavity. Symptoms are a feeling of pressure or fullness of
the ear, pain, hearing loss and possible bleeding from the ear or nose. Middle ear barotrauma mostly happens on descent (pressure increase), as the ET usually permits passive evacuation of middle ear air upon ascent.

Typically, symptoms arise when the pressure differential is around 3-4 m (120-150 mmHg), as this is the point where the ET is firmly “blocked” and the elasticity of the eardrum has reached its maximum. Individual differences in eardrum elasticity may be responsible for higher “resistance” to pain or middle ear symptoms; it has been hypothesized that the degree of mastoid pneumatization may offer some protection, with larger volumes showing greater volume changes than smaller volumes. However, this has not been consistently shown. It is more likely that the different “depths” at which middle ear squeeze occurs are dependent on partial equalization (or otherwise) early in the descent, adding some air to the middle ear cavities, thereby postponing the “painful” point to depths that are sometimes as deep as 10-12 m.

Middle ear barotrauma occurring after air travel can sometimes be related to whether the flight is transcontinental or at night. As the passengers are not fully awake when the initial descent of the plane starts, the ET may already be well “locked” when the first attempts at the Valsalva manoeuvre are done, albeit in vain.

Visual inspection of the eardrum reveals the degree of barotrauma. The TEED scale, a visual evaluation scale, discerns six degrees of barotrauma. Originally described in 1944, the scale, which has been modified by McFie and later by Edmonds, provides an objective measurement; however, it is advisable to also describe the otoscopic findings (Table 2, Figure 3). Other classification systems exist; for example, in aeronautical medicine, middle ear effusion is used as the criterion to define different grades. This illustrates the importance of a descriptive medical notation.

After ascent, some blood may be evacuated via the ET and expelled by the expanding air in the middle ear cavity. When an eardrum rupture is present, little pain is felt, but blood evacuates from the external auditory canal.

The pressure differential necessary to provoke tympanic membrane rupture has been quoted very widely in the literature, ranging from 100-3,000 cm of water, and depends on the individual resistance of the eardrum. “Monomeric” tympanic membranes, resulting from the healing of eardrum

<table>
<thead>
<tr>
<th>Grade</th>
<th>Findings on otoscopy</th>
</tr>
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<tbody>
<tr>
<td>0</td>
<td>Normal examination</td>
</tr>
<tr>
<td>1</td>
<td>Tympanic membrane injection or retraction</td>
</tr>
<tr>
<td>2</td>
<td>Slightly haemorrhagic tympanic membrane</td>
</tr>
<tr>
<td>3</td>
<td>Grossly haemorrhagic tympanic membrane</td>
</tr>
<tr>
<td>4</td>
<td>Haemotympanum</td>
</tr>
<tr>
<td>5</td>
<td>Tympanic membrane perforation</td>
</tr>
</tbody>
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Figure 3
Modified TEED classification of middle ear barotrauma (from Edmonds et al.20)
perforations, do not have a dense fibrous layer between the inner mucosa and outer epithelium. They appear transparent on otoscopy and could move with respiration. Although they usually have little influence on hearing, they are much more fragile and could rupture during very low, albeit sudden, pressure changes, often without pain.

2.4.2.3. Inner ear barotrauma

Inner ear barotrauma may be accompanied by middle ear barotrauma; however, this is not always the case. In most cases, symptoms occur during descent, although it is not uncommon for them to only be noticed during or after the ascent to the surface. Inner ear barotrauma seldom occurs after altitude exposure.

The principal symptoms of inner ear barotrauma are hearing loss and tinnitus. Pathophysiologically, this can be related to the loss of perilymphatic fluid, which may be evidenced by the presence of gas in the cochlea on high-resolution CT scanning. When air enters the cochlea through a round window rupture, this may not always immediately lead to noticeable hearing loss (especially in a diving underwater environment, where directional hearing is in any case less efficient). During the ascent, air in the cochlea will expand, expelling more fluid from the perilymphatic ducts. More pronounced hearing loss, possibly with an accompanying feeling of unsteadiness or vertigo, will then become evident shortly after the dive.22

2.4.2.4. Sinus/nasal barotrauma, including facial nerve squeeze

A mild obstruction of the sinus drainage orifice may be overcome during descent by performing Valsalva manoeuvres. These may lead to increased swelling of the mucosal lining by mechanical irritation, as well as provoke a complete block during ascent. Although sinus barotrauma most often occurs during descent, “reverse block” during ascent is, at least for divers, a most painful and dangerous situation, as further ascent may lead to loss of buoyancy control (because of the excruciating pain) or even bony sinus wall rupture (resulting in air entering the orbital space or pneumocephalus).

When the sinus is subjected to hypopressure on descent, the decreasing volume of intrasinusal air will first provoke exudation of the mucosa, then possibly cause tearing and haemorrhaging of the mucosa. In maxillary sinus barotrauma, pain may be indicated in the upper first and second molar teeth, as their dental roots are protruding in the maxillary sinus floor. In a frontal sinus squeeze, a sharp pain is felt above the eyes, whereas a sphenoid sinus squeeze typically gives rise to occipital pains.

Upon ascending from a dive, the expanding air in the squeezed sinus will expel blood and mucus from the sinus: a profuse nosebleed after diving always indicates sinus barotrauma.

Facial “baroparesis” is one of the more exceptional complications of diving, occurring during the ascent from a dive. Air may remain trapped in the middle ear cavity and cause a compression of the facial nerve along its trajectory in the middle ear. This usually happens after a “difficult” equilibration upon descent, indicating a role for congested mucosa and a functionally impermeable ET. Pressure differentials of up to 300 cm of water pressure are needed to rupture the eardrum. An increase in middle ear pressure of 66 cm of water pressure may cause vertigo and nystagmus (so-called “alternobaric vertigo”), which is not uncommon in divers and aviators. When a bony dehiscence of the facial nerve canal exists, middle ear pressures above the capillary pressure (43.5 cm of water on average), an ischemic neuropraxis will ensue. Symptoms are those of peripheral facial nerve paresis or paralysis, together with an inability to close the eye or frown, as well as lower face motor affliction. Other symptoms, such as reduced lacrimation or reduced salivation, will not readily be detected by the patient. A change in taste on one side of the tongue may be described as a “metallic taste” or “strange feeling of the tongue”. If the neuropraxis persists for more that 3-5 h, irreversible damage to the facial nerve may result.

2.4.2.5. Dental barotraumas

Although not directly related to the ear, dental barotrauma is frequently encountered in divers and aviators. In contrast to middle ear and sinus barotraumas, this lesion often occurs upon ascent, either from a dive of a relatively long duration or when ascending to altitude.

Hollow spaces in the dentine component of a tooth are generally caused by either caries or retraction of an old filling. Metabolically often inert, it causes little symptoms by itself. The contents of
the enclosed gas space are in equilibrium with the
surroundings, both in pressure and in composition,
by virtue of Dalton’s and Henry’s laws of physics.
This is because the dentine is in close contact with
pulp and blood vessels of the tooth.

When descending in water, the increasing
environmental pressure will not directly affect
the volume of the cavity, as dentine is rigid, although
not entirely inelastic and brittle like enamel.
However, by being exposed now to blood and
pulp with a higher total gas pressure, progressive
“saturation” will occur; after a variable amount
of time (but generally more than 30 min), a new
pressure equilibrium will have been reached, with
the cavity now at same internal pressure as the
surrounding pressure.

When ascending from depth (or when rapidly
ascending to altitude after a prolonged stay at low
levels), the air contained in the dentine cavity will
expand (Boyle’s law) and result in expulsion of the
old eroded filling or fracture of either the enamel
layer, the dentine surface or the dental root. In a
study on military aviators, 8% of participants
reported at least one episode of barodontalgia; in 67%,
this was dental disease-related. In divers, the
prevalence of jaw or tooth pain related to dental
problems was 21%, with 1% having experienced
odontocraxis (expulsion of filling).

It has been shown that repeated compressions-
decompressions decrease the stability and solidity
of cements used in the fixation of crowns and
fillings. Notably, zinc phosphate and glass ionomer
cements experienced a reduction in bonding
strength by 90% and 50% respectively, after a series
of repeated pressure cycles to 3 atm. Resin cements
did not exhibit such a decrease in strength.

2.4.2.6. Larynx barotrauma (laryngocele)

Laryngocele usually presents as a cervical mass
with or without voice changes. Most laryngoceles
are asymptomatic and thought to be an occupational
disease among wind instrument players or glass-
blowers. Laryngoceles can also occur in association
with neoplasms of the larynx.

The respiration of compressed air and exhalation
against resistance may increase the size of a pre-
existing laryngocele in divers, causing possibly
fatal airway obstruction.

In the diving medical literature, laryngocele is
listed as an absolute contraindication for diving,
although no such case reports can be found in
this body of literature. A case has been described
with rupture to the sinus piriformis after forceful
sneezing with the Valsalva manoeuvre, while our
own experience lists one patient who presented with
a fracture of the laryngeal cartilage and subsequent
subcutaneous emphysema after scuba diving.

2.4.2.7. Facial squeeze (diving mask)

Much more common is facial barotrauma, caused
by the suction exerted by the diving mask when the
diver descends without blowing air into the mask.
The decreasing air space, which is formed by the
glass visor, the rubber or silicone sides of the mask,
and the skin and eyes of the diver, causes scleral
haemorrhage, as well as possible ecchymosis and
bruising along the contact border of the mask and
skin.

While spectacular (bloodshot eyes), this is
not a very dangerous condition and is resolved
spontaneously within approximately one week.
In the case of divers with a history of retinal
detachment however, this severe suction may be at
the origin of intraorbital pathology and vision loss.

2.4.3. Differential diagnosis

2.4.3.1. Inner ear barotrauma with inner ear
decompression sickness

While inner ear barotrauma has its origin in the
pressure-induced lesion of the round or oval
window, which occurs on descent, symptoms may
only become apparent during or after ascent, as air
bubbles in the cochlea or semicircular canals will
expand according to Boyle’s law. This sometimes
makes the differential diagnosis with inner ear
decompression sickness, caused by nitrogen
bubbles from inadequate desaturation (Henry’s
law), difficult. The differential diagnosis rests on
several anamnestic and clinical observations:

- Dive profile: Inner ear decompression sickness
arises after a dive with at least a certain risk that
decompression sickness will occur. While the
risk of decompression sickness cannot be readily
determined, as it is a multifactorial event, divers
usually indicate having dived either with mandatory
“decompression stops” (as indicated by their dive
computer or dive tables) or very close to that limit.
A depth of at least 25-30 m and a dive duration
of more than 30-45 min seem to be a minimum
condition for decompression bubbles to form,
although exceptions have been reported.
ENT and non-acoustic barotrauma

- Symptom latency: Usually, inner ear decompression sickness has a latency of 20-30 min after surfacing, whereas inner ear barotrauma would be apparent immediately upon surfacing or even shortly before.

- Symptoms: Inner ear decompression sickness symptoms usually present predominantly as vertigo (with nystagmus) and nausea (often with vomiting and general ill-feeling), while inner ear barotrauma has sensorineural hearing loss as its main symptom (sometimes accompanied by a feeling of unsteadiness, without true rotational vertigo or nystagmus).26

- ET dysfunction: In cases where a diver reports having had difficulties equalizing pressure in the middle ear upon descent or if where there are signs of middle ear barotrauma, the possibility of inner ear barotrauma is indicated. However, inner ear barotrauma can occur without (signs of) middle ear barotrauma.

2.4.3.2. Sinus barotrauma vs. dental barotrauma

Pain felt in the upper premolar area of the jaw may be caused by sinus maxillaris barotrauma, in which case, symptoms will typically arise during pressure increase. However, the presence of maxillary sinus polyps may constitute a "one-way valve" mechanism preventing air from exiting the sinus cavity upon ascent ("reverse barotrauma"). A dehiscence of a dental filling or a fracture of the tooth usually occurs during pressure reduction and is also commonly observed during air travel.

2.4.3.3. Facial nerve baroparesis vs. Bell’s palsy

Facial nerve baroparesis is diagnosed by anamnesis (in relation to an ascent from a dive or to altitude involving difficulties in middle ear equalization) and symptoms (a peripheral facial nerve paralysis). The possibility of a peripheral facial nerve paralysis of unknown cause (Bell’s palsy), which coincides exactly with the dive, is remote. In case of doubt, therapeutic measures should be taken as for facial baroparesis.

2.5. Pre-hospital assessment

2.5.1. Clinical history

For any symptom occurring in close relation to an activity or circumstance involving an environmental pressure change, the possibility of barotrauma must be evoked. Based on knowledge from physiopathology and physics, the diagnosis can usually be readily made.

2.5.2. Examination

Clinical examination should be directed at determining the nature of the injury and usually requires no specific tools.

2.5.3. Alarming signs for immediate referral to a hospital or hyperbaric centre

Barotrauma in the ENT region almost never needs to be referred to a hyperbaric recompression centre. As the medical staff of such a centre are usually proficient in diving medicine, a (telephone) consultation should always be considered in case of doubt. Diving syndromes that need urgent recompression (ideally, within a few hours after symptom appearance) are inner ear decompression sickness and cerebral gas embolism. The former may pose a diagnostic problem from inner ear barotrauma, such that, in case of doubt, urgent referral is advised. Patients should be placed on high-flow oxygen (100% via a non-rebreather mask at 15 l/min) and perfused with non-glucose containing solutes (NaCl 0.9% or Hartmann, 1 l over 3 h) to maintain or attempt proper microcirculatory oxygenation in the presence of nitrogen bubbles.27

2.6. Assessment and management at the hospital

2.6.1. Assessment: audiometry, tympanometry and vestibular testing

Visual inspection of the ear drum may show signs of barotrauma (TEED classification) or bulging of the ear drum, indicating increased retro tympanic pressure. In cases of hearing loss, pure tone audiometry and tympanometry are used to discern sensorineural from conductive hearing loss. Tuning fork tests (Weber, Rinne) are very useful and can suffice in an emergency setting.

CT scanning of the sinuses may be useful, as well as high-resolution CT scans of the temporal bones.

Vestibular testing should consist of clinical tests (Romberg, Unterberger, Frenzel glasses), while caloric tests and rotational testing (i.e., a formal ENG) are not considered an emergency. However, in the case of inner ear decompression sickness, these tests should be planned within one or two days and include caloric tests in order to verify the integrity of the peripheral vestibular organ.
2.6.2. Management

The basis for the management of barotrauma is to relieve the pressure, decrease symptoms and prevent secondary complications.

- In the case of middle ear and sinus barotrauma, topical or oral decongestive medication is indicated, as is adequate pain relief. In cases of haemorrhagic effusion, paracentesis is advised; in such cases and also in cases of sinus barotrauma with haemorrhage, prophylactic treatment with antibiotics is usually added.

- Inner ear barotrauma is treated with bed rest (with a 30° head elevation) and observation. If vertiginous symptoms persist or hearing loss worsens, exploration of the middle ear with the sealing of a possible round window fistula is indicated. Case reports have been described whereby treatment with hyperbaric oxygen (i.e., recompression in a hyperbaric chamber in order to hyperoxegenate the perilymphatic and endolymphatic fluids) has proved beneficial. However, as this treatment exposes the patient anew to increased environmental pressure, it carries a risk of renewed inner ear barotrauma. Current recommendations indicate that this should only be undertaken after bilateral paracentesis or with extreme care.

- Facial baroparesis should be considered as a true emergency, with attempts to reduce middle ear pressure or oxygenate the facial nerve rapidly undertaken. This can be done by paracentesis, breathing 100% oxygen or recompression in a hyperbaric chamber while breathing oxygen.

- Tooth barotrauma requires dental examination and treatment and is not very specific.

2.6.3. Return (to diving, flying etc.) after resolution of symptoms

When considering a return to diving (or renewed exposure to pressure changes), the risk of a recurrence of barotrauma should be evaluated, as well as the consequences (sequelae) from the previous barotrauma. If an anatomical cause has been detected (sinus or nasal polyposis, laryngocele, tooth decay etc.), these should be corrected if possible; otherwise, the patient should be advised not to dive again. Equalization techniques and education as to the prevention of barotrauma should be taught to the patient, as most cases of barotrauma are preventable. In the case of inner ear barotrauma, there appears to be a high risk of recurrence, with most patients advised not to dive again. However, it has been shown that well-educated and careful divers can safely return to diving after inner ear barotrauma.

2.7. Take-home messages

Importance of immediate correct diagnosis: Most barotraumas to the ENT sphere are not difficult to diagnose with proper knowledge of basic principles of physics and adequate knowledge of anatomy and physiology of the organs involved. Though seldom life-threatening (pneumencephalon, laryngocele), these lesions may cause severe pain and permanent sequelae (hearing loss, vestibular deficit, facial nerve paralysis) and thus impact quality of life. Therefore, these patients should be treated as emergencies and competently evaluated in the shortest possible time span. Consultation with a diving medicine physician can be obtained free of charge and 24 h per day through the Divers Alert Network Europe (DAN Europe) telephone hotline (freephone number within Belgium: 0800 12382), or through the various emergency telephone numbers listed on the DAN Europe website (www.daneurope.org).

2.8. Specific cases

2.8.1. Flying, parachute jumps and train conductors

Although most barotraumas will occur during specific activities, such as underwater (scuba) diving or commercial air travel, other categories of persons exposed to pressure changes may be victims of such lesions. Such changes could result from free–fall parachute jumping, sports flying in unpressurized aircraft and even high-speed train travel (when entering or exiting a tunnel or when passing other trains, the Bernouilli effect creates a depressurization around the train, lowering the inside pressure rapidly over a range of up to 100 mbar, thereby possibly causing ear discomfort).

2.8.2. “Fitness to dive” evaluation

When evaluating candidates for diving activities, care should be taken to verify:

- the visual aspect of the eardrum
- permeability of the nasal meati and ET
- any history of recurrent or chronic sinusitis or otitis media
- the state of dental health
3. Syndromes caused by pressure changes in specific ear pathology: third mobile window lesions

3.1. Introduction

Several inner ear conditions render the patient prone to temporary or permanent trauma or baro-challenge-induced symptoms, including congenital inner ear malformations, such as the large vestibular aqueduct syndrome (as also observed in Pendred syndrome), jugular bulb abnormalities or an absent modiolus (as found in X-linked Gusher syndrome). Otosclerosis patients who underwent stapedotomy are also at risk from trauma or baro-challenge. Additionally, other inner ear conditions can be acquired after (baro-)trauma, including perilymphatic fistula and superior semicircular canal dehiscence syndrome.

3.2. Symptomatology

Symptoms related to these kinds of third mobile window lesions can present as isolated or in any possible combination. Auditory symptoms include conductive and sensorineural hearing loss, but typically involve low-frequency air-bone gap and supranormal bone conduction thresholds at low frequencies. With regard to the latter, patients become hypersensitive to bodily sounds, which may result in autophony, pulsatile tinnitus (venous hum) or hearing one’s eyeballs, joints or footsteps in a disturbing way. Vertigo spells can occur and are usually triggered by sounds or baro-challenge (e.g., Valsalva, pressure changes in the external auditory canal).

3.3. Pathophysiology

The pathophysiology of the third mobile window has been studied extensively in animal models. The fluid spaces of the inner ear are enclosed in the petrous bone; to be more specific, the otic capsule. The round window and the oval window present a functional barrier between the fluid-filled inner ear and the air-filled middle ear. The cochlear and vestibular aqueducts link the inner ear to the cranial cavity. The vestibular aqueduct connects the endolymphatic space to the endolymphatic sac, which is positioned on the back of the petrous bone. Several smaller foramina enable the passage of blood vessels and nerves. Since fluid is incompressible, the mobile round window enables the stapes to produce a fluid motion (and subsequently a basilar membrane motion) in the cochlea, which eventually leads to the perception of sound. Whenever a “pathologic” third mobile window is created, air-conducted sound energy is directed away from the cochlea, which produces functional hearing loss. Hypersensitivity to bodily sounds can be explained by the lowered impedance on the scala vestibuli side, which results in an increased pressure difference between the scala tympani and scala vestibuli, and thus increased perception of bone-conducted sounds.

3.4. Evaluation and management

Whenever symptoms related to this pathologic third mobile window occur after (baro-)trauma, the patient needs to be referred to an otolaryngologist for further evaluation using audiometry, speech audiometry, electronystagmography (vestibular function testing) and cervical vestibular-evoked myogenic potentials (cVEMPs). High-resolution computed tomography of the temporal bone and magnetic resonance imaging are imaging modalities used to respectively visualize the integrity of the bony capsule of the labyrinth and the inner ear fluid spaces. The pitfalls of computed tomography include partial volume averaging effects, which may result in false-positive findings of dehiscence, which are provoked by slices that are not thin enough or because the bony capsule is thinner than the slice collimation. Therefore, the diagnosis of a third mobile window lesion can only be suggested after confirmation by means of electrophysiological testing. Increased responsiveness of the labyrinth can be demonstrated by eliciting the cVEMP. Air- and bone-conducted sounds trigger the saccular afferents into stimulating the ipsilateral sternocleidomastoid muscle through the sacculo-colic projection. Muscle contraction is measured by using surface electromyography electrodes. Given that the otolithic input to the sternocleidomastoid muscle predominantly originates from the saccular macule, the elicited cVEMP is not affected by sensorineural hearing loss. The increased amplitude of and/or decreased threshold for eliciting of the p13-n23 response on the ipsilateral sternocleidomastoid
mucosal muscle may confirm increased responsiveness. In cases involving bilateral third mobile window lesions, cVEMP testing can be helpful to determine which side is more symptomatic. If the cVEMP response is normal, the potential diagnosis of a mobile third mobile window lesion has to be reconsidered. Positive CT and cVEMP findings are also possible without a typical history of a third mobile window lesion.

Where post-traumatic third mobile window lesions producing incapacitating symptoms are involved, surgery can be offered in selected cases after thorough evaluation. Especially in cases involving the surgical management of superior semicircular dehiscence syndrome, high symptom control rates have been reported. A schematic representation of temporal bone computed tomography in superior semicircular canal dehiscence can be found in Figure 4. The preoperative identification of perilymphatic fistula is much more difficult and controversial, although obliteration of the oval and round windows has been reported to resolve incapacitating symptoms.

In cases where there is a history of stapes surgery, however, third mobile window symptoms are more likely to be caused by perilymphatic fistula, prosthesis dislodgement or perforation of the vestibule. For this reason, stapes surgery is generally considered to be a contraindication to an aviation career, although extensive evaluation in order to determine a patient’s fitness to fly with a waiver may allow for a safe recovery of aviation activity.

3.5. Pre-hospital assessment

Third mobile windows generally do not present as a life-threatening condition, although their symptoms may be reported as incapacitating. Pre-hospital assessment should focus on potentially life-threatening conditions in the first instance.

3.6. Assessment and management at the hospital

The physician should be aware of the existence of third mobile window lesions presenting after trauma and inquire after symptoms, such as hearing loss, hypersensitivity to bodily sounds (which may result in autophony, pulsatile tinnitus or hearing one’s eyeballs, joints or footsteps in a disturbing way) or vertigo spells triggered by sounds or

![Figure 4](image)

Temporal bone computed tomography, multiplanar reconstruction in the axis of the superior semicircular canal. 1 = round window; 2 = oval window; 3 = dehiscence; A = right superior semicircular dehiscence without annotations; B = right superior semicircular dehiscence marked at 3; C = normal left superior semicircular without annotations; D = normal left superior semicircular with annotations.
baro-challenge. A significant problem occurs in diagnostic delay by not identifying a potential third mobile window lesion. If possible, otoscopy, video-oculoscropy, tympanometry and audiometry should be performed at once to identify sensorineural hearing loss. Vestibular function testing and imaging are also indicated.

3.7. Take-home messages

Several presentations – both syndromic and non-syndromic – of third mobile window lesions exist. They pre-exist in some individuals and are not always symptomatic as such. When a patient presents with hearing loss or vertigo after cranial trauma or barotrauma, the possibility of third window lesions as underlying pathology must be considered during the differential diagnosis.

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