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## DEFINITIONS OF SYNDROMES AND CLINICAL SIGNS

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### The Curling Ulcer

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Curling's ulcer is an acute gastric erosion resulting as a complication from severe burns when reduced plasma volume leads to ischemia and cell necrosis (sloughing) of the gastric mucosa. The condition was first described in 1823 by Thomas Blizard Curling, who observed a series of 10 patients.

The Curling's ulcer is the prototype of the so-called "stress ulcers" - these are stress-induced gastritis or gastropathy where the gastric and sometimes esophageal or duodenal mucosal barrier is disrupted secondary to a severe acute illness. It may present in the form of erosive gastritis ranging from asymptomatic superficial lesions, and occult gastrointestinal (GI) bleed to overt clinically significant GI bleeding. The stress ulcers secondary to systemic burns in contrast with the Curling ulcer, stress ulcers in patients with acute traumatic brain injury are known as Cushing ulcer. The gastric body and fundus are common locations for stress ulcerations but can also be seen in antrum and duodenum.

The major risk factors for the development of stress ulcerations include: - Mechanical ventilation for more than 48 hours / Abnormal coagulation profile such as platelet count less than 50,000, INR greater than 1.5 and PTT greater than 2 times the control value / Sepsis or septic shock / Use of vasopressors / Use of high dose systemic corticosteroids (more than 250 mg or the equivalent of hydrocortisone per day) / Hepatic failure / Renal failure / Multiorgan failure / Burns of more than 30% of body surface area / Head trauma / Lack of sanitation during intensive care unit (ICU) stay / History of GI bleeds within a year.

### The Cushing Ulcer

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Cushing ulcer are a type of gastrointestinal ulcer associated with stressful medical conditions of the brain, in particular, trauma (accidents), surgical interventions, strokes or tumors. These types of ulcers usually are single, deep ulcers that are prone to perforation.

They are associated with high gastric acid output and are located in the duodenum or stomach, although they occur more commonly in the stomach. Amongst other complications we can encounter bleeding. The cause of stress ulcers such as Cushing's ulcers is not known, but increased secretion of acid in the stomach is important in their development, and prevention and treatment generally is directed at neutralizing or preventing the secretion of acid.

### Biermer's Anemia

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Vitamin B12 deficiency anemia, of which pernicious anemia is a type is a disease in which not enough red blood cells are produced due to a deficiency of vitamin B12. The most common initial symptom is feeling tired. Other symptoms may include shortness of breath, pale skin, chest pain, numbness in the hands and feet, poor balance, a smooth red tongue, poor reflexes, depression and confusion. Without treatment some of these problems may become permanent.

## **Menetrier disease**

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Menetrier disease is a rare disorder characterized by massive overgrowth of mucous cells (foveola) in the mucous membrane lining the stomach, resulting in large gastric folds. The most common symptom associated with Menetrier disease is pain in the upper middle region of the stomach (epigastric pain).

## **Zollinger-Ellison Syndrome**

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Zollinger–Ellison syndrome is a disease in which tumors cause the stomach to produce too much acid, resulting in peptic ulcers. Symptoms include abdominal pain and diarrhea. The syndrome is caused by a gastrinoma, a neuroendocrine tumor that secretes a hormone called gastrin.

## **Virchow-Troisier Sign**

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Troisier sign is the clinical finding of a hard and enlarged left supraclavicular node (Virchow node) and is considered a sign of metastatic abdominal malignancy.

## **The Trousseau Syndrome**

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The Trousseau sign of malignancy or Trousseau's syndrome is a medical sign involving episodes of vessel inflammation due to blood clot (thrombophlebitis) which are recurrent or appearing in different locations over time (*thrombophlebitis migrans* or migratory thrombophlebitis).

## **Krukenberg tumors**

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Krukenberg tumor is a rare metastatic signet ring cell tumor of the ovary, accounting for 1–2% of all ovarian tumors. The stomach is the primary site in the majority of Krukenberg tumor cases, followed by carcinomas of the colon, appendix and breast, particularly invasive lobular carcinoma, according to Al-Agha OM et al, 2006. The eponym was attributed to this tumor following the description of 5 cases by Friedrich Krukenberg (1871–1946) in 1896, who described it as being common among young women, presenting with ascites, an uneven knobby ovarian surface and lymphatic involvement, according to Young RH in 2006. These tumors are characterised by uncertain pathogenesis, challenging etiological diagnosis and poorer prognosis compared with their primaries. Previously, any metastatic ovarian cancer was referred to as Krukenberg tumor; however, Novak and Gray, in 1938, created new diagnostic criteria to eliminate any confusion. Accordingly, a mucin-secreting signet ring cell carcinoma in the dense fibroblastic stroma of the ovary is referred to as Krukenberg tumor. The diagnosis of Krukenberg tumor is currently based on the diagnostic criteria of the World Health Organization based on the pathological description by Serov and Scully, since 1973. The presence of the following characteristics is required for diagnosis: Stromal involvement, mucin-producing neoplastic signet ring cells and ovarian stromal sarcomatoid proliferation.

## **Budd-Chiari Syndrome**

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Budd-Chiari syndrome is an uncommon condition induced by thrombotic or nonthrombotic complete or partial obstruction of the hepatic venous outflow and is characterized by hepatomegaly, ascites, and abdominal pain.

The prognosis is poor in patients with Budd-Chiari syndrome who remain untreated, with death resulting from progressive liver failure in 3 months to 3 years from the time of the diagnosis, according to a 2005 study by Khuroo MS et al from J. Gastroenterol Hepatol. Following portosystemic shunting, however, the 5-year survival rate for patients with the syndrome is 38-87%. The current 5-year survival rate following liver transplantation is around 70%, according to studies of Montano-Loza et al, Valla et al., Segev et al published in 2015.

## **Gilbert Syndrome**

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(also known as:

Constitutional Liver Dysfunction, Familial non-Hemolytic Jaundice, Gilbert-Lereboullet Syndrome, Hyperbilirubinemia I, Meulengracht's Disease, Unconjugated Benign Bilirubinemia).

Gilbert syndrome is a mild genetic liver disorder in which the body cannot properly process bilirubin. Individuals with Gilbert syndrome have elevated levels of bilirubin (hyperbilirubinemia), because they have a reduced level of a specific liver enzyme required for elimination of bilirubin. Most affected individuals have no symptoms (asymptomatic) or may only exhibit mild yellowing of the skin, mucous membranes, and whites of the eyes (jaundice). Jaundice may not be apparent until adolescence. Bilirubin levels may increase following stress, exertion, dehydration alcohol consumption, fasting, and/or infection. In some individuals, jaundice may only be apparent when triggered by one of these conditions. Gilbert syndrome is inherited as an autosomal recessive trait.

## **Crigler-Najjar Syndrome**

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(also known as: Familial non-hemolytic unconjugated hyperbilirubinemia, Hereditary unconjugated hyperbilirubinemia)

Crigler-Najjar syndrome is a rare genetic disorder characterized by an inability to properly convert and clear bilirubin from the body.

It was first recognized in six infants of three couples who were blood relatives (consanguineous). These cases were reported in the medical literature in 1952 by Drs. Crigler and Najjar. In 1962, Dr. Arias reported a milder version of this disorder, which is now termed Crigler-Najjar syndrome type II.

Affected individuals cannot convert unconjugated bilirubin to the conjugated form because they lack a specific liver enzyme required to break down (metabolize) bilirubin. Since they cannot convert bilirubin, they develop abnormally high levels of unconjugated bilirubin in the blood (hyperbilirubinemia).

The hallmark finding of Crigler-Najjar syndrome is a persistent yellowing of the skin, mucous membranes and whites of the eyes (jaundice). There are two forms of this disorder: Crigler-

Najjar syndrome type I, characterized by a nearly complete lack of enzyme activity and severe, even life-threatening symptoms; and Crigler-Najjar syndrome type II, characterized by partial enzyme activity and milder symptoms. Both forms are inherited as autosomal recessive traits and are caused by errors or disruptions (mutations) of the UGT1A1 gene.

### **Lucey-Driscoll Syndrome**

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Lucey-Driscoll syndrome, a form of transient familial hyperbilirubinemia, and represents a rare metabolic disorder that leads to very high levels of bilirubin in a newborn's blood. Babies with this disorder may be born with severe jaundice and lethargy. It occurs when the body does not properly metabolize the fetal bilirubin. If untreated, this condition can cause seizures, neurologic problems (kernicterus) and even death. Treatment for Lucey-Driscoll syndrome includes phototherapy with blue light (to treat the high level of bilirubin in the blood) and an exchange transfusion is sometimes necessary. Different inheritance patterns have been reported and, in some cases, it occurs in individuals with no family history of the condition.

### **Banti Syndrome**

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(also known as Idiopathic congestive splenomegaly, Idiopathic portal hypertension).

This syndrome is characterized by abnormal enlargement of the spleen (splenomegaly) due to obstruction of blood flow in some veins and abnormally increased blood pressure (hypertension) within the veins of the liver (e.g., hepatic or portal veins), or the spleen (splenic veins). The disorder may be due to any number of different factors causing obstruction of portal, hepatic, or splenic veins including abnormalities present at birth (congenital) of such veins, blood clots, or various underlying disorders causing inflammation and obstruction of veins (vascular obstruction) of the liver.

### **The Aorto-Mesenteric Clamp Syndrome**

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(also known as Wilkie Syndrome)

Is a rare acquired vascular compression disorder in which acute angulation of the superior mesenteric artery (SMA) results in compression of the third part of the duodenum, leading to obstruction.

It should not be confused with nutcracker syndrome (which can be an association), also a superior mesenteric artery compression disorder, where the SMA compresses the left renal vein, although some authors use the terms interchangeably.

The compression of the third portion of the duodenum by the superior mesenteric artery (aorto-mesenteric clamp) is therefore a rare cause of abdominal pain. Its clinical appearance may range between an asymptomatic and accidental radiological finding and an acute duodenal ileus (superior mesenteric artery syndrome), which requires urgent surgical intervention. This is a rare surgical condition, for example in study with 1280 patients, Rosa-Jimenez et al, in 2003, found just 10 patients with this syndrome. So far only 400 cases around the world have been diagnosed properly (with paraclinical imaging).

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## CHAPTER 1. CLINICAL CASE PRESENTATION PLAN

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### 1.1 Introduction

The presentation of clinical cases is a special technique that requires years and years of preparation, starting from the student stage until full professional maturity.

A typical clinical case examination should take no longer than 20 minutes, divided into 3 stages, represented by:

- The physical examination of the patient and obtaining the data from the observation sheet and from the commission;
- Elaboration of the presentation and systematization of the obtained data;
- Presentation of the case to the board.

### 1.2. Presenting a clinical case

Along the following lines we will outline the presentation plan of the cases in front of the clinical board of specialists, detailing the objective exam as well as the major objectives of the systematization of the obtained data and the elaboration of clinical presentation.

There are 28 key points that should be covered in any surgical clinical case presentation:

#### 1. Identification data of the patient:

At this stage we can state:

*"[...] I had to examine the patient \_\_\_\_\_, M/F, profession \_\_\_\_\_, living in \_\_\_\_\_, actually originating in \_\_\_\_\_, admitted in emergency/outpatient clinic, on \_\_\_\_\_ (or 24/48/72 hours ago, when we invoke an emergency admission or a further exploration), for the following symptoms: \_\_\_\_\_ (an array of maximum 3 or 4 major symptoms) [...]"*

**2. Reasons** for presentation: the main symptoms and signs reported by the patient, grouped by categories of symptoms or syndromes (digestive, respiratory, renal, etc...) will be enumerated here.

#### 3. History of the current disease:

At this stage we can state:

*"[...] The patient is admitted for... (symptoms or signs), which have evolved as follows [...]"*

- The current disease suddenly debuted in the following conditions... Or started insidiously, the first signs dating about a few days/weeks/months ago, etc...
- In fact, the disease started years ago and,

- It manifested itself by the following signs...
- Along the way it was complicated...,
- It was associated with...
- For these previously mentioned symptoms the patient is admitted in the clinic for diagnosis and specialized treatment;
- Affirmative, the patient presents... A series of data, when the claims of the patient cannot be controlled.

During the anamnesis, we should not be amazed or mislead by the volubility of the interlocutor, when after a few initial questions the patient can take control of the discussion, but we should conduct a guided anamnesis to avoid loss of time.

The presentation of clinical case should be tightly related to its peculiarities, avoiding the wording of the symptomatology of a disease as described in the semiology textbooks.

During the clinical case presentation, we can correlate and present as such traditional syndromes by their names (Saint`s Triad, Bouveret`s Syndrome, CHARCOT's sign, Owen's Triad, etc...).

**4. Interpretation of anamneses:** if the disease has a clear diagnosis, we can go with:

*"[...] It is probably the next disease/syndrome, to which we have been guided by the following manifestations [...]"*.

At this stage we will avoid disclosing as much of the positive diagnosis but, on the other hand, the board must/should be suggested our option for a particular diagnosis.

**5. Current status of the patient** comprises of:

- A. Overall general examination on apparatus, systems or segments as well;
- B. Local examination (detailed);
- C. Vaginal touch;
- D. Rectal touch.

Among the many variants of examination, we believe that the most appropriate in the conditions of competition, would be the simultaneous conduct of the objective examination with anamnesis. After the first preliminary questions and the determination of the anatomical area concerned to the greatest extent, we invite the patient to undress, obviously on segments, as we will also be carrying out the objective exam, starting with the cephalic region, thorax and upper limbs and then moving down towards the abdomen and lower limbs, at first in orthostatic position, then in the dorsal decubitus and finally in the ventral decubitus.

We also consider that the extremity of head, cervical region and thorax assume a more suggestive examination in orthostatic position, while the abdomen, lumbar region and lower limbs are exposed better to a complete examination in clinostatism/recumbency. Hernias, incisional hernias or varicose disease are easier to be identified in orthostatic position, while for palpation of the abdomen, it is best to position the patient in the dorsal decubitus.

Rectal and vaginal touch will necessarily end the objective exam, being essential in the complete construction of diagnosis for a surgical case, often disguised as an outbreak in the genital sphere, which is actually a surgical emergency.

**6. Presumptive diagnosis/suggestion of an affection:** this section usually starts with the following phrase:

*"[...] based on the data provided by anamnesis (symptoms), by the way they evolved and after the objective exam (recorded pathological signs), clinically, I interpret the case as a/an \_\_\_\_\_ [...]" or "[...] clinically, a condition in the sphere of \_\_\_\_\_, more specifically the organ \_\_\_\_\_ [...]."*

Under no circumstances will the positive diagnosis be stated without the benefit of pathological and imaging biological data!

**7. The paraclinical investigations** will be requested from the examination board, in close relationship with each sign or symptom stated previously. In order to remember them all, they can be grouped as it follows:

- For establishing the diagnosis of certainty: imaging/laboratory;
- For establishing the general balance of the patient: laboratory;
- For anesthesia and surgical procedure: functional explorations/laboratory.

It is highly recommended to mention only the pathological values and especially those that will later confirm the alleged diagnosis and diagnosis of secondary affections.

The candidate will reserve the right to comment and justify at each point of the biological or imaging data that is missing from the observation sheet, with important implications for strategy, technique and surgical approach, intraoperative incidents and accidents, all of which are directly targeted at post-operative complications.

## **8. Presumptive Positive Diagnosis**

In this section we can start with:

*"[...] at this time I allow myself to advance the positive diagnosis, with a character of presumption, suggested by anamnesis, supported by the objective exam and by the paraclinic explorations (laboratory, radiology, functional explorations, endoscopy, ultrasound, CT, MRI, etc.), establishing a complete positive diagnosis after differential diagnosis, and the diagnosis of certainty after intraoperative exploration and intraoperative pathology report [...]."*

The positive diagnosis will include:

- **Anatomical diagnosis:** organ and disease (eg.: duodenal ulcer, appendicitis, cholecystitis, pancreatitis);
- **Etiological diagnosis,** meaning the cause that triggered the disease: atherosclerosis or diabetes determines arteriopathy, migrated biliary stones lead to jaundice, perforation determines peritonitis, etc...
- **Physiopathological or functional diagnosis:** we state in what condition the organ or affected system is in compensated or decompensated

(decompensated secondary stenosis, myocardopathy in the stage of cardiac insufficiency, compensated chronic renal failure, etc...).

- **Topographical diagnosis:** this will later play a major role on the selection of the surgical technique and approach route adopted (classical or laparoscopic).
- **Diagnosis of evolutionary stage:** is it a chronic disease or an acute event? In this section we can anticipate the possibility of continuing medical, adjuvant or neoadjuvant treatment with the surgical one.
- **Diagnosis of anatomo-clinic form** (eventual **staging** where appropriate, especially for neoplasms, hiatal hernia, etc.).
- **Diagnosis of complications or pathological associations:** ruptured esophageal varicose veins, superior digestive hemorrhage, hemorrhagic gastric ulcer, secondary anemia, etc...

**9. Differential diagnosis:** will be performed according to the following criteria:

- Depending on the affected organ, but also in relation to neighboring organs;
- Depending on the main symptom (e.g.: pain);
- Depending on the main sign (e.g.: jaundice);
- Between the different clinical forms of the disease;
- Between the various evolutionary stages of the disease;
- In relation to major complications known for that particular disease: hemorrhage, occlusion, peritonitis, etc...

The alternatives to be further discussed (only some of the most important and most common) will be progressively excluded, either through the manifestations of the underlying disease, or through the negative or positive results of biological data or imaging explorations.

When the diagnosis is extremely obvious we can adopt the formula of avoiding the differential diagnosis all together: “[...] in the particular case that we examined, making a differential diagnosis would have a purely theoretical value and it is not justified, given the certainty provided by specific investigations [...]”.

**10. The complete positive diagnosis:** will be stated after the differential diagnosis, comprising all the data of the presumptive positive diagnosis, but this time formulated in a more complex manner. In addition, at this stage, the anatomopathological diagnosis will be stated where appropriate.

**11. The particularities of the case:** are represented by the particular elements that distinguish our case from the usual evolution known from the medical literature data, which the disease has in most cases, or on The complications or morally unusual associations that the patient presents, giving him a certain individuality.

**12. The evolution of the disease without treatment (the so called “natural evolution”):** the evolutionary possibilities of the disease will be stated in the absence of adequate treatment.



Generally, an untreated disease evolves into a chronic state and in time it is worsening, the possibility of a stationary evolution for years or even the chance of spontaneous remission only rarely exists. Most of the times, however, a local or general acute complication can change the course of the disease in a fatal way, sometimes leading to unwanted outcome.

At this stage we can state:

*"[...] It will have to be noted that if this patient is not treated, the following complications may occur in his/hers evolution \_\_\_\_\_ (local or general, organic or functional), which is why the prognosis may be a bad one or an even death [...]"*

**13. Medical treatment:** at this step we have the following options:

- The underlying disease depicts only a preoperative preparation treatment... / Stabilization of this acute event (antibiotherapy)... / Pain relief (antispastic, antalgic), anti-inflammatory treatment, hepatic rehabilitation in jaundice (hepatotropic, amino acids, albumin, vitamin K), etc...
- Regarding the associated affections of the underlying/main disease... Attempted haemostasis or restoration of volemia and rebalancing of hydro-electrolytic or acid-base... / or treatment of shock..., / or restoring vital functions... (specific measures of intensive therapy for the correction of shock, restoration of volemia, biological rehabilitation, restoration of vital functions, etc...);
- Related affections \_\_\_\_\_ (pulmonary, cardiac, renal, decompensated diabetes, etc) \_\_\_\_\_
- The patient exceeded the stage of medical treatment, so discussing him is purely theoretical, the patient being in a stage of preparation for surgery.

**14. Surgical treatment:** it is aimed at intercepting the cause of the disease, removal of the lesion, maintaining as much as possible the function of the organ of the body and the therapy of the complications and morbid associations.

Non-operative surgical treatment may consist in the maneuver of taxis in an incarcerated inguinal hernia, the primary processing of a burn, or the reduction of a closed or open fracture, emergency measures, sometimes taken for reasons to relieve the suffering of the patient until surgery.

Surgery will be adapted to the patient's age, anatomico-clinical form, evolutionary stage, particularities of the case and intraoperative findings.

**15. The diagnosis of possibilities for surgery:** results from the vital functions and imbalances as well as their correction possibilities, in conjunction with the state of the affected organ, the major emergency nature of the disease or the possibility of timing the surgery for a later stage. All this data will be analyzed for a more thorough study of the case and its risks

At this step we can continue with the clinical case presentation as follows:

*"[...] consequently, from the clinical trial in conjunction with the other data, it follows that the patient may undergo surgery [...]"*.

**16. Choice of surgical method:** the main question to be answered here is: radical or palliative?

At this step we can continue with the clinical case presentation as follows:

*"[...] in this case, in close regard to the general condition of the patient... and its associated conditions, I choose the radical/palliative variant of treatment [...]"*.

### **17. The choice of the surgical technique**

Along this step we present the choice of classical or laparoscopic (with its variants) approach and we present our motivation for one or the other.

At this step we can continue with the clinical case presentation as it follows:

*"[...] I'm opting in this case for an intervention... Because it is more readily tolerated by the sick, offers a better light on the lesion, the results are better, etc. [...]"*

### **18. Choice of surgical procedures**

The majority of known surgical procedures will be stated, removing the inappropriate ones for the case (exposing the reasons why), and selecting only a few of the most appropriate ones for the case (with motivation).

At this step we can continue with the clinical case presentation as it follows:

*"[...] In this case I would opt for... The final decision will be taken after the intraoperative exploration of the lesions and appearance of the anatomical region, which will decide the choice of the most appropriate method [...]"*

**19. Indications (I) and contraindications (CI):** all the evolutionary possibilities of the case will be evaluated, including the setting of the operator risk according to different risk scales (here you can set out statistics data, percentage of survival or complications, risk scales commonly used in literature, possibly, data from personal experience).

**20. Operator Timing:** surgical timing has to be well-chosen; the patient can be operated on the following days after a judicious preparation. Thus, a surgical intervention can be performed in 2 ways: a real emergency or after a short preoperative rebalancing (a "delayed emergency").

### **21. Preoperative preparation**

It is aimed at psychological training, balancing of biological functions, correction of metabolic and functional imbalances, premedication, local preparation of the organ concerned (colon, stomach) and the future surgical site and, most importantly, the patient's

consent after being informed in detail about the surgery and its possible consequences (both negative and positive).

This is the step of the final steps taken for ensuring a smooth and uneventful evolution of the case. Proper blood supply, cardiologic exam and anesthesia assessment, prophylaxis of postoperative thromboembolic disease (i.e. anticoagulant treatment).

## 22. The surgical intervention

It is the key moment of the clinical case presentation, in which the candidate will have to specify the following aspects:

- **Preanaesthesia;**
- **Anaesthesia:** should provide analgesic, relaxation, anesthetic sleep and tranquility in the operator's field. It is paramount to choose the proper anesthesia technique in close relationship with the surgical pathology. For example, under no circumstances will spinal anesthesia be chosen for a patient who presents an incisional hernia, for which we choose to repair the parietal defect with textile material (allograft);
- **The description of the intervention:** will have to comprise the objectives, the principle of operation, the surgical technique, discussing the main surgical steps and the variants of the incision when opting for classical surgery or trocar placement when opting for the laparoscopic route; laparoscopic technique involves the same objectives, obtained with various instruments, which must be remembered together with the operative device and the principles of laparoscopic methods;
- **Surgical exploration:** involves the complete inventory of infra-and supramezocolic viscera (possible laparoscopic and only rarely open), and then insist on the anatomical-pathological aspect of the lesion;
- **Discussing the opportunity of prophylactic antibiotherapy (24 hours),** is addressed exclusively to local or regional contaminations occurring during the intervention, without being able to substitute for preoperative preparation measures.

## 23. Incidents and intra-operative accidents

Regardless of the complexity of the case and the numerous possibilities of intra-operative accidents, the candidate will refer only to those incidents strictly subordinate to the disease (nature, localization, stage).

The vascular, visceral, nerve, ureteral lesions, the main or hepatic biliary pathways and the way of solving them should be taken into account, the most important aspect related to these being the intraoperative recognition of the lesions.

**24. Postoperative care:** postoperative care begins with the completion of surgery, still on the operating table and runs throughout the entire period of the hospitalization.