MECHANICAL ASPHYXIA

The asphyxia **can be defined as a process characterised pathological changes in the air passages that are determined by lack of oxygen, with consecutive hypoxia and hypercapnia.**

Although useful, this definition seems limited by the fact that it suggests only some forms with visible pathological changes.

The modern concepts try to correlate the term asphyxia (greek "a sphygmos") with the modern concept of hypoxia or anoxia.

The term *mechanical asphyxia* is deliberately limited to the death or the sub-lethal trauma resulting from a mechanically induced cerebral hypoxia, accompanied by physical signs and the obstruction of venous return.

So, the notion of mechanical asphyxia only concentrates on those types of violent deaths that appear consecutive to cerebral hypoxia caused by a mechanical trauma.

The aetiology of this entity includes:

- ***the air passage obstruction;***
- ***the interference with the cervical vital structures***
- ***the low concentration of oxygen in the breathed air.***

From the forensic point of view, asphyxia can be classified in:

- ***pathological asphyxia's*** **– hemopathies, cardiopaties, tumours.**
- ***mechanical asphyxia (violent):***
  - **compression:** ***- hanging***
    - ***strangulation with a ligature or by hand***
    - ***non-specific cervical compression***
    - ***thoracic compression***
  - **postural asphyxia**
  - **autoerotic asphyxia**
  - **occlusion of air passages:**
    - ***suffocation***
    - ***foreign objects in the air passages***
    - ***drowning (submersion).***

PATHOPHYSIOLOGICAL MECHANISMS IN MECHANICAL ASPHYXIA

For a better understanding of the complex phenomenon of asphyxia, it is essential to bear in mind that the vascular system is a closed circuit, so a certain degree of hypoxia in a perfused organ can be obtains either by reducing the blood flow, or by obstructing the venous drainage.

The first organ that has to suffer from anoxia is the brain, because it is the less resistant to the lack of oxygen.

Hypoxia, the consecutive hypercapnia and the excessive increase of lactic acid concentration will determine an amplification of the intra-capillary pressure, followed by oedema and local vasodilation.

In the initial phase of asphyxia, the vital organs intensify their activity.

In the same time functional alterations such as profound breathing, tachycardia and high blood pressure occur, as a result of the hypoxic excitation of the sino-carotidian node.

These changes are followed by a progressive deterioration of all vital functions, up to their total collapse.

The asphyxic process has a rather typical evolution that follows four distinctive phases:

a. The pre-asphyxic (latent) phase **– approximately one-minute long, with a predominantly nervous simptomatology with severe inspiratory dyspnoea that will finally be followed by loss of consciousness;**

b. The compensated (convulsive) phase – convulsions, consciousness alterations, expiratory dyspnoea, lack of reflexes, sometimes asphyxic ejaculation;

c. The decompensated (asphyxic) phase **– severe alteration of vital function, followed by apnoea, primary or secondary to the cardiac arrest;**

d. The final phase **– heart survival after the respiratory arrest.**

The duration of this process varies with the type of asphyxia and its aetiology:

- after 20 seconds the cerebral circulation stops

- after one minute the consciousness is lost

- after 3 minutes clinical death is onset and

- after 8 minutes the electrical death of the heart occurs.

The force **applied to the neck does not necessarily have to be strong to produce the obstruction of various cervical tubular structures. Although the experimental data are old, they are still extremely significant:**

Author	Jugular veins	Carotid arteries	Trachea	Vertebral arteries
Brouardel (1897)	2	5	15	30
Reuter (1901)		3,5		16,6
Ranschke (1957)				
The smallest tension necessary to obstruct all cervical structures:				5-10
The highest necessary tension to obstruct all cervical structures:				20-40

The necessary force (kg) for the obstruction of all cervical structures (Purdue)

The duration of the force necessary for death onset due to alterations of the cerebral oxygenation mechanism can be influenced by:

- the degree of interference with oxygenation;

- restoration speed of the involved organism

- the nature of the applied force

- the oxygen consumption ratio

- the subject's age

- possible alcohol or drug abuse

The survival time of a completely anoxic brain, in average environmental conditions is around 4-5 minutes.

The only condition for a rapid death is the existence of a self-sustaining mechanism able to deprive the brain from blood and oxygen.

An external ligature insures such conditions, for example, but also by inhibitory reflex-like mechanisms, that impede a normal cardiac function. Such mechanisms are:

- *the instantaneous neurogenous cardiac arrest*

- other forms of cumulative cardiac disorder

The interference with the vital cervical structures can determine cardiac fibrillations, thus reducing the cardiac flow (Rossen).

The common final result is the irreversible arrest of the pump function, that is the actual cause of death in the manual compression of the neck.

GENERAL MORPHO-PATHOLOGY IN MECHANICAL ASPHYXIAS

The notion of „asphyxic signs" includes a category of objective changes that can be recognised and part of mechanical asphyxia.

Two forms of general asphyxic signs can be distinguished from the anatomo-pathological point of view.

They are nothing else but physical manifestations of the venous return blockage.

The spectrum of pathological changes ranges from gross asphyxic signs and obvious contusions, to simple diffuse petechiae and vague traumatic traces/signs.

General external asphyxic signs include a series of important changes, such as:

- *cyanosis,*
- *soft tissue swelling,*
- *petechiae (cutaneous and mucous),*
- *wide lividities*
- *hypoxic effects.*

Specific external signs are represented by those changes that confirm:

- *the occlusion of the respiratory orifices*
- *the compression of cervical tubular structures*
- *the thoracic compression.*

GENERAL EXTERNAL ASPHYXIC SIGNS

1. Cyanosis

The dark, red-blackish blood observed through the pale skin gives a cyanotic aspect to the face and other body parts.

At close inspection, the congestion of the checks, conjunctivas and labial mucous membrane can be observed.

The facial skin has a bluish, spoted discoloration, more accentuated in suffocation, strangulation and thoracic compression and reduced or even absent in some hanging cases.

Peripheral cyanosis is a common finding in almost all cadavers and it is definitely not considered a marker of ante-mortem asphyxia.

2. The swelling of soft tissues

As a result of the blocked venous return circulation in the head and cervical region, veins become congestive, especially those of the face, neck and viscera.

The increased hydrostatic pressure determines oedema in the soft tissues.

The results are a congestive swelling of the face and tongue and the distension of the orbital content, resulting in exophthalmia.

3. External petechiae (cutaneous or mucous)

External petechiae are venous haemorrhages onset in permeable tissues, where they can be observed as small dark-reddish spots on the skin or mucous membranes.

Some petechiae can be more intense, macular or purpura-like, located sub-conjunctival.

External petechiae usually appear on the periorbitar skin (mask or butterfly-shaped), on the conjunctivas, on the skin behind the ears, on the face and neck, on the bucal and epiglottis mucous membrane, etc.

They cannot only be observed in cases of mechanical asphyxia, but also in other types of cases, as a result of severe increase of intra-thoracic pressure (asthma-like seizures, cardiac failure, respiratory failure, defecation effort).

So, the increased venous pressure is indispensable for the onset of conjunctiva or facial petechiae, regardless of the cause of death – asphyxic or non-asphyxic, pathological nature.

Conjunctival or facial petechiae are common signs of cephalic venous compression and cannot be regarded as absolute proves for an asphyxic mechanism.

4. Extensive lividities

Early onset of extensive, darker lividities (livor mortis) can be observed in most cases of mechanical asphyxia. Their appearance is dark-violet.

This particularity can be explained by the higher degree of blood fluidity that allows the blood to easily diffuse in the tissues.

Although typical, the lividities do not necessarily represent specific changes of mechanical asphyxias, since they also appear in other types of pathological or violent deaths.

5. The effects of hypoxia

The hypoxic syncope requires special attention.

Cerebral hypoxia and the consecutive hypercapnia can induce specific effects, such as: vomiting, urinary or faecal incontinence etc.

These are central, cerebral reflexes that can also be notices in pathological deaths.

The cerebral circulatory arrest induces unconsciousness, usually after approximately 10 seconds.

Consciousness restoration mostly depends on the reset of cerebral perfusion in the 4-5 minute limit interval during which the cerebral tissue is almost immune to the hypoxic state.

GENERAL INTERNAL ASPHYXIC SIGNS

1. Blood fluidity

Both the peripheral and the central blood is usually fluid, red-blackish, due to the increased concentration of carbon dioxide during the asphyxic process, followed by the increase of fibrinolithic activity and of some anti-coagulant factors.

2. Visceral congestion

Visceral congestion is an inconstant sign with no special significance, because it can also be observed in other types of death.

A typical, classical but inconstant, asphyxic finding is the reflex spleen constriction, an immediate compensatory reaction of hypoxia.

3. Visceral petechiae

Internal petechiae are small, dark-red spots, with a diameter up to 1-2 mm. Their main location is sub-pleural, on the surface of the lungs, at their base and between the scissure.

This blood suffusion can also be observed on the mucous membrane of the respiratory tract, in the depth of parenchyma organs or on the epicardium, thymus and rarely on the intestinal serous membrane.

The mechanism of petechiae onset starts without the capillary overload, followed by the capillary and intra-alveolar blood pressure.

Some capillaries cannot resist to the increased pressure and perforate, especially in the pleural cavity.

4. Acute pulmonary emphysema

The pulmonary emphysema can be a specific sign of mechanical asphyxia. At autopsy, the surfaces of the lungs display pale, distanced areas, prominent in the pleural cavity and are easily depressed.

The emphysematous lungs cover the pericardium with the anterior margins, are pale-bluish to pink-greyish, with a certain degree of anaemia; in cases of slow asphyxia, they are intense hyperaemic.

If other causes of deaths are overruled and but other elements suggest asphyxia, the pulmonary emphysema can be a relevant sign.

Pulmonary vessels are intensely dilated, blood-flooded; some areas are haemorrhagic, with blood in the alveoli.

5. Pulmonary oedema

Is an inconstant sign; when accentuated, it might indicate a prolonged agony. When the oedema is local, it usually is post-mortem and must not be mistaken with the real, generalised oedema.

6. The dilatation of the right heart

In some cases of mechanical asphyxia, the autopsy shows a dilatation of the right heart; it also appears filled with liquid, blackish blood, because of accentuated pulmonary stasis.

7. The haemorrhage in the temporal muscles

It appears in the shape of macular haemorrhagic spots, either due to intensive congestion or to capillary ruptures.

COMPRESSION ASPHYXIA

1. HANGING

Hanging involves the total or partial suspension of the body, intermediated by a ligature fixed around the neck, with consecutive compression due to the entire or a part of the body weight.

Most hanging cases are suicides, rarely accidents and very rarely homicides.

Hanging is considered ***typical*** when the superior part of the ligature is situated in the occipital area and ***atypical*** if the superior part of the ligature is situated in any other area (anterior, lateral).

It is considered ***complete*** if the body is completely suspended and ***incomplete*** if the body has one or more contact points with the grounder any other sustaining plan.

The ligature can be: soft (scarf, shall), semi-rough and rough (cables, wires).

The ligature can be unique – with unique, double, triple or multiple turns, depending on the number of times it is circled around the neck.

The knot is extremely important because in many situations it offers indications regarding the profession of the victim or the assailant.

Hanging: typical, with occipital knot, and atypical

The asphyxic mechanism in hanging

The cervical traction exercised by the ligature applies pressure on the cervical region, proportional with the weight of the suspended body.

Three main mechanisms are involved in the death-generating syndrome in hanging:

- haemo-dynamic;

- asphyxic;

- nervous, represented by the excitation of the sino-carotidian node or of the vagal nerves, in cases of brutal compression of the neck, when instantaneous neurogenic cardiac arrest (INCA) can onset/occur.

Depending on the mechanism involved, the macroscopic findings during the autopsy will differ.

In subjects with predominant cerebral haemo-dynamic alterations cephalic cyanosis will be marked due to intense stasis (*blue asphyxia*).

The asphyxic mechanism will determine moderate cyanosis and the protrusion of the tongue that appears fixed between the teeth;

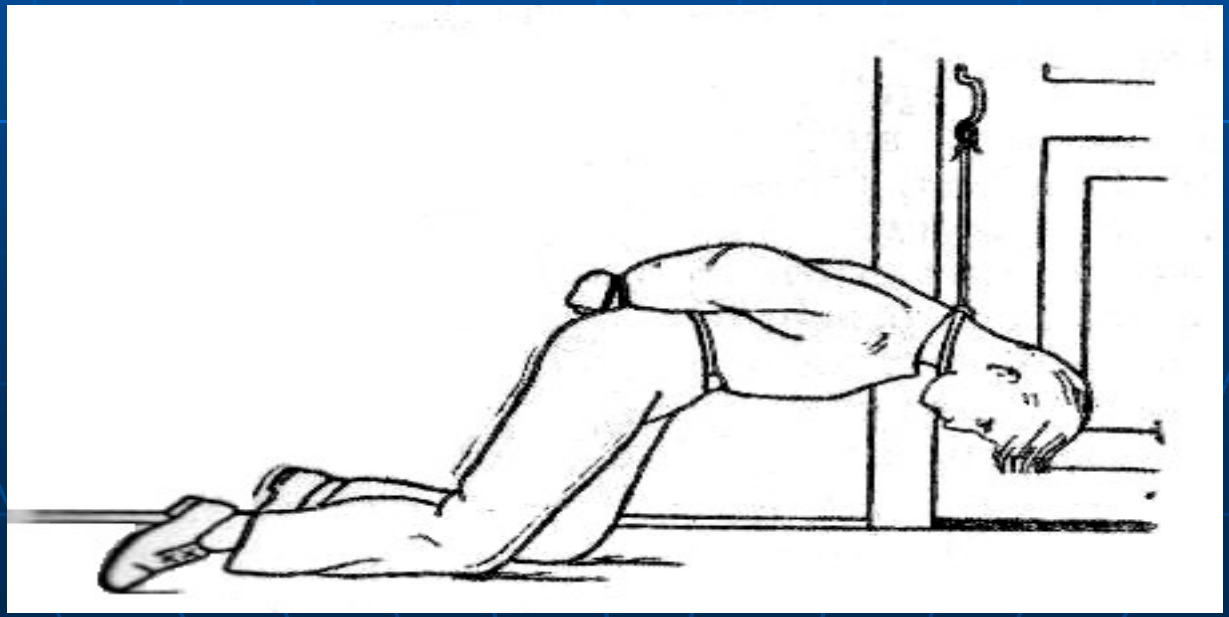
Subjects deceased due to nervous reflex mechanism are pale (white asphyxia).

a. Incomplete hangings **the stress is mainly exercised upon the cervical vascular structures and only in a smaller degree or not at all on the air passages, so death can be explained using experimental data regarding the necessary compressive force.**

In such cases, a certain degree o transport anoxia to the brain onsets; it can finally lead to death (Fig. 2).

If the body touches the ground, pressure on the ligature is in direct relation with the position of the pressure point on the neck; the lower it is situated, the more accentuated the hypoxia signs are.

Cardiac activity is obligatory for the signs of asphyxia to onset. INCA cannot be incriminated in a hanging with low cervical suspension point, while cumulative cardiac insuficiency (CCI) can.



b. Complete suspension **is the elective method of most hanging suicides. In these cases, the whole body weight presses on the ligature. This method is certainly fatal, with the exception of cases in which the attempter gives up in the first few seconds while still conscious, or is saved in time.**

External asphyxic signs are less evident or even absent and if such is the case, the face will be strikingly pale, with serene features.

A body suspended in a ligature exercises high pressure to the neck, and thus impedes blood from reaching the head. Most deaths by complete hanging onset in 4-5 minutes, while the loss of consciousness in 10 seconds.

Bradicardia and ventricular fibrillation are typical for hanging without falling; this suggests that CCI appear in association with cerebral ischemia during complete suspension. In such situations the rapid loss of consciousness onsets first, followed by a gradual interference of cardiac failure and prolonged cerebral ischemia.

A frequent sign is the spontaneous semen emission. This is a controversy element because it is either considered a post-mortem phenomenon in connection with the onset of rigor mortis in the seminal vesicles, or a vital phenomenon, result of pelvic reflex congestion or of a reflex act generated by the excitation of a cervical reflexogenous area.

The morpho-pathology of hanging lesions

I. External lesions

a. Tongue protrusion **– is a characteristic of hanging at any level of suspension. The tip of the tongue is often dark-brownish, dry and the anterior extremity of the tongue appears captive between a firm dental grips. The explanation is that the ligature lifts the gloso-laringeal skeleton, compressing the tongue and pushing it forwards.**

b. The lividities **are widespread and intense. They can sometimes suggest the hanging by their localisation on the inferior limbs, but only if the body hung for at least 14-16 hours, so that the lividities can become immovable.**

c. The hanging ligature mark **or the *ligature groove* is the typical marker of hanging and the most important element in this type of mechanical asphyxia. It has a deeper portion around the area of maximum pressure or all around the neck. Its width is variable and it is ascendant towards the suspension point.**

Reported to the thyroid cartilage the ligature mark can have a superior, medium or inferior position; this positioning will dictate the predominant death-generating syndrome (haemo-dynamic, asphyxic, neurogenous). In complete hangings the ligature mark most of the times situated in the superior part, while in incomplete hangings it will be medium or inferior.

The orientation and direction **of the hanging ligature mark is always typical, oblique and ascendant towards the knot, when the ligature is simple (with one turn).**

The shape of the ligature mark varies from case to case, depending on many factors. There are two main types of hanging marks: *incomplete* around the neck and *complete*, circular.

Because the ligature is suspended in a fix point, the ligature mark does not interest the whole area of the neck, but it is interrupted in the area corresponding to the knot. In some cases this area will reveal the knot pattern, mainly if it voluminous.

The dimensions of the hanging ligature mark are in direct relation with the texture of the ligature. The maximum depth of the ligature mark is always situated opposite to the knot, where the compression force is maximal. The ligature mark can sometimes reproduce the pattern of the ligature in amazing details.

The consistency of the ligature mark varies with the material of the ligature. Thus, rougher ligatures make the skin dehydrate, so the ligature mark will be rougher, parchment-like. Softer ligatures will determine a softer mark, only slightly more consistent then the adjacent tissues.

The colour of the ligature mark is generally brown, especially when it is deeper. In other cases, the skin becomes yellowish, brownish or orange.

II. Internal lesions

- can be observed in the tissues subjacent to the mark. An important element in the investigation of a hanging case is the microscopic examination of the tissues adjacent to the mark. It can reveal small haemorrhages as subcutaneous infiltrates and blood stasis and emulsion in the shape of small drops in the adipose tissues.

Thyroid cartilage and/or hyoid bone fractures can be noted, usually with focal haemorrhage; this is a positive sign of intra-vitam hanging. The traction and elongation can determine fissures or ruptures of the carotid intima (Amussat sign) and haemorrhages in the peri-laringeal tissues.

The internal organs display accentuated stasis with liquid, dark-coloured blood. The lungs present acute marginal emphysema, sometimes associated with sub-pleural petechiae.

Hanging with falling of the body is a well-known execution method; it is also used for suicidal purposes. The ligature is fixed to one end; the other end is placed around the victim's neck. The body is left to fall by opening a flap door under it or by removing the support from under the feet. In these cases the death mechanism is usually non-asphyxic, because the body weight exercises a sudden traction force upon the neck, which leads to cerebral trunk ruptures or even to its separation from the marrow.

2. LIGATURE STRANGLING

Ligature strangling is consists of applying a mechanical compression to the cervical region; as a result, the air and/or blood passage through the tubular structures of the neck is blocked.

The death mechanism is mainly the acute cerebral anoxia.

The difference between hanging and ligature strangulation is that in the first the compressive force is passive, produced by the body weight, while in the latter it is active and it depends on the strength of the aggressor.

The face, cervical area and the superior third of the thorax are usually cyanotic, with millimetric petechiae and subconjunctival haemorrhages.

Often a pink foamy matter appears in the nostrils and the tongue is fixated between the teeth.

The cervical area displays the strangulation mark or the ligature sign; it is necessary to assess its position and nature.

A classic strangulation ligature mark is horizontal on the neck, inferior to or on the larynx, complete and less profound.

The ligature signs usually include linear erosions that can sometimes reproduce the shape of the traumatising agent.

Often they form a clear delimitation (tide mark), above which the asphyxiation signs are accentuated, offering a specific image.

The lesions revealed during the autopsy of the cervical area less numerous and less characteristic then in other types of asphyxia.

In the same plane with the ligature mark there can be bruises and haematomas of the antero-lateral cervical muscles, sometimes even a circular bruising of the carotid adventitia.

The Amussat lesions are somewhat more frequent then in hanging.

Suicide by ligature strangulation is very rare, but possible, special attention is recommended in order to avoid superposing with homicide markers.

Accidental ligature strangulation is rare but possible, for instance by catching a scarf in a running mechanism.

Children are at high risk of involuntary strangulation with different ropes, for example the swing ropes.

3. MANUAL STRANGULATION (THROTTLING)

In the fatal throttling, such pressure is applied to the neck that, combined with enough strength and duration, determines the victim's death. The death mechanism can involve asphyxia, cumulative cardiac insuficiency (CCI) or a combination of both. A large range of medium to severe cervical lesions appears.

Superficial lesions are usually milder then the profound ones.

Typical hand strangulation signs include round or oval, fingertip shaped bruising, of 1-2 cm diameter each. They can be unique or multiple, isolated, grouped or confluent.

Semicircular excoriation produced by the nails can appear on the edge of these bruises or isolated. The criminal or the victim can either inflict them, in an attempt to escape the attack.

If a struggle takes place, other lesions can appear on the body. Throttling is often associated with sexual assault, so the use of laser, UV light, sampling for bio-serological examination and a thorough search for evidence must follow photographing the scene and precedes the examination of the body.

Ideally, samples should be taken from all the victim's sexually penetrable parts, in hope of discovering seminal liquid of either sex.

Photography from oblique-lateral incidence is also needed in order to visualise the laryngeal profile.

A meticulous examination of all asphyxic signs, especially petechiae is also necessary.

4. NON-SPECIFIC CERVICAL COMPRESSION

The term reunites all non-specific causes of cervical compression, both sudden and strong, or weak and prolonged.

Strong sudden compressions are: hitting the neck with the hand or a blunt object or jumping on the victim's neck after it is down.

Any of these forms of violence can induce severe lesions of the soft tissues, hyoid bone and larynx.

The lesions that appear are never present in hanging: vertical fractures of the thyroid or cricoid cartilage, or of the superior tracheal ring.

Crushing the larynx on a rough plane – the vertebral column, produces them.

Death can be rapid, with crush lesions and blood infiltrates; it can also be cause by severe oedema of the air passages, followed by asphyxia.

Cerebral ischemia due to vascular occlusion is not involved in these short-time pressures to the neck.

Mild, prolonged pressure to the neck usually involves manual pressure, like in man-to-man struggles.

It is to be reminded that any pressure applied to the neck can determine CCI or an aggravation of the vascular permissiveness.

5. THE THORACIC COMPRESSION ASPHYXIA

Mechanical asphyxia by thoracic compression can appear in a multitude of situations, such as:

- traffic accidents (collisions), if the victim is projected outside the vehicle or remains blocked in the vehicle;

- occupational hazards in industry or construction;

- earth-quakes or other tectonic movements;

- the crumbling of a building or a mine gallery

- human crowding, when the persons that fall are stepped over or end up under a pile of fallen bodies.

The intra-thoracic pressure that rapidly increases over the the venous level affects the respiratory movements and the cardiac activity.

The blood cannot re-enter the thorax, being thus forced to return.

Under these circumstances, numerous post-capillary venules brake under high hydrostatic pressure and produce marked physical signs of high venous pressure.

Confluent subconjunctival haemorrhages determine a red, shiny sclera, swelled, purpura-like face – typical aspect even in survivors.

Multiple rib, sternum, clavicle and even spine fractures can coexist.

Venous high-pressure signs are absent in prolonged, low intensity thoracic pressure.

6. POSTURAL AND POSITIONAL ASPHYXIA

Postural asphyxia appears in special circumstances and involves two components. First, the coughing reflex has to be abolished by known and provable causes like alcohol or drug abuse, bulbar paralysis or traumatic loss of consciousness. Secondly, the breathing capacity has to be mechanically compromised. The death is always asphyxic, never neurogenous. The interpretation of the death-generating syndrome in subjects forced to stay in up-side-down position raises special problems.

The forced restriction of thoracic expansion, without aggressive human intervention, can interfere with breathing if it is sufficiently intensive and prolonged. The phenomenon can turn into crush asphyxia if the pressure involved grows in intensity.

7. AUTO-EROTIC ASPHYXIA (SEXUAL ASPHYXIA)

Autoerotic asphyxia is characterised by:

- ***the deliberate induction or amplification of orgasm by self-asphyxiation;***
- ***bondage, most frequently on the limbs, neck and genitalia;***
- ***use of mirrors, photo or video cameras;***
- ***secrecy, locked doors;***
- ***sexual deviations, often sadomasochism or transvestism;***
- ***presence of pornographic materials around the deceased;***
- ***evidence of repeated autoerotic practices.***

Most victims of autoerotic asphyxia are male. In fact few female cases are reported.

Death occurs due to a dangerously prolonged practice, with consecutive loss of consciousness.

The victim becomes unable to self-rescue, so the autoerotic asphyxia is rather accidental then intentional.

The pathological aspects are various, depending on the type of asphyxia.

Hanging can determine congestion or pallor, depending on the location of the suspension points.

If the victim uses a gag the body will display florid signs of asphyxia, while in suffocation with a plastic bag the findings are minimal or even absent.

8. OCCLUSION ASPHYXIA

I. The occlusion of the respiratory passages (smothering)

The mechanical asphyxia by occlusion refers to the blocking of the nose and mouth with various objects, such as: the aggressor's hands or soft fabrics (pillows, clothing), that adept to the profile of the face.

a. Manual suffocation **is possible because the hands of an adult can cover and compress the face with sufficient intensity as to produce the total obstruction of the respiratory orifices.**

b. Suffocation with soft materials (smothering) **is more often used as means of violent asphyxiation. Because the death-generating process is prolonged (8-10 minutes), the external and internal general signs are accentuated.**

The examination of the victim reveals cervico-facial cyanosis, subconjunctival haemorrhages and bluish discoloration of the nails.

In palmar suffocation, abrasions around the mouth and nose can be noticed. In addition, the vestibular face of the lips can present bruises and even wounds, due to their crushing on the teeth.

Visceral, especially pulmonary, congestion can appear. The lungs can also display areas of acute emphysema associated with accentuated haemorrhagic oedema and subpleural, lenticular, well-delimited haemorrhagic suffusions (Tardieu asphyxic spots).

They are intense reddish, sometimes brownish, variable in number, often grouped near to the hillum, inter-scissural and at the base of the lungs.

They can also appear on the parietal or diaphragmatic pleura or pericardial. An important feature of the Tardieu petechiae is that they are resistant to putrefaction, so they can be observed even months after death (Derobert).

In some case of smothering with textile material, fibre or small textile fragments can be found in the mouth or nostrils; they can raise suspicions and orientate the investigation.

The forensic diagnosis of occlusion asphyxia must take in consideration the possibility of homicide, suicide or accidental death.

Homicide is the most frequent situation. It is labelled as such by corroborating the specific signs of asphyxia with other particular findings regarding the suffocating agent, external signs of violence on the face, and on the rest of the body, because of the struggle between the victim and the aggressor.

Suicide is extremely rare, but accidental suffocation is more frequent and can appear, for example, in subjects in an advanced state of drunkenness that fall face down on various soft materials or in suckling, during their sleep, due to accidental covering of the face with soft materials (pampers, bed covers etc.), or plastic material.

c. Some authors consider placing a plastic bag on the head **a plausible suicide option, often combined with alcohol or drugs overdose.**

The signs of asphyxia are vague or even absent. Typically, the face is pale, with a relaxed, sleep-like expression.

Usually condensed liquid is found in the interior of the bag, regardless if the victim is deceased or still alive. This element cannot be used to sustain that the victim was alive when the bag was placed on the head, because the humidity can result from perspiration but also from dehydration and post-mortem heat loss.

The death-generating mechanism is yet insufficiently known. Some deaths can be due to cardiac arrhythmias, probable induced by the carbon dioxide narcosis, others due to CCI that seems more probable a mechanism then INCA.

The differential diagnosis of smothering in child victims is one of the most difficult in all forensic practises. The major similarity is with the sudden infant death syndrome (SIDS). A meticulous autopsy, sustained with microscopic, radiological, toxicological, virusological and biochemical investigations solves most initial uncertainties.

In SIDS hypostasis is often accentuated, sometimes with surrounding pallor that is not necessarily a sign of suffocation, although this can also be the cause. The presence of textile fibres in the mouth or nostrils is sometimes of uncertain significance.

II. The occlusion of the air passages with foreign objects

a. The forceful introduction of foreign objects in the air passages

The foreign object forcefully introduced in the mouth is usually textile (gag). An improvised textile gag can be partially permeable for air; also the nasal air passage is free and capable to maintain a healthy victim alive.

The occlusion can be immediate, with rapid on-set of the asphyxic mechanism or prolonged, progressive, if the foreign object does not completely obstruct the air passages.

In such situations, the irritation of the air passages mucous membrane will be followed by oedema that completes the obstruction.

In case of vegetal seeds aspiration the occlusion of the air passages is favoured by the swelling of the seeds by imbibition with bronchial secretion.

In these cases, the occlusion is determined by three components:

- **foreign object**
- **its swelling**
- **the oedema of the mucous membrane.**

In thanato-genesis, the asphyxic syndrome can be accompanied by a reflex mechanism (INCA), starting from the nervous terminations of the laryngeal mucous membrane that can trigger a fatal inhibitive reflex.

b. Accidental aspiration of foreign objects

This type of mechanical asphyxia can be observed in both children and adults. In all cases, the diagnosis is relatively simple, because the asphyxic signs are accompanied by the presence of the foreign object in the air passages.

Accidental aspiration of foreign objects is often associated with intoxication, most frequently with alcohol. The typical scene is of an alcoholic found face down in mud or other viscous matters.

c. Asphyxia with food bolus

This type of asphyxia involves the blockage of the glottis or trachoea with accidentally aspired food. These accidents usually occur during meals. The risk groups involved are:

- elderly subjects, with mastication or swelling disabilities;
- mentally handicapped subjects;
- subjects with bulbar paralysis or other swallow mechanism disorders;
- greedy eaters
- alcohol addicts;
- drug addicts.

The typical case involves intense coughing, eructation and aspiration of the food bolus. If the incident is severe, it will lead to death with clear signs of asphyxia.

In some cases death onsets silently, without the episode of paroxysmal cough. In these cases, the mechanism involved is INCA and the asphyxic signs will be absent; the aspired particle, regardless of its size, stimulates the neurogenic reflex and if it is small enough, it can escape the autoptic examination, even more so if death occurs with no witnesses present.

d. Asphyxia with gastric aspirate

This type of asphyxia is often associated with drunkenness. The subject has a full stomach and regurgitates in the larynx.

Because of the ethylic hypo-reflexivity the regurgitated matter is aspired in the larynx, trachea and bronchi.

The autopsy often reveals gastric content in the trachea but this is not a positive sign, since it appears in 25% of all deaths (agonic aspirate).

Presumptions can be made in the presence of an acid smell and a marked autolysis of the lungs.

The positive diagnosis requires either the presence of an obstruction of the air passages including the inferior one, with a significant quantity of gastric content, or the presence of microscopically detected gastric content in the bronchiole and alveoli.

e. Asphyxia due to haemorrhagic aspirate

Is an important cause of death that appears mostly consecutive to various acts of violence. The large quantity of inhaled blood can even obstruct large bronchia, determining the onset of asphyxic death with signs of venous return obstruction. The danger of blood aspiration can be emphasised by alcohol or drug abuse.

f. Irrespirable gases (confined atmosphere)

This form of asphyxia is encountered in workers that do not use protection masks in an atmosphere with dangerous gases.

The CO_2 narcosis involves specific phases: hypercapnia stimulates the cerebral receptors for CO_2 that will maintain a constant pH and blood gases correction during life.

It is probable that this highly specialised mechanism becomes deficient or overwhelmed in some situations.

The sudden excess of acid gases and the induces significant decrease of blood pH can represent severe stress factors, capable of inducing cardiac arrhythmias, that can be associated with breathing disorders, apnoea attacks and other disorders.

If mine gases are involved, the victim do not display asphyxic signs, which rather suggests a sudden, severe cardiac disorder then a breathing disorder.

III. Occlusion with liquids (Drowning)

Drowning is an acute asphyxia, characterised by broncho-alveolar flooding with various fluids, with subsequent irreversible lesions that rapidly lead to death.

The mechanism of drowning

The flooding of the air passages after submersion triggers a cascade of patho-physiological phenomena that can lead to cardio-respiratory arrest.

The inhaled water determines the decrease of the oxygen concentration and the increase of the carbon dioxide concentration, with consecutive acidosis. The entire death-generating process of drowning in water lasts around 3-4 min. and is developed in five distinctive phases:

- The surprise phase (5-10 seconds);

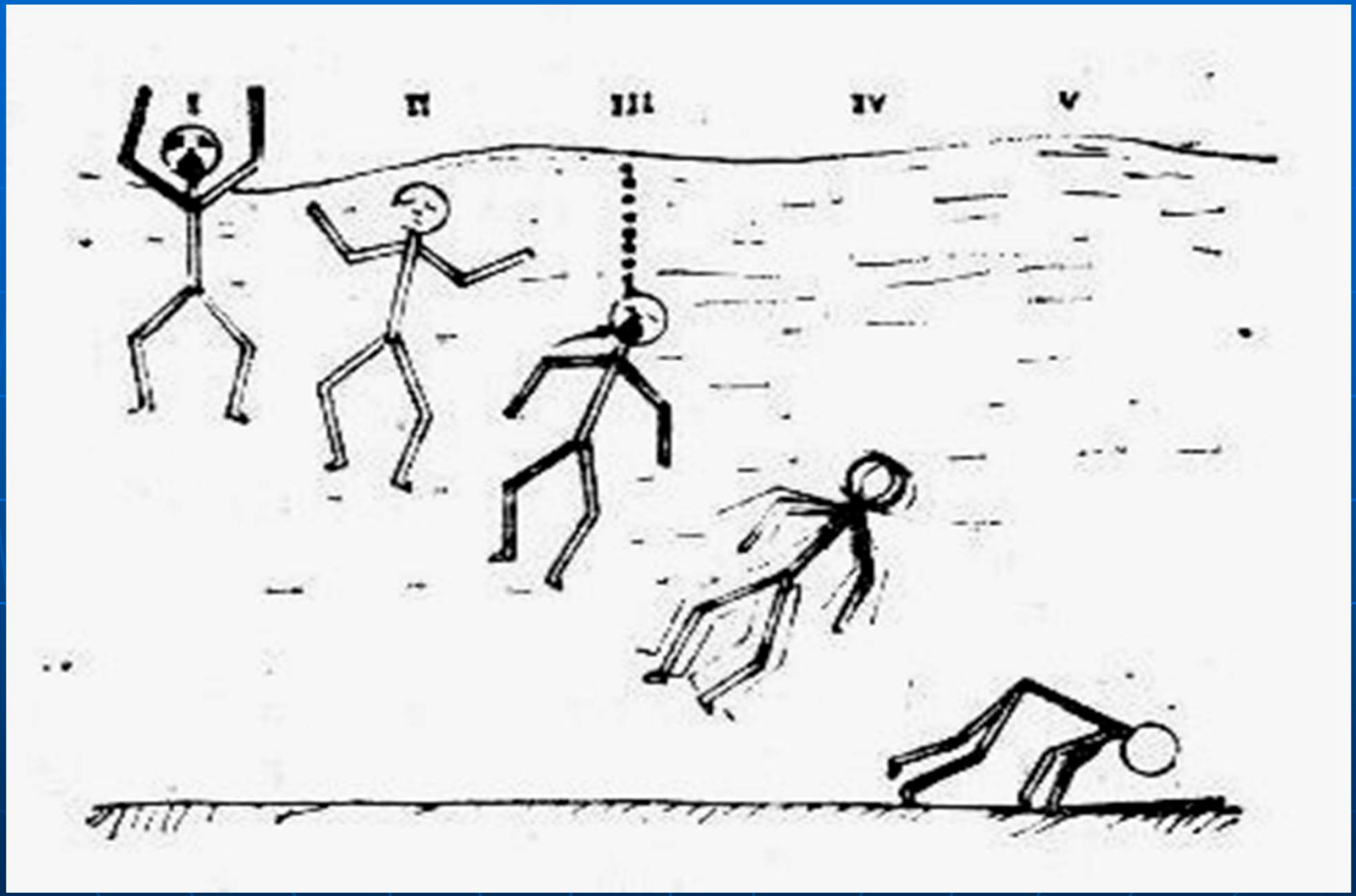
- The first respiratory arrest (1 minute), with agitation in the attempt to reach the surface;

- The phase of ample breathing, with abolished mobility (1 minute), associated with the apparition of foam at the nose and mouth;

- The second respiratory arrest (1 minute) with abolished sensitivity and the on set of hypoxic convulsions;

- Irregular, chaotic, terminal breathing (the last external vital sign).

Drowning in humans seems similar to the one described in experimental animals, although it may differ by more attempts to come to surface (Brouardel)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Death by drowning in sweet water on-sets in approximately 4 min., while drowning in salt waters lasts around 8 min.. Finally, the body falls at the bottom of the water where it remains for a period. It will be lifted to the surface by putrefaction gases, if it is not fixed on the bottom by various objects.

Water inflicts three types of aggression on the submersed body. They are:

- the anoxic process (air passages occlusion)
- the mechanical action (alveolar shock associated with alveolo-capilarry blockage and alveolar ruptures)
- the thermal action (decrease of body temperature).

Therefore, we can encounter drowning by blue asphyxia (anoxia) or by white asphyxia (reflex inhibition).

a. Blue asphyxia drowning can occur because of the water invasion in the air passages and later in the blood flow. The result will be the onset of typical asphyxic changes.

Sweet water is more hypotonic than the blood, so it will cross the alveolar walls into the capillary vessels, with consecutive haemo-dilution.

The quantity of water that crosses this barrier is so big that in 3 minutes it represents half of the total blood volume.

The haemo-dilution is accompanied by an increased blood volume and potassium release.

The concentration of serum electrolytes decreases dramatically, except for potassium that shows a slight increase.

The result is severe haemolysis and ventricular fibrillation.

In sweet water the cause of death is frequently the ventricular fibrillation (due to the potassium released by haemolysis), or the hypoxia associated with cardiac failure by volume overload (Spitz).

See water is hypertonic so the blood fluids will cross into the alveoli.

The consequence is pulmonary oedema, either serous or intense haemorrhagic. Rapid haemo-concentration and low blood volume, with decreased arterial and venous pressure accompany the acute pulmonary oedema.

In the meantime, the seawater ions diffused in the blood flow. So drowning in seawater is due to primary acute pulmonary oedema through shock and alveolar irritation. The cardiac arrest is caused by conduction disorders and progressive cardiac failure, after 6-8 minutes of submersion.

Both in salty and sweet water drowning the common factor is the fulminant pulmonary oedema, with liquid that contains serum proteins.

The mechanism of this type of oedema is extremely complex, because it involves a multitude of factors.

This factors are:

- haemodynamic: circulatory overload (in sweet water), pulmonary hypertension, arterial hypertension (as a result of hyper-capnoea and convulsions);

- humoral: hypoproteinemy due to haemodilution;

- local: alveolar blood escape (in salt water), increased superficial alveolar tension, bronchiolar spasm.

Considering that the on-set of circulatory arrest takes 3-6 minutes and the on-set of irreversible cerebral lesions 4-5 minutes, we can estimate that a submersion of 7-10 minutes is fatal.

b. White asphyxia drowning **has a mechanism similar to the primary syncope, either of medical (epilepsy, stroke) or of traumatic aetiology and it interests a reflex zone (solar plexus, scrotum).**

This type of drowning is also known as hydrocution.

It is realised through an inhibitive mechanism generated by stimuli from external receptors.

In a first phase, the death-generating process involves a reflex syncope, followed by secondary asphyxia.

Death by hydrocution is either due to syncope mechanical asphyxia, or to a thermal shock.

Specific morphologic changes for drowning

The aspect of a drowned body varies in direct relation with the duration of the submersion and the consecutive post-mortem changes.

A. The recently drowned body **is not decomposed, identifiable; it presents some morphological changes that constitute a characteristic ensemble.**

I. External signs

a. Facial cyanosis **is a blue-reddish discoloration, associated with conjunctiva hyperaemia or haemorrhage. The marked, extensive facial and body cyanosis can be the result of blue asphyxia. It is opposite to the wax-like pallor of the drowning victims with syncope or white asphyxia.**

b. The mushroom-shaped foam **is an important sign of drowning. It appears at the nose and mouth as a balloon of whitish foamy matter, formed of small bubbles.**

The foam is a mixture of mucus, proteins, alveolar surfactant and water inhaled in the phase of ample breathing. The foam is also present in the mouth and in the air passages and thoracic compression will render it evident at the mouth and nose. Sometimes it can be pinkish or reddish because of the presence of blood.

The foam appears after a 2-3 hour submersion and after the extraction of the body from the water, it can persist approximately 5 days during summer and 8 days during winter.

c. Cutis anserina (the goose flesh) **is a post-mortem change due to rigor mortis in the arrectores pilorum muscles. Low temperature waters favour it.**

d. The skin maceration **is determined by its imbibition in water. Initially it appears on the fingertips, and then it extends to the palms and upper-arms.**

The skin becomes moist, soft, wrinkled and whitish, with a typical aspect of „washing clothes hands". Similar aspects are present on the feet.

In temperate climates, these changes appear after 2-4 hours on the fingertips, and they extend up to the wrist in 24 hours.

II. Internal signs

a. Haemorrhages **in the sterno-cleido-mastoidian and pectoral muscles can be visualised during the autopsy. They can be the result of violent self-rescue attempts.**

b. The presence of foam **and/or inhaled extraneous matter in the trachea and bronchi (sand particles, mud etc.)**

c. Subpleural haemorrhagic suffusions **can be present in all forms of mechanical asphyxia, but because of haemo-dilution they are paler and more extensive than the Tardieu petechiae. The alveolar walls can be ruptured, with small subsequent haemorrhages; if they are located sub-pleural, they are called Paltauf spots.**

d. The lungs can present three types of specific changes:

- hyper-hydration (watery emphysema)

- hyper-aeration

- hyper-hydro-aeration

In hyper-hydration the lungs are imbibed with liquid, heavy, oedematous and so distended that their margins can compress the heart. There are costal marks on the surface, they remain depressed after manual pressure and do not shrink after extraction from the body.

Hyper-aeration is characterised by light, aerated, crepitating, dry lungs, with pneumatic aspect.

Hyper-hydro-aeration is an intermediary phase between the first two, and it is the most frequent.

The lungs of drowning victims are characteristic, but not a positive dg. sign in the absence of other findings. If associated with the mushroom foam and the foamy fluids in the air passages, then the dg. of drowning is almost certain. The microscopic examination of the lungs is inconclusive for the diagnostic of drowning.

e. The cardiac changes **are represented by the presence of approximately 50 ml liquid in the pericardium and small haemorrhagic petechiae on the epicardium.**

f. The stomach **can contain large amounts of water swollen during drowning. In some cases, the water can cross the pylorus and is found in the duodenum.**

B. The putrefied drowned body

The putrefaction of a drowned body onsets slowly, after a long stagnation in water.

There are no significant differences between putrefaction in air and water.

The face is swollen to such an extent that it renders the person unidentifiable.

The posthumous superficial venous net, the epidermal blisters with blurry liquid and the large epidermal detachments are all present.

The two particular aspects in drowning are the swelling and protrusion of the eyes giving a fish-eye aspect, and the nocturnal phosphorescence or some drowned bodies due to the presence of some luminescent bacteria.

The epidermis becomes loosen after 2 weeks and after 3-4 weeks it can be slipped of as a glove (or stocking, of the feet) together with the nails.

This phenomenon is called „the death glove".

Adipocere formation is dependent on the water temperature and oxygenation.

Laboratory tests in drowning

a. Physico-chemical tests

Researchers have tried to demonstrate various characteristics of blood in the right and left heart, secondary to haemodilution or haemo-concentration. Manktelov also examined the pulmonary surfactant. None of the methods led to a reliable drowning test, because results are variable and inconstant.

b. Diatom analyses

Diatoms are algae with a silicon cover, resistant to heat and acid environment. There are approximately 15,000 species; almost half of them live in sweet waters, the rest in stationary or seawaters. The size of the diatoms varies between: 2µm and 1 mm in length (medium length 10-80 µm) and up to 10 µm width.

The diatoms' protective layer is extremely resistant and can be easily detected in organs. The detection of diatoms and plankton might be the only available diagnostic method in cases with post-mortem mutilation and/or advanced putrefaction. If the cause of death is not drowning, the possible sources of diatoms are:

- **contamination from laboratory instruments and recipients;**
- **inhalation;**
- **ingestion.**

So there is no reliable test that can prove or confirm death by drowning. The diatom analysis remains the most trusted determination.

Forensic problems concerning the death by submersion

In cases of drowning, the elements to be established are: the identity of the person, the estimated time of submersion, the cause of death, the circumstances of death etc.

Identification procedures are the same as for any other identification.

The duration of the submersion can be estimated based on the relation between post-mortem changes and the time of death.

a. The cooling of the body depends on the water temperature but there is no established relation concerning this phenomenon. The general effect of rapid cooling is the postponement of most post-mortem changes.

b. Rigor mortis does not provide exact information regarding its onset and evolution, both delayed by the cooling of the body.

c. The lividities appear on the face, the anterior side of the thorax, hands, distal part of the arms and feet. The explanation is that in water, the drowned body usually floats facedown, with the gluteal region upwards and the hands and feet downwards. In turbulent waters, lividities are vague, difficult to notice.

d. The decomposition of the submersed body can be delayed in running or intensely polluted waters.

According to Casper, at constant temperature the putrefaction degree of a body exposed in open air for 1 week (1 month), corresponds with the one of a body that stayed in water for 2 weeks (2 months), or one buried in the ground for 8 weeks (8 months). The most important factor is the water temperature.

The first signs of decomposition appear on the face, neck and superior half of the trunk. With the advancement of decomposition, a large volume of gases accumulates in the tissues; it will render the body capable of floating. The body reappears on the surface after 3-5 days post-mortem, but more probably after 7-14 days.

Obviously, any attempt to estimate the submersion period is relative and the variation interval increases with the prolongation of the submersion period. Some authors state that it is impossible to estimate the period of submersion if it is longer then 1 month.

Here are some approximate calculations used by forensic experts for the estimation of duration of submersion:

- after 1 month: The epidermis of the hands and feet is whitish, wrinkled and detachable (the death glove);

- between 2 and 3 months: The fat tissue transforms in adipocere and after 1 year the adipocere also covered the limbs;

- between 4 and 5 months: The scalp is destroyed and the skull is revealed; due to various mechanical factors the body can be dismembered or partially destroyed.

Medico-legal forms of death by drowning

a. Accidental drowning

Most reported drowning cases are accidents. Except for injuries due to the fall, the immediate danger of the contact with water is the sudden cooling of the body. This determines a reaction, probably triggered by thermal skin receptors, resulting in sometimes-uncontrollable hyperventilation (the initial, surprise phase), high blood pressure, high pulse and ventricular extrasystoles. The result is water inhalation and circulatory collapse.

The survival interval is in direct relation with the water temperature. Alcohol intoxication is undoubtedly a serious risk factor; the vascular dilation induced by alcohol determines an increase of the external temperature that can amplify the heat loss.

b. Suicidal drowning

This form of submersion death is far from unusual. The subject is often more or less dressed, possibly under the influence of alcohol and/or drugs. In some cases, the victim ties a heavy weight to the body.

c. Homicidal drowning

This form or submersion death is extremely rare, probably because it is difficult for an aggressor to drown a healthy, conscious victim. Homicidal drowning requires that the victim is first receives a heavy blow or it is under the influence of various substances – with consequent alteration or loss of consciousness.