SAFETY

simulation for medical practice

SIMULATION APPROACH FOR EDUCATION AND TRAINING IN EMERGENCY

THERMAL EMERGENCIES AND TOXICOLOGICAL EMERGENCIES

University of Foggia- UniFG

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Summary

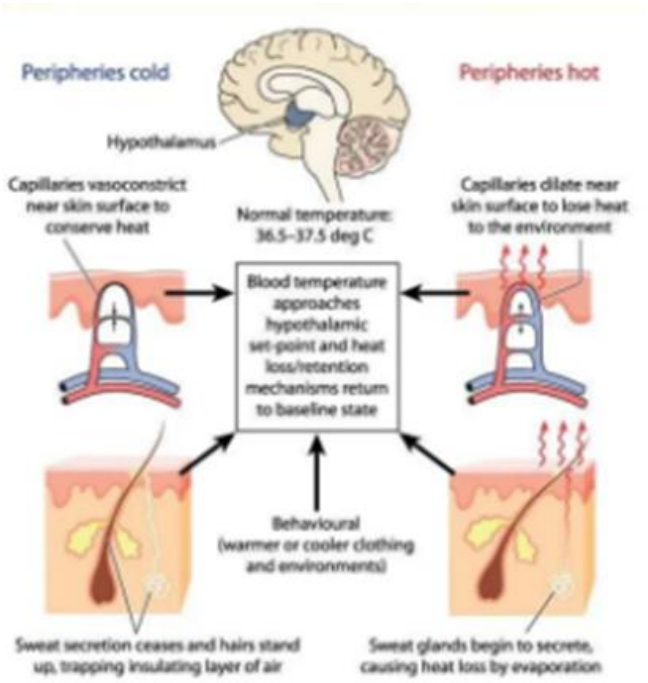
THERMAL EMERGENCIES

ABSTRACT

Thermoregulation is a vital function of the autonomic nervous system in response to cold and heat stress. Thermoregulatory physiology sustains health by keeping body core temperature within a degree or two of 37 °C, which enables normal cellular function. Heat production and dissipation are dependent on a coordinated set of autonomic responses. The clinical detection of thermoregulatory impairment provides important diagnostic and localizing information in the evaluation of disorders that impair thermoregulatory pathways, including autonomic neuropathies and ganglionopathies. Failure of neural thermoregulatory mechanisms or exposure to extreme or sustained temperatures that overwhelm the body's thermoregulatory capacity can also result in potentially life-threatening departures from normothermia.

The autonomic responses to heat stress include cutaneous vasodilation, which liberates heat by radiant and convective heat loss, and sweating, which liberates heat by evaporation.

Each year in the United States, for example, approximately 2000 people die from weather-related causes of death (Berko et al., 2014). The National Center for Health Statistics found that 63% of these were attributed to exposure to excessive or prolonged natural cold, hypothermia, or both; whereas 31% were attributed to exposure to excessive natural heat, heat stroke, or sunstroke.



1. HEAT RELATED DISORDERS

Heat illness is a spectrum of disorders due to environmental exposure to heat. It includes minor conditions such as heat cramps, heat exhaustion, as well as the more severe condition known as heat stroke.

Risk Factors in Heat illness are: obesity, age extremes (young and elderly), poor conditioning and lack of acclimatization,strenuous labor during high temperature, medical condition (cardiovascular diseases), and medications.

1a. HEAT CRAMPS

- ***Introduction***

Heat cramps are represented by painful muscle spasms that occur during or following intense muscle activity carried out in a warm environment . They affect individuals who have difficulty acclimatizing through the normal compensation mechanism.

They predominantly affect young people. They can occur during or after a period of latency of several hours.

During physical activity, the increase in body temperature is balanced through increased sweating, which involves a dispersion of heat. Along with sweating, there is loss of water, sodium, potassium and chlorine. When the concentration of these salts are reduced to the sub-at the critical level, cramps arise.

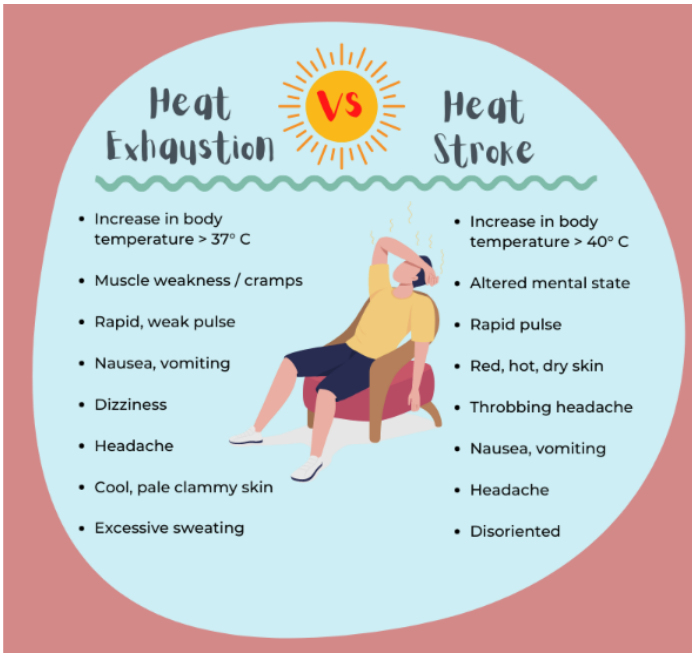
- ***Clinical signs***

- Painful contraction of the skeletal muscles, with sudden onset, with fatigue particularly of muscle mass, especially at the level of the calves, thighs and shoulders.
- Painful spasm can be felt in abdominal muscles.
- Muscle fasciculations may be found.
- The skin may be sweaty or dry, hot or cold.

- ***Treatment***

- On site: give the patient a liter of water in which 5 grams of salt have been dissolved.
- Rehydrating solution: Normosol R flac 1000-1500 ml ev.
- Monitoring of diuresis, cardiovascular parameters and state of consciousness.

- In the emergency room: muscle relaxant therapy with thiocolchicoside 1 fl im every 12 hours.



1b. HEAT EXHAUSTION

- ***Introduction***

Heat exhaustion is a systemic reaction to prolonged exposure to high environmental temperatures (hours or days) and is caused by the depletion of liquids and mineral salts (sodium) which concomitantly excessive vasodilation that drastically lowers blood pressure.

Factors that can favor its onset are in addition to hot and humid weather,physical strength and some drugs such as diuretics, antihistamines, beta-blockers, alcohol, and amphetamines.

The increase in body temperature is usually below 40°C and neurological function can quickly return to normal as soon as the patient is transferred to a cool, ventilated environment.

If this situation is not quickly recognized, it evolves in a short time into heat stroke.

- ***Clinical signs***

- Tiredness
- headache
- hyperthermia (up to 40 °C)
- Dizziness
- hypotension

- Nausea
- loss of consciousness

- ***Diagnosis***

Lab Tasting

- Blood count -> hemoconcentration;
- Electrolytes -> hypernatremia: if the patient has not drunk anything ; if the patients has been drinking water he/she have isotonic dehydration with normal levels of sodium and chlorine.
- Muscle enzymes -> increased creatine phosphokinase (CPK).
- Evaluate renal, hepatic and blood glucose function (differential diagnosis hypoglycemia).

Routine panel

- Electrocardiogram (ECG)
- Arterial Blood Gases (ABG)
- Chest X-Ray

- ***Skills needed for diagnosis***

Basic skills:

Peripheral venous cannulation to secure vascular access and provide blood samples

for lab testing.

Arterial puncture to provide ABG.

ECG lead placement, SpO2 and respiratory rate monitoring, non-invasive BP

measurement and interpretation.

Urinary catheter insertion to monitor urine output.

- ***Therapy***

On site:

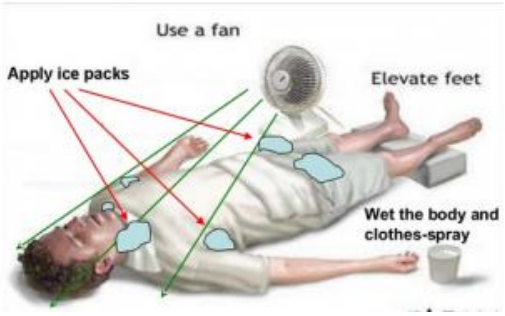
Transfer the patient to a cool, shady environment.

Place him/her in supine decubitus with the lower limbs raised to 35 - 40°.

Replenish liquids -> give the patient a liter of water in which 5 grams of salt have been dissolved;

Administer intravenous fluids and electrolytes if they are too confused to drink and mayvomit -> Rehydrating solution: Normosol R flac 1000-1500 ml ev.

Antipyretic therapy -> acetylsalicylic acid 325 mg 1 cpr every 12 hours.



In the emergency room:

Continue to infuse liquids

Monitoring of diuresis, cardiovascular parameters and state of consciousness

- ECG (place ECG electrodes).
- Respiratory rate.
- Peripheral oxygen saturation (place SpO2 sensor)
- Non-invasive BP (place inflatable cuff).
- Urinary catheter

1c. HEAT STROKE

- ***Introduction***

Heat stroke is a serious syndrome caused by exposure to high environmental temperature and occurs when the body loses the ability to compensate for the increase in body temperature through normal heat dispersion mechanisms. If no quick intervention is taken, the patient could also die.

A body temperature above 41 ° C causes progressive denaturation of protein structures, insufficiency of vital cellular processes that produce energy and loss of the functions of cell membranes. There are several theories proposed to explain how heat can cause all these changes, however, these are alluncertain. They include increased flow of ions, especially sodium

and potassium, Ca+ATPase deficiency, increased glycolysis, sodium and potassium depletion, and impaired neurotransmitter release.

The main mechanism of compensation for the increase in body temperature is sweating. In heat stroke, the threshold of tolerance of organism to high temperatures is exceeded, and sweating stops abruptly. This leads to an alteration of the thermoregulatory mechanisms and a further rise in body temperature, resulting in the formation of cerebral edema. Multi-organ and multisystem dysfunction (MODS) also appear (rhabdomyolysis, acute pulmonary edema, CID, renal failure, liver failure, electrolyte and acid-base balance disorders).

Substances that inhibit cooling and cause dehydration such as alcohol, stimulants, medicaments and age-related physiological changes predispose to the so-called "classic" or non-exertive heat stroke (NEHS), most often in elderly and sick individuals in summer situations with insufficient ventilation.

- ***Clinical Signs***

  It is defined by the TRIAD:

  - hyperpyrexia (internal body temperature > 40.5 °C
  - CNS alterations
  - Anhidrosis

Anhidrosis is frequently found in the elderly patient, while in the young patient, especially if heat stroke occurs during physical activity, it can be absent despite rectal temperatures even > 43 ° C.

In the initial phase the skin is warm and dry, dark red or purplish ("red stage"); then headache, nausea, vomiting, dizziness, psychomotor agitation, delirium, convulsions tend to appear. The rectal temperature becomes very high > 40-42 ° C.

Subsequently, the confusional state appears, followed by a loss of consciousness, which can progress towards irreversible coma ("gray stage"): pale facial skin, cyanotic lips and symptoms of hypovolemic shock appear.

The finding of hematuria, hematemesis, petechiae, bruising, and prolonged bleeding are all clinical signs that allow to diagnose a picture of concomitant CID.

- ***Diagnosis***

Lab Testing

• Blood count -> hemoconcentration;

• Electrolytes -> hypokalaemia, hypophosphatemia, hypocalcaemia

- Muscle enzymes -> increased creatine phosphokinase (CPK)

Routine panel

- Electrocardiogram (ECG)
- Arterial Blood Gases (EAB): metabolic acidosis due to increased production of lactic acid and/or respiratory alkalosis
- Hemocoagulation parameters, renal function, liver function, blood sugar (CID)
- Chest X-Ray

- ***Skills needed for diagnosis***

Basic skills:

Peripheral venous cannulation to secure vascular access and provide blood samples

for laboratory testing.

Arterial puncture to provide ABG.

ECG lead placement, SpO2 and respiratory rate monitoring, non-invasive BP measurement and interpretation.

Urinary catheter insertion to monitor urine output.

- ***Therapy***

On site

The goal is to cool the patient by loweringthe body temperature by 0.1 ° C / min.

Transport the patient to a cool, ventilated place.

If he/she is unconscious, place him/her in a safe lateral position,

Take off the patient's clothes and find out.

Wet the patient with irrigations of fresh water for a few minutes.

Wrap the patient in a wet sheet to induce evaporative cooling.

It may be helpful to place the patient in a bathtub that is full enough to cover the trunk and extremities (by immersion). Be careful that the water is not frozen, as you could have excessive vasoconstriction of the blood vessels of the skin with paradoxical obstacle to the dispersion of internal body heat.

Ice packs on the head, neck, armpits, wrists and groin.

If the patient is conscious and does not vomit, administer hydro-saline rehydrating drinks.

High flow oxygen therapy (10 l/min) using Venturi mask at 24-28%.

Soluzione reidratante-> Normosol R flac: 1000-1500 ml ev a 250 ml/h.

Monitoring of diuresis, cardiovascular parameters and state of consciousness.

- ECG (place ECG electrodes).

- Respiratory rate.

- Peripheral oxygen saturation (place SpO 2 sensor).

- Non-invasive BP (place inflatable cuff).

- Urinary catheter.

- Body temperature,

In the emergency room

Ensure a high flow of oxygen. A fan holder may be required.

Urinary flow equal to the water load introduced should be maintained, if necessary using mannitol or furosemide, to prevent secondary renal failure, especially following rhabdomyolysis.

Intravenous fluids should be used with caution and therefore CVP and PCWP monitoring may be necessary.

In the most severe cases, massive rehydration should be performed: 5 liters in 6-7 hours. It is advisable to alternate the infusion of saline solution with that of glucosata at 5-10%.

Antipyretic-> acetylsalicylic acid 325 mg 1 cpr pgni 12 hours.

If continuous chills -> diazepam 10 mg im.

Cooling maneuvers should be suspended when the body temperature reaches 39 °C.

2. ACCIDENTAL HYPOTHERMIA

Definition

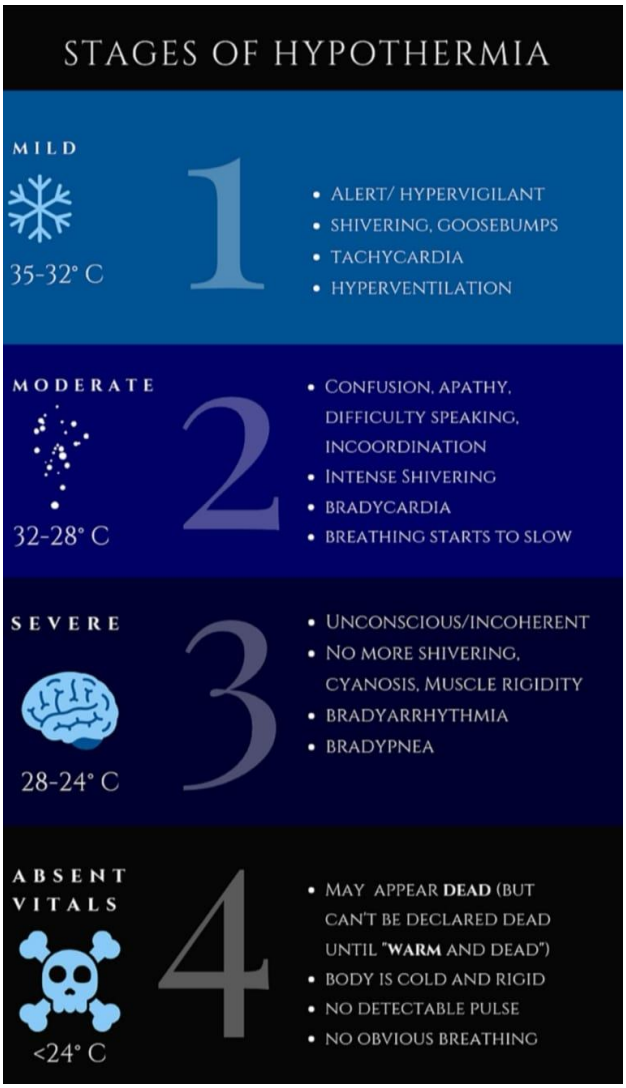
Hypothermia is defined as a reduction in internal body temperature below 35°C. It can be classified as mild (35-32°C), moderate (32-30°C) or severe (below 30°C). Hypothermia is usually observed in cold climates, however it can also develop in temperate regions, on into air-conditioned areas during the summer.

Etiological aspects

Accidental hypothermia can be classified as primary or secondary.

Primary hypothermia occurs when a healthy individual is exposed to temperatures low enough to overwhelm the body's ability to maintain adequate thermoregulation.

Secondary hypothermia occurs in patients in whom the underlying medical pathology disrupts the appropriate mechanisms of thermoregulation. Young and older patients, especially those over the age of 65, and all those who present alterations of the sensory are more exposed. The elderly people may lose sensitivity to cold, while infants easily become hypothermic due to the altered ratio of body mass to volemia.



The main causes of hypothermia are:

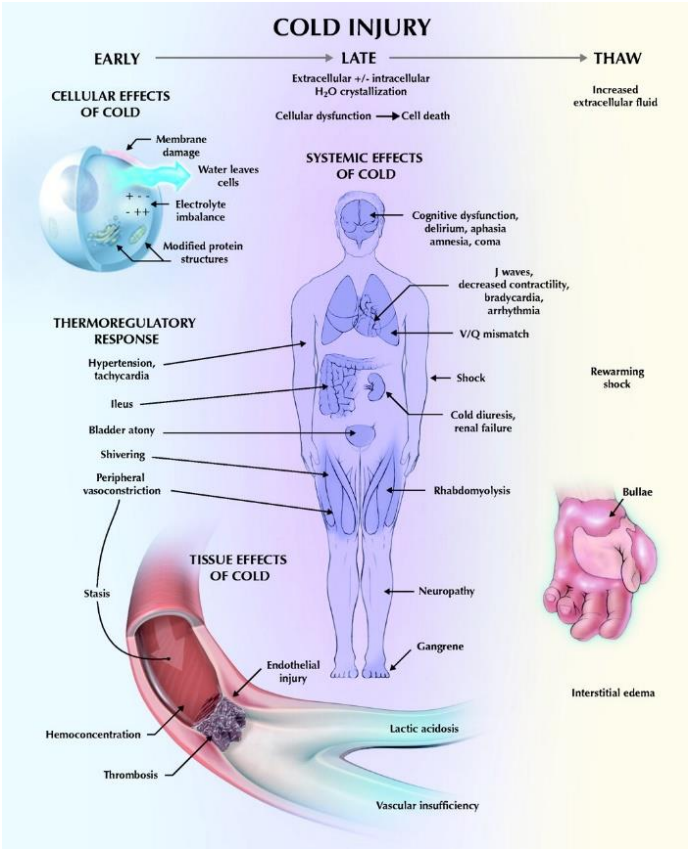
- Accidental: exposure to the cold in immersion or non-immersion conditions.
- From metabolic causes: endocrine pathologies due to hypofunction, such as hypothyroidism, hypocorticoadrenalism, hypopituitarism that lead to a reduction in basal metabolism. Hypoglycemia can lead to hypothermia following secondary hypothalamic dysfunction.
- From hypothalamic and/or SNC dysfunctions: found during head trauma, tumors, cerebrovascular, ischemic or hemorrhagic accidents.
- From alcohol and/or drugs: they are directly responsible, in the USA and Europe, for most cases of hypothermia. Ethanol is a vasodilator and antagonizes compensatory vasoconstriction. As a result of its depressant effects on the SNC, patients remain insensitive to the cold. It also prevents chills, predisposes to hypoglycemia and it has a direct effect on the hypothalamus. The drugs involved in the onset of hypothermia are benzodiazepines, opiates, barbiturates, tricyclic antidepressants, beta blockers and insulin.

- From sepsis: for alterations of the regulation at the hypothalamic level and this clinical finding represents a negative prognostic factor.
- From serious dermatological diseases: it can derive from an alteration of the thermoregulatory function of the skin (significant burns and severe exfoliative dermatitis).

Pathophysiological aspects

A body temperature between 32 and 35°C is mild hypothermia. In this phase the patient is in a condition of excitation (dynamic and responsive state), in which the physiological responses tending to retain and generate heat are produced.

With the further reduction of temperature, the metabolism progressively slows down, leading to a reduction in tissue oxygenation, intracellular oxygen utilization, and carbon dioxide production. When the temperature drops below 32 ° C, the general excitation gives way to a state of slowing down (adynamic and non-responsive state) in which there is a progressive reduction of all bodily functions.



- CARDIOVASCULAR SYSTEM

The initial cardiac response to cold stress is tachycardia. The observation of a tachycardia in the hypothermic patient, however, must push the clinician to look for associated pathologies such as a condition of hypovolemia, hemorrhagic or not, sepsis, ingestion of drugs.

With the persistence of the condition of hypothermia, tachycardia is replaced by bradycardia, a consequence of a spontaneous decrease in depolarization and slowing down of the conduction system, refractory to atropine.

Cardiac output and blood pressure tend to decrease markedly, both due to negative inotropic effects and due to a reduction in blood volume, due to the passage of blood into the extravascular spaces. The most characteristic alterations on the ECG consist of the reversal of the T wave, the prolongation of the PR interval the QRS complex, and the QT interval, together with the appearance of the J wave (characteristic but not pathognomonic of hypothermia, due to an ongoing myocardial lesion). Onset of arrhythmias such as sinus bradycardia, flutter,atrial fibrillation, ventricular fibrillation and asystole.

- RESPIRATORY SYSTEM

Hypothermia initially results in tachypnea followed by a reduction in respiratory rate and current volume. Hypoventilation associated with hypothermia is due to two mechanisms: slowing down metabolism and depression of respiratory activity which can lead to respiratory arrest.

Cold-induced bronchorrhea, as well as thickening of secretions and reduced ciliary activity, make aspiration pneumonia a frequent complication.

Hypothermia also results in a rapid deviation to the left of the oxyhemoglobin dissociation curve, reducing the transfer of oxygen to the tissues.

- NEUROLOGICAL SYSTEM

At the level of the CNS, a progressive depression of the state of consciousness occurs, linearly with the lowering of the temperature. A mild initial state of incoordination is followed by confusion, lethargy and coma. The pupils can be dilated and non-reactive. All these alterations are associated with a widespread reduction in cerebral vascular circulation.

- RENAL SYSTEM

Hypothermia at the renal level causes a centralization of the circulation that hypoperfuses the kidney as well as a direct dysfunction of the renal tubule. This cause the kidney to lose the ability to concentrate urine; a "cold diuresis" is therefore determined, which leads to a significant loss of fluids and a condition of dehydration and hypovolemic shock. The hypothermic patient, when

immobile, may undergo rhabdomyolysis and, in the context of a combined pathogenetic mechanism, which sees myoglobinuria and hypoperfusion interact synergistically, acute tubular necrosis may appear.

Clinical scenarios

Stage I: vasoconstriction, body temperature of 34°C

Intense chills and active movements

Pale, cold and anserine skin

Asthenia

Frequent and rhythmic pulse

Elevated blood pressure

Stage II: depletion of thermogenetic factors and cooling of nerve centers, body temperature ranging between 30 and 25 ° C

Grey, sweaty skin (especially at the palmar and axillary level)

No chills

Marked asthenia

Reduction of the vision and the hearing

Small and rare wrist

Cessation of voluntary movements

Arterial hypotension, oliguria

Appearance of confusion, up to coma and then death from cardiac arrest and paralysis of the breath

Clinical methodology in emergency

LABORATORY DIAGNOSTICS:

- EGA (may not be reliable because most machines that process EGA take 37°C as the reference temperature. PaO2 and PaCO2 values should be reduced by approximately 7.2% and 4.4%, respectively, for each grade <37°C)
- COMPLETE BLOOD COUNT
- GLYCEMIA (tendency to hyperglycemia)
- DOSAGE OF CREATININE, AZOTEMIA, ELECTROLYTES, CPK, AMYLASE (possible pancreatitis secondary to hypothermia)
- EMOCOLTURE
- THYROID HORMONES AND SERUM CORTISOL
- TOXICOLOGICAL EXAMINATION
- URINE TEST

THERAPEUTIC APPROACH IN EMERGENCY:

Patient in cardiorespiratory arrest: support of vital functions with basic methods (BLS)

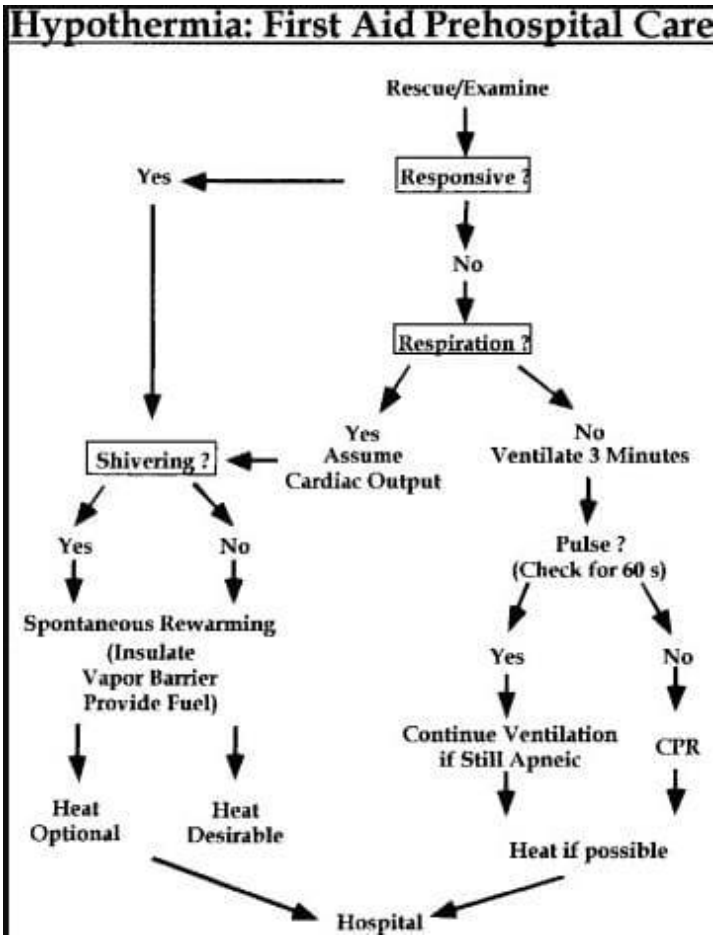
If the patient is in cardio-respiratory arrest: a hypothermic patient in cardiorespiratory arrest should NOT be considered clinically irrecoverable in any case, unless this is completely evident despite the execution of the basic and advanced resuscitation maneuvers to support vital functions. Resuscitation must be prolonged until the patient is warmed up: in case of no answer, the patient can be judge biologically dead. Hypothermia, in fact, protects the brain from lack of oxygen and a person can survive a cardiac arrest for a much longer period than normal.

- First of all, in order to prevent further heat loss in the victim, remove any wet clothing, however, may sure to protect the patient from wind and cold. The patient must be dried and covered with blankets or with a thermal sheet.
- Head, neck and chest should be left uncovered, to allow the execution of resuscitation maneuvers.
- Severe hypothermia and death are sometimes difficult to differentiate, so the American Heart Association's BLS guidelines recommend to evaluate the respiratory activity, the carotid pulse, and the indirect signs of circulation for 30-45 seconds before confirming respiratory and cardiac arrest. Dilated pupils should not be considered a sign of death.

Hypothermia can cause stiffness of the chest wall that make ventilation particularly difficult. The goal is ventilate the patient with a sufficient volume that cause a visible chest expansion.
If the victim shows no signs of circulation, chest compressions should be initiated. The goal is compress the chest, much more rigid than in conditions of normothermy, with a sternal excursion not less than 4-5cm. If the semi-automatic defibrillator (AED) detects a defibrillable rhythm, the discharge must be delivered and CPR must be performed.

- In case of persistence of shockable rhythm, it is necessary to proceed with cardiopulmonary resuscitation (CPR) and, when possible, transport the patient to the most suitable hospital and, among the most suitable, to the nearest site.
- Avoid suspending maneuvers until the victim is heated.
- The decision to discontinue cardiopulmonary resuscitation should be taken in a hospital setting.
- Physical aspects of the hypothermic victim's body, such as apparent rigor mortis, bilateral fixed mydriasis, hypostatic areas are not a criterion for suspending CPR.
- Resuscitation may be suspended on the site if the victim has obvious lethal wounds or if the body is frozen to the point that the chest compressions are impossible and/or when the nose and mouth are completely blocked by ice.
- <u>Support of vital functions with advanced methods (ACLS)</u>
- Oxygen heated to 40-46°C and humidified.
- Orotracheal intubation, when indicated, should not be delayed.
- It is desirable to cannulate a central venous vessel.
- The heart of a hypothermic patient tends to be poorly responsive to the action of drugs, attempts at defibrillation and stimulation with pacemakers.
- The metabolism of drugs is significantly slowed down and this exposes the patient, if returned in normothermic conditions, to dangerous phenomena of accumulation toxicity. This is the rationale for avoiding the administration of adrenaline and any other medication until the internal temperature has reached at least 30 ° C. Once this temperature is reached, it is advisable to double the time intervals between the administration of the drugs, the dosages of which must be the lowest indicated in the protocols.
- Ventricular fibrillation may not be responsive to defibrillation if the internal temperature is below 30°C.

- If the patient does not respond to the first attempt at defibrillation, followed by immediate resumption of CPR, it is advisable to postpone the other electric shocks when the internal temperature exceeds 30 ° C.
- All liquids to be infused intravenously should be heated before administration (37-41°C)
- If hypotension refractory to ev administration of fluids and heating procedures, dopamine (1-5 mg/kg/min) may be administered.

1. 

2. ***<u>Patient NOT in cardiorespiratory arrest: First aid management</u>***

- Remove wet clothing and protect the patient with blankets.
- Transport the injured person to a closed place or otherwise protected from environmental factors by keeping the head on a lower plane than the feet.
- Carry out external active heating.
- If the patient is alert, hot and sugary drinks should be administered.
- Mentally engage the injured person, trying not to make him fall asleep.

- Apply a sterile bandage on visible lesions.

Ensure central active heating which can be obtained by:

- Inhalation of humidified and heated oxygen, through facial mask.
- Apply peripheral venous access.
  Ev infusion of heated liquids (37-38 ° C): crystalloids ("mixed" solution, physiological and 5% glucose) + colloids (or concentrated blood cells).
- Peritoneal dialysis: introducing in a few minutes, 2 or 3 liters of physiological solution at 37-38 ° C that will be removed immediately after. The maneuver will be repeated several times in order to increase body temperature.
- Heated hemodialysis in the most severe cases.
- Antibiotic therapy: ceftriaxone 2gr ev x 2/die.
- Bladder catheter for diuresis monitoring.

3.Bibliography

1. Lugo-Amador, Nannette M; Rothenhaus, Todd; Moyer, Peter (2004). "Heat-related illness". Emergency Medicine Clinics of North America. 22 (2): 315–27, viii. doi:10.1016/j.emc.2004.01.004. PMID 15163570.

2. Lipman, GS; Eifling, KP; Ellis, MA; Gaudio, FG; Otten, EM; Grissom, CK; Wilderness Medical Society (December 2013). "Wilderness Medical Society practice guidelines for the prevention and treatment of heat-related illness". Wilderness & Environmental Medicine. 24 (4): 351–61. doi:10.1016/j.wem.2013.07.004. PMID 24140191.

3. Leon LR, Bouchama A (April 2015). "Heat stroke". Comprehensive Physiology. 5 (2): 611–647. doi:10.1002/cphy.c140017. ISBN 9780470650714. PMID 25880507.

4. Gaudio FG, Grissom CK (April 2016). "Cooling Methods in Heat Stroke". The Journal of Emergency Medicine. 50 (4): 607–616. doi:10.1016/j.jemermed.2015.09.014. PMID 26525947.

5. Epstein Y, Yanovich R (June 2019). "Heatstroke". The New England Journal of Medicine. 380 (25): 2449–2459. doi:10.1056/NEJMra1810762. PMID 31216400.

6. Bouchama A, Knochel JP (June 2002). "Heat stroke". The New England Journal of Medicine. 346 (25): 1978–1988. doi:10.1056/nejmra011089. PMID 12075060.

7. Hifumi T, Kondo Y, Shimizu K, Miyake Y (2018). "Heat stroke". Journal of Intensive Care. 6: 30. doi:10.1186/s40560-018-0298-4. PMC 5964884. PMID 29850022.

8. Morris, Andrew; Patel, Gaurav (2022). "Heat Stroke". StatPearls. StatPearls Publishing. Retrieved May 12, 2022

9. Brown DJ, Brugger H, Boyd J, Paal P (November 2012). "Accidental hypothermia". The New England Journal of Medicine. 367 (20): 1930–8. doi:10.1056/NEJMra1114208. PMID 23150960. S2CID 205116341.

10. Fears, J. Wayne (February 14, 2011). The Pocket Outdoor Survival Guide: The Ultimate Guide for Short-Term Survival. Simon and Schuster. ISBN 978-1-62636-680-0.

11. Robertson, David (2012). Primer on the autonomic nervous system (3rd ed.). Amsterdam: Elsevier/AP. p. 288. ISBN 9780123865250. Archived from the original on September 8, 2017.

12. McCullough L, Arora S (December 2004). "Diagnosis and treatment of hypothermia". American Family Physician. 70 (12): 2325–32. PMID 15617296.

13. Petrone P, Asensio JA, Marini CP (October 2014). "Management of accidental hypothermia and cold injury". Current Problems in Surgery. 51 (10): 417–31. doi:10.1067/j.cpsurg.2014.07.004. PMID 25242454.

14. Turk, EE (June 2010). "Hypothermia". Forensic Science, Medicine, and Pathology. 6 (2): 106–15. doi:10.1007/s12024-010-9142-4. PMID 20151230.

TOXICOLOGICAL EMERGENCIES

ABSTRACT

Acute poisoning is a medical emergency that represents up to 10% of the workload of the emergency room. Toxic substances, after introduced into the organism, are absorbed and can cause various damage, up to becoming lethal.
Acute drug poisoning is more frequent in children and adults with the purpose of committing suicide.
The presumed diagnosis is made through history taking and the confirmation at home of suspect material for improper / excessive intake of substances. In the case of an unconscious patient, signs and symptoms attributable to the different forms of intoxication will be sought and validated in some cases by specific laboratory..
It is therefore important to try to understand which substances have been taken, when (the time factor is essential, since symptoms can appear 6-24 hours after intake, for paracetamol up to 48 hours post-intake), in which quantity (in the finding of more empty blisters the higher dose is considered) and the chronicity of intake.

4.GENERAL CONSIDERATIONS

Acute intoxication is defined as the set of secondary ailments and injuries in voluntary or accidental contact, of short or long duration, with exogenous substances defined as toxic.

Acute intoxication can occur from

- Ingestion
- Contact
- Inhalation
- Inoculation

The different routes of exposure have different risks of complications related to the time of exposure, certainly ingestion is nowadays the most frequent route. The distribution in the population shows two age peaks between 10 months and 5 years in mostly accidental children and from 18 to 45 years voluntary for suicidal and demonstrative purposes. Cases of intoxication in old age are related to iatrogenic substances taken by mistake.

Initial Evaluation

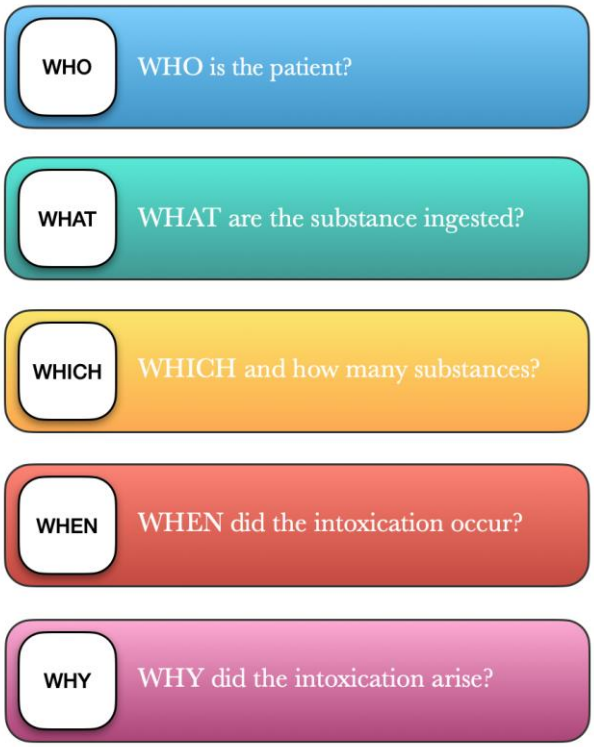
After a quick assessment of the safety of the scene - remember: for some exposure to toxic inhalants it may be necessary to use particular PPE or special specialist units for decontamination - it is advisable to perform a quick clinical look with an ABCDE approach and interventions appropriate

relating to the problems identified in the various stages of evaluation. Especially important in the initial approach with an unconscious patient and an unknown toxic agent is the administration of the **coma cocktail:**

- **thiamine:** 100 mg iv or im

- **33% glucose:** 100 ml iv. If necessary, repeat the dose or start an infusion of 10% glucose, provided that the capillary glycaemia is less than 80 mg/dl, otherwise the parenteral route is not indicated.
- **Naloxone:** 0.4-3 mg iv + 0.4 mg im. Administration can go up to 10mg in codeine poisoning and in cases where airway management is required. Administration may go up to 10 mg in codeine poisoning and in cases where airway management is required. Its administration can lead to awakenings with agitation; therefore, it may be useful to resort to means of physical containment.
- **Flumazenil:** 0.2 mg iv in 30 sec. repeatable after 30 sec. Its use is contraindicated in patients with concomitant intoxication by tricyclic antidepressants, patients dependent on benzodiazepines, patients in therapy with benzodiazepines for control of convulsive states.

Through a careful analysis of the situation, the rescuer could better reconstruct what happened and get hold of knowledge of rescue. An excellent reconstruction of the scenario is the application of



the ***"5W"* rule** (who, which, what, when, why).

Everything should be carefully marked both in the pre- and in-hospital phase, since the substances belonging to a particular pharmacological or chemical class often produce the same signs and symptoms identifiable in particular "toxicological syndromes": through specific laboratory and

instrumental tests it is possible to confirm the presence of the toxic substance and the suspected syndrome and finally apply the most appropriate therapeutic protocol, perhaps with the concomitant administration of specific antidotes where possible.

Secondary Evaluation

Regarding, the secondary phase in the treatment of the intoxicated patient, the evaluations of phases D and E will be investigated to evaluate the possible differential diagnoses.

1. NEUROLOGICAL EXAMINATION: evaluate if the patient is alert or in a coma, by examining the pupils, also paying attention to abnormal eye movements, evaluate muscle tone, the presence of tremor, myoclonus, chills.

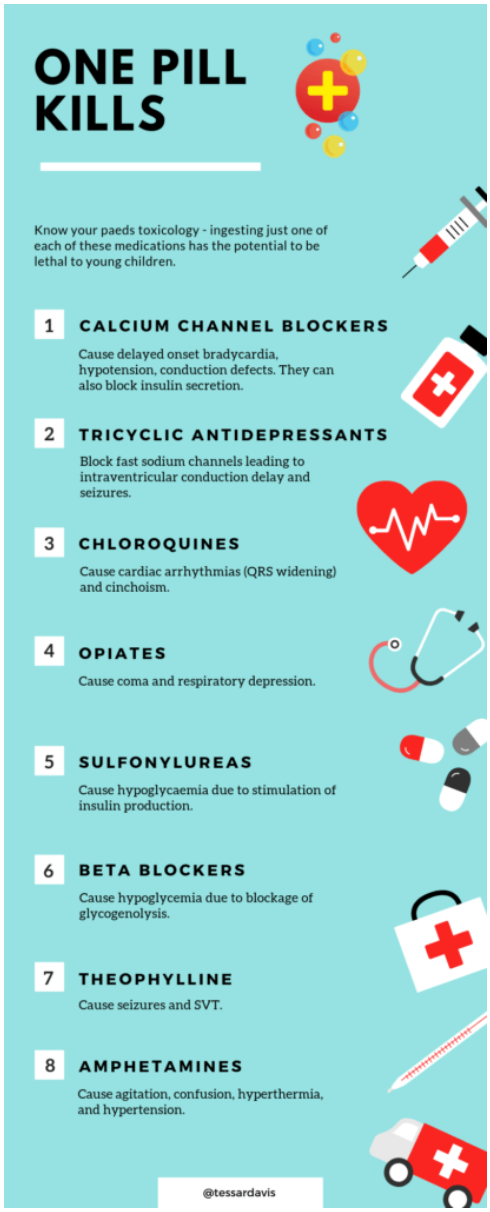
2. SKIN EXAMINATION: to evaluate for dry skin, rash, presence of patches or other transdermal devices, skin color (cyanosis, paleness, redness) and skin lesions

3. OLFACTORY TEST: evaluate the smell of breath, the smell of blood, urine and serum. The smell of the skin (bitter almonds: cyanide; fruity: ketosis; garlic: arsenic, dmso, selenium; carrots: cicutoxin; pears:tear gas)

Clinical Notes

- Clinical manifestations affecting the respiratory system:
    - *Ventilatory failure for central depression*: Tricyclic antidepressants, alcohol, hypnotics, sedatives, clonidine, opiates
    - *Paralysis of the respiratory muscles*: organophosphorus, carbamates, strychnine, botulinum.
    - *Cellular hypoxia*: Cyanide, CO, hydrogen sulfide.
    - *Non-cardiogenic EPA*: cocaine, paraquat, ethylene glycol.
    - *EPA cardiogenic*: beta-blockers, quinidine, verapamil
    - *Bronchospasm*: hydrocarbons, organophosphorus, metal fumes
- Manifestations affecting the cardio-circulatory system:
    - *Bradycardia / BAV:* Tricyclic antidepressants, beta-blockers, procainamide, opiates, clonidine, digitalis, organophosphorus, calcium channel blockers, lithium.

- *Tachyarrhythmias:* amphetamines, theophylline, tricyclic antidepressants, antihistamines, atropine, thyroid hormones
- *Hypotension:* beta-blockers, calcium channel blockers.
- *Hypertension*: amphetamines, MAOIs, tricyclic antidepressants.



- <u>The manifestations of the Central Nervous System:</u>
  - *Coma:* anticholinergics, tricyclic antidepressants, ethanol, clonidine, opiates, CO, cyanide, hypoglycemic agents, salicylates, lithium.
  - *Convulsions*: amphetamines, insecticides, haloperidol, methanol, CO.
  - *Hypothermia*: ethanol, opiates, hypoglycemic agents, barbiturates.
  - *Hyperthermia:* anticholinergics, antipsychotics, LSD, cocaine, O. thyroid

- <u>Hepatic manifestations:</u>
  - *Hepatic insufficiency:* paracetamol, amanita phalloides, ethanol, hydrocarbons, insecticides.

- <u>Renal manifestations</u>:
  - *Renal insufficiency*: paracetamol, naphthalene haemolysis, heavy metals, cocaine, CO, strychnine.

- <u>Disorders of metabolism:</u>
  - *Hypoglycemia:* hypoglycemic agents, ethanol, salicylates.
  - *Hyperglycemia*: corticosteroids, glucagon.

- <u>Disorders of haemostasis</u>:
  - *bleeding*: rodenticides, mothballs

TOXICAL SYNDROMES		
Syndrome	**Signs and symptoms**	**Toxic**
Anticholinergic syndrome	delirium, tachycardia, dry skin, dilated pupils, myocolines, hyperpyrexia, urinary retention and constipation	antihistamines, scopolamine, antiparkinsonian drugs, antipsychotics, antidepressants, muscle relaxants and some plants
Sympathithomimetic syndrome, hypertachia	paranoia , sweating, mydriasis and hyper-reflexia. In severe cases there is seizures, hypotension and arrhythmia.	cocaine, amphetamines, methamphetamines, decongestants, caffeine and theophylline
Syndrome	coma, respiratory depression, myosis, hypotension, bradycardia, hypothermia, reduction of peristalsis and hyporeflexia	narcotics of all types, especially opiates, barbiturates, benzodiazepines and ethyl alcohol
Opiate	confusion , agitation, tremor, muscle fasciculations, extrapyramidal syndrome, hyperreflexia, hyperpyrexia, diaphoresis and tachycardia	SSRI intoxication
Cholinergic syndrome	confusion, weakness, salivation, lacrimation, urinary and fecal incontinence, abdominal cramps, emitted, myosis, brads / tachycardia and	intoxication from organophosphates and insecticides, nerve gases, physostigmine, and some fungi.

5.PRINCIPLES OF THERAPY

In the hospital setting it is strongly recommended to start a specific laboratory diagnostic protocol with evaluation of glycemia, electrolytes, renal and hepatic function, blood count with formula, plasma osmolarity and anion gap, toxicological screening and ethanolemia, urinalysis and search for active metabolites. Perform EGA, ECG, perform pregnancy tests in women, of childbearing age, continuous monitoring of vital signs and place bladder catheter.
In addition, stabilize the haemodynamics: in case of hypotension with crystalloid infusion even up to 5l / day and in case of lack of haemodynamic response, it will be necessary to proceed with the administration of positive inotropes in order to guarantee a PAS> 90 - 100mmHG and a diuresis > 0.5 m / kg / h.
The temperature control in *hypothermia* will be guaranteed through active and passive heating means; however, in *hyperthermia* we will proceed with lukewarm sponging. The control of any *convulsions* can be carried out with diazepam 0.1 / 0.2 mg / kg iv, while *agitation, delirium and psychosis* alloperidol 0.1-0.2 mg / kg iv or im in 1 minute can be administered.
After the initial phase of stabilization of the vital parameters, identification of the toxic agent and the ascribable syndrome, a poison control center must always be involved, which will take care of setting the specific therapy for the acute phase of the patient, and advises if monitoring in the ICU is necessary. Evaluate the clinical course and the appearance of complications (generally 24-48 hours); keep the patient under surveillance, especially in cases of voluntary intoxication, with risk of suicide, request any psychiatric consultations to assess the need for hospitalization in dedicated facilities.

DECONTAMINATION will be necessary when exposure to the toxic substance concerns a large body surface, beyond whether it occurs by contact or ingestion in which the possible damaging mechanism and the absorption phase change instead.

Ocular decontamination must be carried out with the use of ocular retractors and irrigation of abundant aqueous solution in order to establish a pH of 7.4 of the lower conjunctival sac, the local instillation of ophthalmic anesthetics associated with antibiotics such as tetracyclines is also useful.

Skin decontamination involves the removal of contaminated clothes and abundant washing with aqueous solution in the contact areas, avoiding the flow of liquids if the toxic substance is a caustic substance to avoid further contact of healthy areas.

Respiratory decontamination secondary to exposure to toxic gases or vapors involves the administration of O2 at high flows, mechanical ventilation and in some cases iperbaric therapy.

Since most serious poisoning occurs by ingestion of the toxic substance, gastrointestinal decontamination is carried out with the aim of:

1. recover the toxic substance if indicated (gastric lavage),
2. prevent the absorption of the ingested substance,
3. favor the elimination of the absorbed toxic

1 - 2) PREVENT ABSORPTION

The toxical substance can be absorbed by different organs: in case of ingestion, especially within 2 hours of contact with noxa, it is necessary to evaluate different therapeutic options.

- Gastric emptying:

Current evidence shows that neither gastric lavage nor emesis are able to change the course in mild / moderate poisoning, since very few substances are actually recovered. To date, gastric lavage is indicated in ingestion within 1 hour of an extremely toxic agent or in very high quantities (in such cases it can also be evaluated for ingestions beyond an hour). Administration of Ipecac is currently not recommended due to the risk of ab ingestis. EGDS is contraindicated in ingestion of caustics, substances with petroleum, gastrointestinal haemorrhage, haemodynamic instability. The material is prepared for oro-tracheal intubation with the risk of difficult intubation expected due to compromise of the upper airways; the suction system and the EGDS material are prepared. For washing, 0.9% NaCl at 38 ° C is used (the patient is at risk of hypothermia!). Wash and aspirate until clear liquid similar to the one being inserted is aspirated. If the administration of activated charcoal is indicated, it is inserted at the last through EGDS.

- *Activated carbon*:

Activated carbon represents the method of choice for most cases, where doses of 1 g / kg via SNG are administered, with the aim of having a carbon: substance ratio of 10: 1; ideally it should be administered within 1 hour of ingesting the poison, but even up to 6 hours there were positive effects. Activated carbon is not effective in blocking the intake of lithium, ethyl alcohol, hydrocarbons, cyanides and strong acids / bases. For substances with important entero-hepatic recirculation (carbamazepine, theophylline, quinine, dapsone), charcoal can be administered again at 2-4 hours.

- Intestinal irrigation:

It is based on the administration of polyethylene glycol (Isocolan 1-2 L / h in adults, 200-500 ml / h in children) administered orally or via SNG until the contents of the intestinal lumen appear clear. It is indicated in the case of substances that cannot be absorbed by activated carbon (such as lithium and iron); it cannot be administered in cases of ileus or hemodynamic instability. For those with bolas, Isocolan should be administered by performing an adequate intestinal irrigation. Surgery

plays a key role in the case of rupture of the bolas with acute cocaine poisoning or in the case of intestinal obstruction.

3) ELIMINATION OF THE SUBSTANCE:

As regards the facilitation of the elimination of the substance from the body, the two methods of choice are the administration of sodium bicarbonate or hemodiafiltration.

• $NaHCO_3$:

Sodium bicarbonate allows ingested acids to be found in the urine in ionized form, so as to accumulate more faintly- Adiuretic is usually not combined to force diuresis. It is indicated in case of severe intoxication by salicylates, Phenobarbital and / or severe rhabdomyolysis (CK over 16,000 U / l). It also allows cardiotoxic manifestations such as QRS enlargement, elongated QT and BAV to be treated more effectively. It is contraindicated in case of hypervolemia with pulmonary edema. NaBic 8.4% 100 ml in glucose 5% 1,000 ml is administered at the rate of 250 ml / h IV, adjusting the rate in such a way as to have blood pH <7.55 and urinary pH between 7.5-8.0; the risk of hypokalaemia is important and potassium is associated with IV in 1-2 hours (never as a bolus).

• Hemodyalisis:

It has a role in eliminating the small hydrophilic molecules that can easily overcome the semi-permeable membrane (lithium, salicylates, methanol, theophylline, potassium, some antibiotics) are indicated in particularly serious conditions and with worsening of the clinical picture despite the maximal supportive therapy. The techniques used are:

- Hemodialysis
- Hemoperfusion
- Peritoneal dialysis

Antidote	Adult Dosage	Indication
Dextrose (glucose)	1 g / kg IV	Insulin hypoglycemia
Fab antidigoxin Acute toxicity	5 -10 vials	Digoxin intoxication and other steroids cardioattive
Flumazenil	0,2mg EV	Benzodiazepines
Glucagon	3 - 10mg iv	Calcium-blockers
Hydroxocobalamin	70 mg IV (maximum *5* g). It can be repeated up to 3 times. Administer with sodium thiosulfate.	Cyanide Nitroprusside
Lipid emulsion 20% iv	Bolus of 100 ml IV over 1 min followed by 400 ml IV in pills of 20 min	Toxicity from local anesthetics Rescue therapy for lipophilic cardiotoxins
Methylene blue	1-2 mg / kg IV	Oxidizing toxins (e.g.,nitrites, benzocaine,sulfonamide)
Naloxone	As much as needed Start with:0.1 - 0.4 mg IV	Clonidine
Thiamine	100 mg IV	Wemicke Beriberi syndrome

N-acetylcysteine	140mg / kg IV	Paracetamol
Physostigmine	0,25 - 2 mg iv	Atropine
Atropine Parylidoxine	2 - 5 mg iv 1 - 2 g iv	Organophosphorus
5% ethanol in 5% glucose	Load 15ml/kg iv Maintenance infusion 2 - 4ml / kg / h	Methyl alcohol, ethylene glycol
O2	100%	Carbon monoxide

6.Bibliography

1. Tintinnali JE, et al. Tintinnali's emergency medicine urgency. Vol. II SECTION 15 PAGE 1269-1420

2. Balzanelli MG et al Manual of Emergency Medicine and First Aid. Cap 30 pages 745-759

3. Andreotti l et al Territorial medical emergencies, diagnosis and therapy. Chapter 23 Page 473-479

4. Balzanelli MG, Agro 'FE Et al. Territorial emergency medicine therapeutic guidelines 118. Cap46 Pag 136-149

5. Center for Behavioral Health Statistics and Quality. National survey on drug use and health. Rockville, MD: Substance Abuse & Mental Health Services Administration, 2013.

6. Who. Guidlines for estabilishig a poison center. Jenuary 2021