BOOK 2

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NEUROLOGY
IN CLINICAL CASES

Dr. Kamen Genadiev Kamenov

NEUROLOGY IN CLINICAL CASES

BOOK TWO

This book combines in itself real clinical experience with practice-oriented theoretical knowledge in neurology. Its 70 documents present a total of 85 clinical cases that are illustrated with altogether 593 images and 14 classifications tables. Besides, there are also 9 short biographies that present 9 physicians who have contributed to the development of the neurology as a science worldwide.
We wish a pleasant and fruitful work to the readers of the book.

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Author's photography on the title page:
Karimski volcano, Kamchatka, Russia

Table of contents

077. The subdural haematoma: when its treatment should be surgical

This 75-years-old man has been admitted into our hospital two hours earlier. In fact, her wife and her daughter have accompanied him because since the last few days his gait had become unstable even if he had no falls at all. In addition, the members of his family have noticed that he has become confused and that he could not anymore carry out some of his usual everyday activities in a correct way – for example, now he cannot button and unbutton his cloths and during the last two days when eating he was not using the table set but he eats directly with his fingers).

The man is right-hander and he passes most of his time being alone in nature, not far away from his village, and, by character, he is not talkative.

He has no known allergies, since the last ten years, he has arterial hypertension that is well-balanced under appropriate drug treatment and since the last five years he has a type 2 diabetes mellitus that is also well-balanced under appropriate diet and per oral antidiabetic treatment.

In his life, he has had two operations: a varicose veins operation in both lower limbs approximately 20 years ago and an appendectomy in the distant past.

His wife confirms a moderate but regular alcohol consumption adding that his man has the habit to drink secretly. And she also tells us that 19 days earlier his husband has fallen backwards and has hit the back of his head on a concrete surface; that same day he has been examined in the emergency ward of our hospital and because his clinical examination was unremarkable he has entered his home the same day.

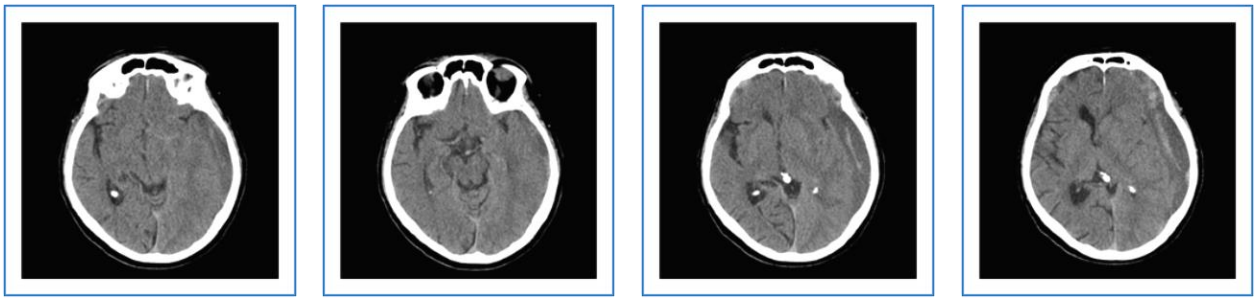
Our neurological examination at his admission reveals the presence of the following problems:

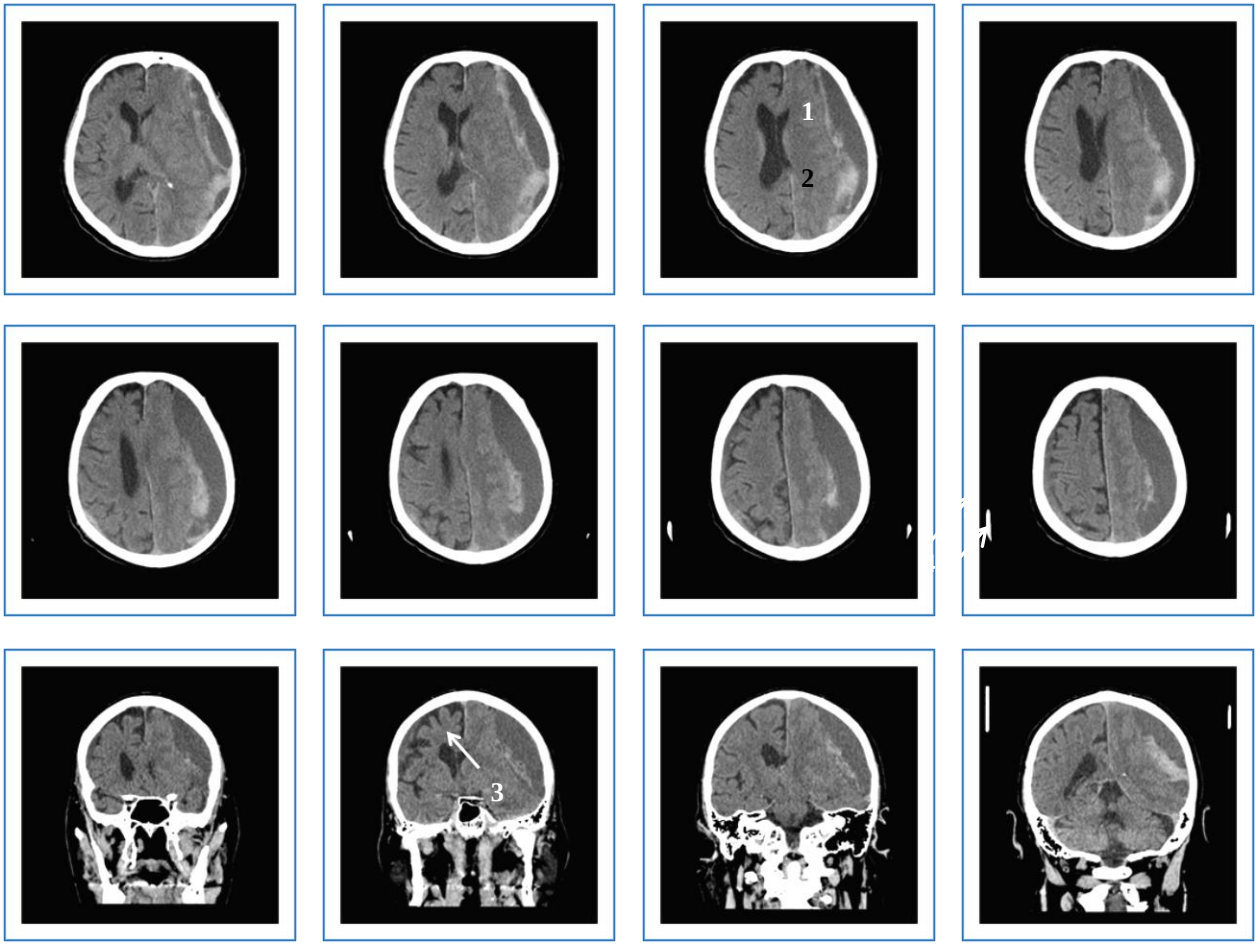
- a disorientation in time: «Today is the 1st of March (May) 2016» but the real date if the 1st of June 2016; in the same time he is oriented for the place and for the situation (he knows the city name and he knows that he is in a hospital;
- a slight weakness of his right upper limb at 4+/5;
- a slight ideomotor apraxia;
- urinary incontinence (wet panties, smell of urines).

The somatic examination did not show any notable features.

In this situation, performing a brain scan becomes a must: the main goal is to look for the presence of suffering of the dominant (left) cerebral hemisphere in this right-hander.

The brain scan has been done immediately and it showed the following result:





Presence of a large subacute subdural haematoma (**1**) with a recent bleeding (**2**) with a maximal thickness of 35 mm and shift of the midline structures (**3**) of approximately 8 to 10 mm with subfalcine herniation. *All these problems are due to the mechanism of direct injury (coup injury).*

Presence also of a subdural haematoma on the right (**4**) with a thickness of 10 mm. *This problem is due to the mechanism of counterblow (contrecoup injury).*

The result of the brain scan allows establishing **the aetiological diagnosis**, explaining **the neurological deficits** and choosing **the adequate therapy** for this patient:

- **the aetiological diagnosis** is the presence of subacute subdural haematomata due to the head trauma that has happened 19 days ago: the left-side subdural haematoma is due to the coup injury and it causes this patient's clinical symptoms and the right-sided subdural haematoma is due to the countercoup injury causing apparently no clinical symptoms at the moment this diagnosis is made;
- **the neurological deficits** are due to the compression of the parenchyma of the left brain hemisphere that is the dominant brain hemisphere in this patient;
- **the adequate therapy** in this patient's case is neurosurgery.

Meanwhile the blood analyses are ready: the coagulation is correct, there is no inflammatory syndrome but the following values are pathological: **haemoglobin at 177 g/l**, **GGT 2,5 times higher than the normal, ASAT and ALAT just over the upper limit of the normal, glycaemia at 8,4 mmol/l, total proteins at 86,2 g/l, urea elevated at 10,1, calcium elevated at 2,62 mmol/l.**

As a result of all that we have done, we have immediately contacted our neurosurgical colleagues who have continued the therapeutical care for this patient: he has been operated on in the evening of this same day of his hospital admission and he presented a progressive and finally complete recovery of his neurological deficits, so he could return to his home.

This is the typical clinical picture in case of chronic or subacute subdural haematoma: an elderly person (with regular alcohol consumption) who has had a head trauma (days earlier) in general without an immediate gravity and who presents an afebrile confusion and focal neurological deficits.
After the anamnesis and the clinical examination, the aetiological diagnosis is established thanks to the brain imaging studies (brain scan or brain MRI).
The decision about the optimal therapeutical approach belongs to the neurosurgeons.

In order to know more

The subdural haematoma

The subdural haematoma (**SDH**) is the result of a head trauma that leads to the accumulation of blood between the inner layer of the dura mater and the arachnoid that covers the brain. This head trauma sometimes is so mild that it passes unnoticed in the everyday life.
The origin of this blood in the vast majority of cases of SDH is venous, due to the rupture of one or several veins that cross the subarachnoid space. There are also a few cases of SDH of arterial origin – in this case the reason for the bleeding is a lesion of one of the arteries that crosses the subarachnoid space. Sometimes there is a second source of bleeding and that is the damaged brain parenchyma – this type of lesion can accompany the vascular lesion.
According to their evolution in the time the subdural haematomata are classified as

- acute (appearance from a few minutes to a few days after the trauma);
- subacute (this category is being disputed by some authors);
- chronic (appearance several weeks after the trauma).

After the brain trauma there is a shorter or longer lucid interval followed by the appearance of clinical signs. The clinical presentation is quite variable and it depends on the speed of formation of the subdural haematoma, on its volume and on its localisation:

- signs of intracranial hypertension,
- signs that not always allow the localisation of the SDH, ex. gr. afebrile confusion, blurred vision, generalized epileptic seizure or cognitive impairment in case of slow evolution and
- localizing signs, most frequently presenting as focal deficits.

Some subdural haematomata (mostly the chronic SDH) could have no clinical signs and they are found by chance with a brain imaging study (scan or MRI) has been done for another reason.
The final diagnosis is established by the brain imaging, mostly by the brain scan and, more rarely, by the brain MRI.
The optimal therapeutic result for the subdural haematomata depends on several factors: the clinical presentation, the volume of the subdural haematoma and the patient's general health state.
The SDH without or with very few clinical signs, without mass effect on the brain parenchyma and the SDH in case of patients in poor general health state are treated by good hydration and, if necessary, by symptomatic therapeutic measures. The prescription of corticosteroid in case of subdural haematoma is a therapeutic option that is not adopted by all the physicians.
The subdural haematomata that cause clinical deficits and / or mass effect on the brain parenchyma are subject to surgical treatment. The classical operation is the haematoma evacuation by drainage after trepanation. The voluminous subdural haematomata that cause important clinical deficits could need a more consistent neurosurgical operation in order to evacuate the blood and the coagula that have been formed and in order to ligature the bleeding vessels. In any case, the choice of the intervention type depends on the decision of the neurosurgeon(s).
A timely treatment of a subdural haematoma allows a good healing and vice versa.

Predisposing factors for the formation of subdural haematomata

The risk factors for the formation of subdural haematomata are the following ones:

- the head traumas in general and, among them, mostly the traumas with acceleration – deceleration and also the head traumas that cause torsion of the central nervous system structures;
- the cerebral atrophy: as a consequence of the loss of a part of its substance the brain is no more in contact with the inner table of the skull and thus the veins that traverse the subdural space can be damaged very easily;
- the age: in the classical cases both extremes of the age – the children and the elderly – are concerned;
- the presence of cardiovascular diseases (arterial hypertension);
- the vitamin-K antagonists or the antiplatelet drugs;
- the thrombocytopaenia;
- the diabetes mellitus;
- the disorders of the coagulation;
- the chronic consumption of alcohol;
- the subarachnoid cysts;
- the (spontaneous) intracranial hypertension;
- the chronic dehydration.

In some cases of SDH there is neither identifiable cause, nor identifiable risk factor for their appearance and because of this we speak of spontaneous subdural haematomata (*for more information on them please consult the section « In order to know more » of document 078 «* **The subdural haematoma: when it should not be operated ».***)*

Basic principles for the therapeutic approach to the subdural haematomata

The timely treatment of a subdural haematoma allows a complete healing in most cases and, vice versa, in case of delay of this treatment the healing could be incomplete.
The decision if a subdural haematoma should or should not be operated on is taken by the neurosurgeons on the basis of the general state of the patient, his / her level of consciousness, the presence or the absence of focal neurological deficits and the results of the brain imaging studies.
On the part of the neurologist it should be a serious fault to treat alone a patient with subdural haematoma without presenting him to a neurosurgeon and without discussion this patient's treatment with the neurosurgeon.

097. Suffering of the femoral nerve: always think about the hip joint (2)

This morning during the medical visit in our ward we take care of a new patient: she is 62 years old and she has been admitted in the hospital 36 hours earlier because of fever (39,1 °C = 102,4 °F) and strong pain in her right lower limb, mostly in the right inguinal fold with irradiation along the anterior surface of the right thigh.

She arrives in the ward with the diagnosis of sepsis with unknown aetiology and unknown primary localisation. The colleagues from the emergency ward have already started an antibiotic treatment with two antistaphyloccocal antibiotics: vancomycin in permanent perfusion with a rate of 30 mg / kg b.w. / 24 hours with yet undetermined duration and gentamicin in a dose of 3 mg / kg b.w. / 24 hours for 3 days.

Now we have to continue the medical care for this patient who has a sepsis and a neuropathy of the right femoral nerve. And we have to answer the following questions:

- where is the infectious focus?
- what is the causative agent (aerobic and anaerobic blood cultures have already been taken before the beginning of the antibiotic therapy)?
- what should be the optimal curative treatment?
- how could we optimize the symptomatic treatment?

As always, the best thing to do is to begin from the beginning, i.e. to take the medical history once again. It allowed us to understand that the degradation of our patient's health has started more than one month earlier with pain during walking in the right hip joint. Little by little this pain has become stronger up to the point when the patient could barely walk. The patient has not lost weight but her appetite is not the same as before and she has noted that a low-grade fever has been almost constantly present during the last week.

This patient does not have any known allergies, she has not been operated in her life and she does not have any chronic illness, so she does not take any medication.

The clinical examination shows a difficulty in carrying out the passive and the active movements in all direction in the right hip joint and these limited movements are very painful. Moreover, the gentle pressure over the right femoral head (in the middle of the right inguinal fold) provokes a quite strong pain but the same manoeuvre is painless in the left inguinal fold. The rest of the clinical examination is without particularities.

The initial biological analysis shows an acute inflammatory syndrome: polynuclear hyperleukocytose (16830 leukocytes per mm cube with 79 % polynuclear ones), a CRP of 558.5 mg/l, a procalcitonin of 0,58 µg/l, an erythrocyte sedimentation rate of 75 mm / 1st hour, the alkaline phosphatases are elevated at 1.5 times the upper normal value. The rest is without particularities. The urinalysis proved to be without particularities.

On the third day of this patient's in-hospital treatment we received the result from the blood cultures: growth of a Staphylococcus aureus resistant to methicillin. So, for the time being the antibiotic therapy seems to be correct but we still miss the primary localisation of the infectious process (even if we already think about an infectious pathology of the right hip joint). We note that a whole body scan has been done in the emergency ward but it did not allow finding where the infection was.

In the meantime a heart ultrasound has been done and it proved to be normal (excluding the possibility for an endocarditis in this patient).

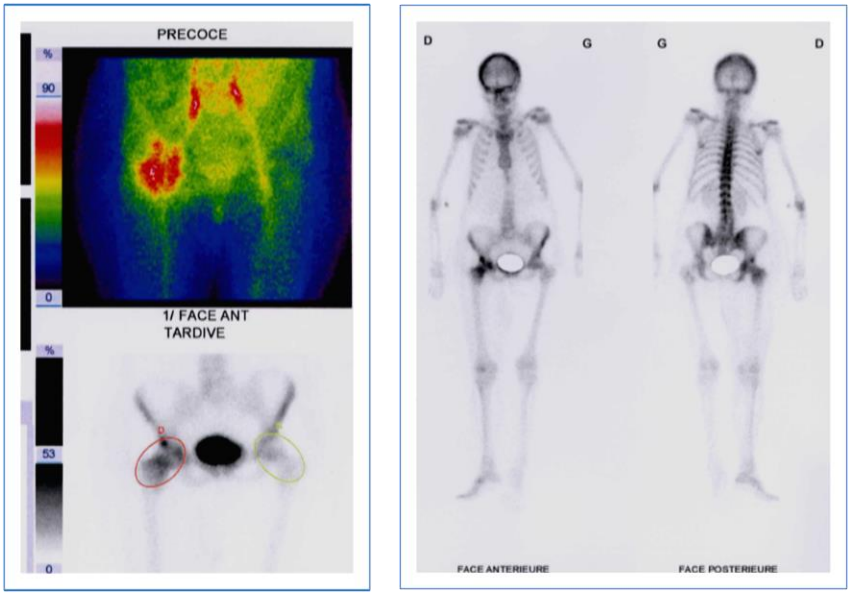
But the problem is not resolved, up to now we could simply exclude some gravity (by exclusion of the endocarditis) in this case of sepsis due to Staphylococcus aureus resistant to the methicillin.

What should be done?

The best diagnostic option at that moment is to ask for a bone scintigraphy: this type of examination is perfectly adapted for the discovery of bone pathology, and it is very sensible in case of infectious bone pathology.

This examination could be done on the next day, the fifth day of medical care for this patient, and its result was clear: this patient had osteoarthritis of the right hip joint.

At this point we already disposed of the aetiological bacterial diagnosis and of the anatomical localization of the infectious problem.



Bone scintigraphy with ^{99m}Tc methylene diphosphonate (MDP): **Scintigraphy in favour of osteoarthritis of the right hip joint**.

So now we already disposed of the necessary clinical and para clinical data in order to optimize the antibiotic treatment: the I.V. antibiotic therapy that has been chosen for this patient was the following one:

- vancomycin in continuous perfusion in a dose of 30 mg / kg b.w. / 24 hours and
- rifampicin in a dose of 15 mg / kg b.w. / 24 hours in 2 perfusions of one hour each.

The initially previewed duration of the antibiotic treatment was 7 weeks (the actual recommendations in case of this pathology are for such antibiotic treatment with a duration between 6 and 12 weeks).

The patient has been planned for a specialized orthopaedic consultation.

Of course, alongside with the specific antibiotic treatment this patient has had appropriate pain medication and also all complex of measures necessary for the primary prevention of the possible complications for her prolonged bed rest.

In order to know more

The bone scintigraphy

The scintigraphy is a medical imaging examination and more specifically it is a nuclear medicine emitting imaging examination. It consists of the delivery into the human body of a gamma radiation emitting product that is called radiopharmaceutical. The signals of this radiopharmaceutical are being registered by a gamma camera.
The radiopharmaceutical itself consists of carrier and of a radioactive marker.
The carrier is different according to the specificity of the examination.
The radioactive marker is also different according to the different organs that are being examined.
The scintigraphy could be bone, kidney, myocardium, lung, thyroid, digestive, hepatobiliary, brain and breast scintigraphy.
In case of bone scintigraphy the carrier is a bisphosphonate and the radioactive marker is technetium-99m (99mTc).
The concentration into the bones of the radiopharmaceutical that is specific for the bone scintigraphy is due mostly to the fact that the bisphosphonates participate in the bone metabolism and more specifically in the osteoblast activity, i.e. in the formation of new bone.
The bone pathologies that could be examined by the bone scintigraphy have multiple aetiologies and they could be acute ones and chronic ones: bone infections, primary and secondary bone tumours, algodystrophy, vertebral compression fractures, inflammatory arthropathies, osteomalacia, multiple myeloma…
In most cases the zone of bone pathology captures the radiopharmaceutical more avidly than the normal bone tissue, so there is a hyperfixation. More rarely the pathological bone could be with hypofixation, ex. gr. in case of osteonecrosis, after radiotherapy, in case of lytic bone metastases.

Our patient has been admitted to the hospital in a state of sepsis, so we would like to specify this medical condition:

The sepsis

The sepsis is the combination of a systemic inflammatory response syndrome and a bacteraemia.

The classical definition of sepsis from 1992 establishes three stages for the sepsis according to the increasing gravity of the clinical picture. Nowadays there is a fourth stage added, the mildest one, and it is named systemic inflammatory response syndrome:

1. Systemic inflammatory response syndrome.
2. Non-complicated sepsis.
3. Sepsis complicated by the acute failure of one or several organs.
4. The septic shock is a severe sepsis with arterial hypotension that is resistant to adequate vascular fluid filling in the dose of 20 to 40 ml / kg b.w.

The aetiology can be bacterial, viral or fungal.

The clinical presentation in the time can be hyperacute, acute, subacute, chronic and recurrent.

The issue from the sepsis is not always a good one and the prognosis is becoming worse with the advance of the sepsis stage.

The long term (5 years) vital prognosis in patients who have survived a sepsis is worse (they become ill more frequently and their death rate is higher) as compared to people who have not had sepsis.

The systemic inflammatory response syndrome (SIRS)

This syndrome is a systemic response of the body to a severe external aggression that is not always of infectious origin. Its diagnosis requires at least 2 of the following signs:

- temperature either > 38,3 °C or < 36,0 °C;
- tachycardia (i.e., heart rate > 90 heart beats per minute);
- polypnea (> 20 respiratory cycles per minute) or hypocapnia (pCO_2 < 32 mmHg);
- either hyperleukocytosis > 12 000 / ml, or leukopenia < 4 000 / ml, or 10 % of immature cells in the peripheral blood (after exclusion of other causes for their presence).

Non-infectious causes for this syndrome: inflammatory diseases, tissue destruction (major surgery, traumas, subarachnoid bleeding, pancreatitis, tissue infarction…), reactions to some treatment (for ex. blood transfusion…), metabolic causes (acute adrenal insufficiency, hyperthyroid crisis), tumoral pathologies.

The Staphylococcus aureus

The Staphylococcus aureus is a Gram + microorganism that forms grape-like clusters and these structures have given its name (« staphylo » coming from ancient Greek and meaning « brunch of grape » and « coque », coming from the same language meaning « raisin » or « seed »). The adjective "aureus" comes from the Latin meaning "golden" and characterising the spongy yellow colour that have the Staphylococcus aureus colonies.

The Staphylococcus aureus is the most pathogenic species of the Staphylococcus genus.

The Staphylococcus aureus is ubiquitous and it is both commensal and pathogenic for the human beings. It is a *mesophile* (the optimal temperature for him is 37 °C), *neutrophile* (the optimal pH for it is 7,0), *halophile* (it tolerates the high concentration of salt), *thermosensible* (its growth is retarded in cold ambiance but it can survive; on the contrary, it is killed by freezing; it is easily killed by the high temperatures: in 1 minute at 78 °C and in 10 minutes at 64 °C), it is *optional aero- and anaerobic* and it has a *strong capacity of mutations* (that is one of the factors for its resistance to the antibiotics). At enzymatic level the Staphylococcus aureus is coagulase +, oxydase – and it ferments the glucose without gaz.

The Staphylococcus aureus is presented as a commensal by 15 to 30 % of the human being at the following preferred localisations: the nose, the throat, the perineum.

Its pathogenicity is being explained by the fact that it possesses an *invasive capacity* (it could multiply and disseminate into the organism mostly because of its enzymes: coagulase, fibrinolysin, phosphatase, hyaluronidase, protease, deoxyribonuclease) and it also possesses *a toxic capacity* by the production of toxins (staphylolysines, leucocidins and, in some strains, enterotoxins).

The pathologies induced in humans by Staphylococcus aureus are multiple: osteomyelitides (responsible germ in 90 % of the cases), meningitis, endocarditis, pneumonias, urinary tract infections, phlebitis, enteritis, acute myositis and different localized purulent infections: furuncles, whitlow, folliculitis…; sepsis and also **food intoxications**.

The possession of the enzyme penicillinase (a type of β-lactamase) makes the Staphylococcus aureus resistant to the penicillin. This enzyme cannot protect the Staphylococcus aureus from the antibiotics methicillin, nafcillin, oxacillin, cloxacillin, dicloxacillin and flucloxacillin. On the contrary, even these antibiotics are powerless in the presence of Staphylococcus aureus that are resistant to the methicillin = Methicillin-Resistant Staphylococcus Aureus (MRSA). Since not long ago there are also strains of Vancomycin-Resistant Staphylococcus Aureus (VRSA).

There is no efficient vaccine against Staphylococcus aureus. At the time being there are efforts to achieve the inhibition of this germ by the local introduction of the inhibiting strain JK16 of Staphylococcus epidermidis. The action mechanism here is the amensalism.

The consumption of hot tea and / or coffee diminishes the carriage of Staphylococcus aureus in the nasal cavity, including the carriage of MRSA: certain constituents of the essential oils that are contained in the tea and in the coffee make the bacterial membrane permeable and that finally leads to the death of the bacteria.
The prevention and the treatment of the infections with Staphylococcus aureus include hygienic measures in the everyday life both in the hospital as well as outside the hospitals.

The MRSA

The **M**ethicillin-**R**esistant is known since 1961 when it has been isolated for the first time in a patient in the United Kingdom, only two years after the introduction of the methicillin for the treatment of infections with penicillin-resistant Staphylococcus aureus. Since that time, it has been discovered all over the world. Recently a new strain of MRSA that is pathogenic to humans has been detected. This strain is of bovine origin, and it is undetectable by the usual research methods.
It is believed that the MRSA appeared because of a natural selection of the Staphylococcus aureus towards the beta-lactam antibiotics.
The clinical presentations of the infections with the MRSA are very varied: from an asymptomatic carriage up to septic shock. The antibiotic treatment should be chosen according to the clinical situation. The study of usage of bacteriophages against the MRSA is underway.
The prevention is based on protective measures concerning the MRSA contact persons (contact isolation and disposable protective clothing when taking care for MRSA patients), surface disinfection with products that are bactericidal for the MRSA: alcohol, quaternary ammonium, NAV-CO2 systems (**N**on-flammable **A**lcohol **V**apour in **C**arbon **D**i**O**xyde systems).

The VRSA

The **VRSA** is the **V**ancomycin-**R**esistant **S**taphylococcus **A**ureus.
The **VRSA** is known since 1997 when it has been isolated for the first time in Japan. Since that time, it has been identified in Asia, in the United Kingdom, in France, in the USA, in Brazil.
There are 3 strains of VRSA with different resistance to the vancomycin: VISA, hVISA and VRSA.
The **VISA** (**V**ancomycin **I**ntermediate **S**taphylococcus **A**ureus) is the strain of VRSA that has been detected first. It resists to the vancomycin because of a thickening of its wall that diminishes the capacity of this antibiotic to penetrate and to exert its bactericidal action. It is also known under the name of **GISA** (**G**lycopeptide **I**ntermediate **S**taphylococcus **A**ureus) and this name is used to underline the fact that this strain of Staphylococcus aureus is resistant to all glycopeptide antibiotics. The treatment of first choice: ceftobiprole (fifth-generation cephalosporin).
The **hVISA** (**h**eterogeneous **V**ancomycin **I**ntermediate **S**taphylococcus **A**ureus) has a resistance to the vancomycin that is observed only if this bacterium is cultured at a temperature < 37 °C.
The **VRSA** (**V**ancomycin **R**esistant **S**taphylococcus **A**ureus) has been initially isolated in the United States in a diabetic patient under dialyse. Since July 2013 the first cases of VRSA have appeared in Europe. Its high resistance to vancomycin has been conferred to it by genetic transfer from strains of Enterococcus faecalis. In fact, these are two genes that code for a resistance to antibiotics:

- the mecA gene that codes for the methicillin resistance and
- the vanA gene that renders the microorganism resistant both to vancomycin and to teicoplanin.

The treatment of first choice: trimethoprim / sulfamethoxazole.

116. The first demyelinating episode: its evolution can be so different from one clinical case to another (1st case)

This new 41-years-old patient comes for consultation after a phone call on the part of her G.P. who insisted for a consultation the same day, because "she was not well, and she could get paralyzed".

Her actual medical problem has begun about 4 weeks earlier in an insidious way, with the appearance of sensations "as if electricity was running down her body" when she was bending her head forwards.

She has also begun dropping objects with her left hand, her writing worsened and now, in order to stabilize the pen, she has to hold it in a different way, not as usual.

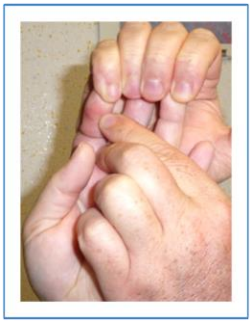
It is important to note that this patient is a natural left-hander.

There is no other particularity in her medical history: the patient has no allergy, she does not take any medication, she has never been operated in her life and she has an administrative job.

Then we proceed to this patient's clinical examination. Her somatic examination does not reveal any particularity. On the contrary, her neurological examination shows the following problems:

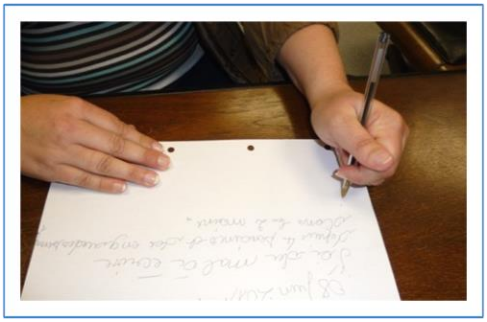
- a positive Lhermitte's sign (a sensation of electricity that runs down the body starting from the neck at the moments when the head is bent forwards;
- the deep tendon reflexes are present at + everywhere except for the right upper limb, where they are ++;
- the Wartenberg's sign is positive on the right;
- the Strümpel's sign is positive bilaterally;
- latent weakness of the left superior limb with
- loss of the fine motricity of the fingers of the left hand and
- an obvious dysmetria for the finger – nose test on the left.

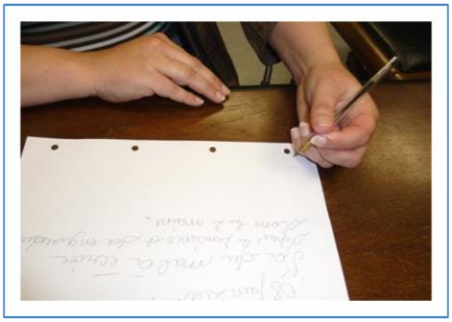
Illustration of the test for the Wartenberg's sign in our patient:



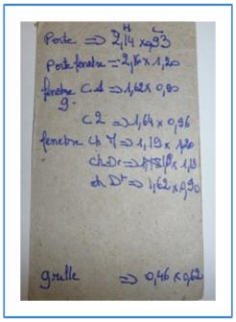
It is clearly positive for her right hand, and it is negative for her left hand (for details please see below in the section "In order to know more").

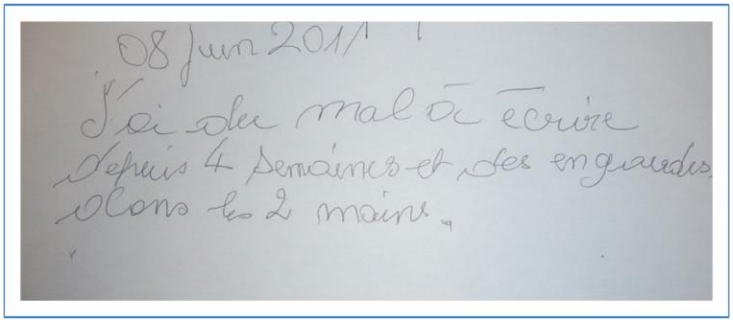
Illustration of the difficulties to write that our patient has (we remind that she is natural left-hander):





On the 1st image: the actual way she holds the pen (in this way the patient tries to compensate the loss of the fine motor skills of the fingers of her left hand) and on the 2nd image: the usual way for her to hold her pen.





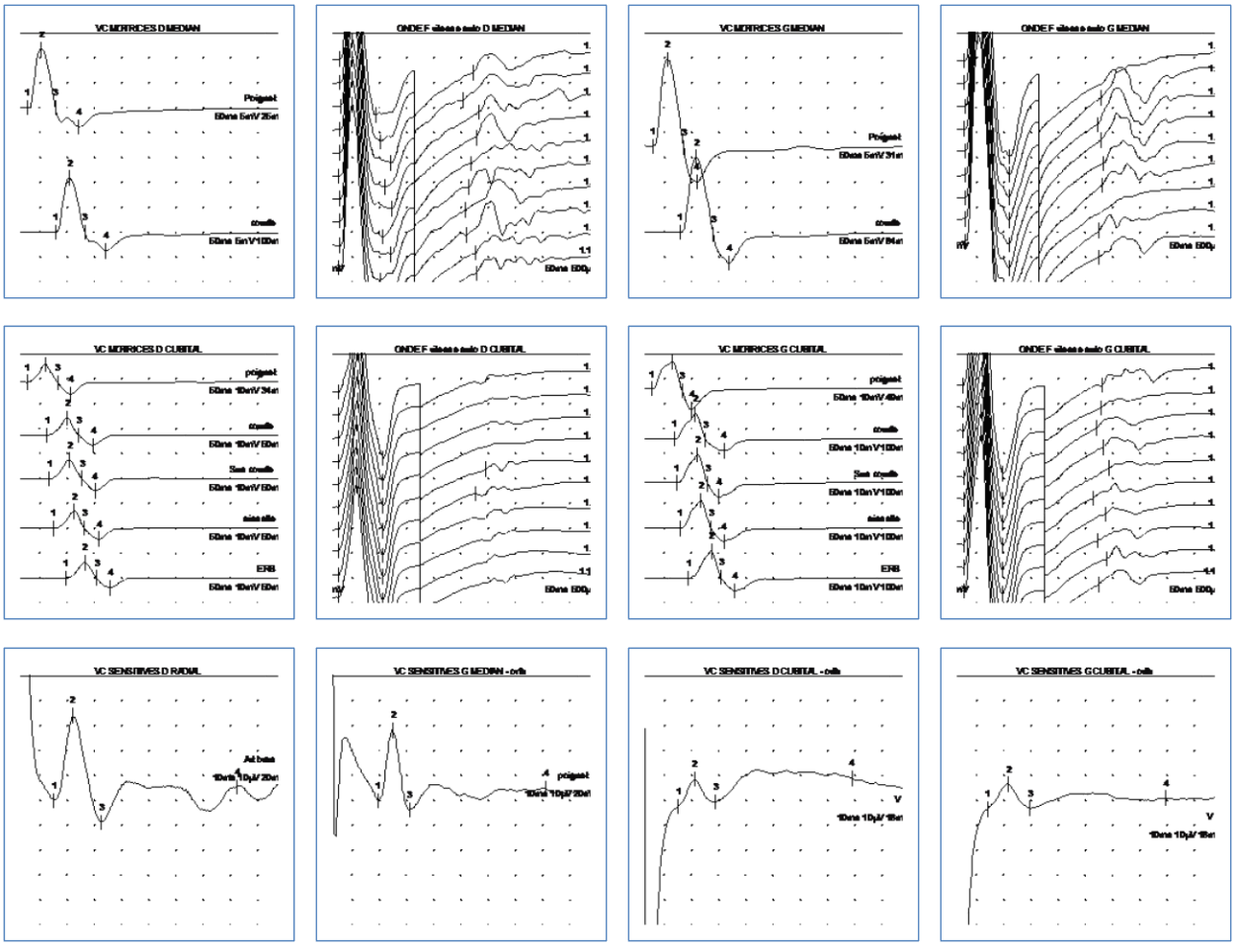
The result is the degradation of our patient's writing: on the 1st image is represented this patient's normal writing and on the 2nd image is represented her actual writing since the installation of the loss of the fine motor skills of her left hand.

On the basis of this data from the patient's medical history and from the result of her neurological examination we dispose of all, that is necessary in order to be able to establish the anatomical site where this patient's neurological problem is located. Here the main aid comes from the description of the Lhermitte's sign that indicates **a problem at the level of the cervical segments of the spinal cord** (for more precisions please see below in the section "In order to know more").

We started the paraclinical workup for this patient with the following examinations:

A scan of the cervical segments of the spinal column showed the beginning of a cervical arthrosis, mostly present at the C5/C6 level on the left but without narrowing of the vertebral canal at cervical level, so no arguments for an extrinsic compression of the cervical spinal cord.

An ENMG that turned out to be completely normal and that allowed excluding the presence of a generalized or localized (at cervical level) suffering of the peripheral nervous system:



Results of the motor conduction study:

Nerve / Sites	Latence, ms	Amplitude 1-2, mV	Surface, mVms	Decrease S, %	Distance, cm	Velocity, m/s
RIGHT MEDIAN NERVE						
Wrist	2,80	12,0	34,6	100		
Elbow	7,80	11,1	32,8	94,8	29	58,0
LEFT MEDIAN NERVE						
Wrist	2,90	17,5	55,4	100		
Elbow	8,00	15,1	48,6	87,7	28	54,9
RIGHT ULNAR NERVE						
Wrist	2,70	7,5	19,5	100		
Below the elbow	6,40	6,9	17,7	90,7	23,5	63,5
Elbow	6,85	7,5	20,5	106	2,9	64,4
Above the elbow	7,60	6,9	18,0	92,4	4,1	54,7
Armpit	9,70	6,2	17,6	90,6	11,3	53,8
LEFT ULNAR NERVE						
Wrist	2,80	11,4	37,8	100		
Below the elbow	6,95	10,2	30,5	80,6	23,3	56,1
Elbow	7,35	11,3	33,3	88,1	3,2	80,0
Above the elbow	7,95	11,1	31,9	84,4	4	66,7
Armpit	9,50	10,9	30,3	80,2	12,2	78,7

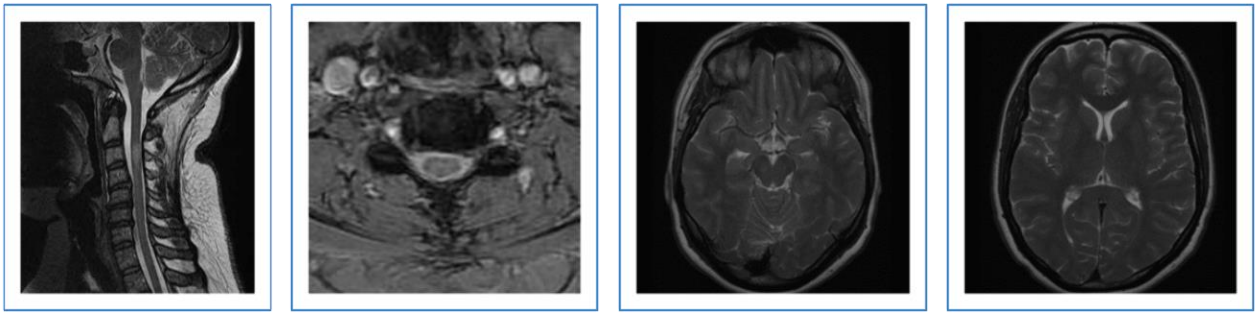
Results of the study of the F waves:

Nerve	Minimal latency F, ms	Distance, cm	Velocity F, m/s	Minimal F-M, ms
RIGHT MEDIAN NERVE	25,40	73	67,1	22,70
LEFT MEDIAN NERVE	27,35	73	61,7	24,50
RIGHT CUBITAL NERVE	27,70	73	60,5	25,05
LEFT CUBITAL NERVE	26,40	73	63,9	23,65

Results of the sensory conduction study:

Nerve / Sites	Site	Latency 1, ms	Amplitude, µV	Distance, cm	Velocity, m/s
RIGHT MEDIAN NERVE – ortho Wrist	Длан	1,75	39,7	8,2	46,9
LEFT MEDIAN NERVE – ortho Wrist	Длан	1,95	32,4	9	46,2
RIGHT ULNAR NERVE – ortho Wrist	Длан	1,50	10,9	8,5	56,7
LEFT ULNAR NERVE – ortho Wrist	Длан	1,40	9,8	8,2	58,6
RIGHT RADIAL NERVE – retro Anatomical snuffbox	Предмишница	1,50	43,3	9	60,0

The patient's cerebral MRI and spinal cord MRI showed the following results:



Presence of a plaque of demyelination in the cervical spinal cord at the level of the vertebrae C3 and C4. This plaque has a length of 2 centimetres (1st image), it is situated in the centre and in the posterior part of both halves of the spinal cord; this lesion is incomplete as it does not concern the ventral and the lateral parts of each half of the suffering segments of the spinal cord (2nd image).
The cerebral MRI turned out to be completely normal. It is represented here by images 3 and 4.

After that the standard blood analysis has been completed by a specialized blood analysis in case of demyelinating disease of the central nervous system (for details please see the section "The blood analysis in case of demyelinating disease of the central nervous system" of our document 104 "Multiple sclerosis: the clinical beginning of a typical case (1st case)". This blood analysis turned out to be completely normal in our patient.

At this moment, in order to complete our patient's paraclinical examinations we needed information if there is immunological activity in the central nervous system or not.

Hence, this patient had also a lumbar puncture that gave the following results:

Studied parameter	Test tube 1	Test tube 2	Test tube 3
Aspect before the centrifugation	limpid	limpid	limpid
Erythrocytes	1 / mm^3	0 / mm^3	0 / mm^3
Leukocytes	0 / mm^3	0 / mm^3	0 / mm^3
Proteinorachia	0,20 g / l		
Glycorachia	3.30 mmol / l (for a capillary glycaemia of 5.00 mmol / l)		
Bacteriological study	no visible microorganisms		
Bacterial culture	negative (result available in 48 hours)		
Mycological culture	negative (result available in 48 hours)		
Search for Koch's bacillus	negative (result available in 7 days)		
Search for Borrelia burgdorferi	absence of intrathecal synthesis of anti-Borrelia antibodies (IgG antibodies < 10 kU/l, IgM antibodies: negative; index 0.0)		
Търсене на интратекална синтеза на IgG	**Presence of intrathecal synthesis of IgG** **confirmed by the isoelectric focusing** albumin: serum 51.40 g/l; CSF 142.00 mg/l; IgG: serum 12.90 g/l; CSF 43.10 mg/l; Albumin Quotient at 0.28 (superior limit of the norm at 0.65); Ratio CSF IgG / CSF albumin at 0.25 (superior limit of the norm at 0.24); Ratio serum IgG / serum albumin at 0.30; Index IgG = IgG /albumin CSF // IgG / serum albumin at 1.20 (superior limit of the norm at 0.65)		

Based on these data from the medical history, the clinical examination and the paraclinical examinations we could conclude that our patient has presented a first demyelinating episode of her central nervous system with a functional suffering estimated, according to the EDSS scale, at 3.5.

We took the necessary time to discuss with her about the nature of her problem and the therapeutic options that are available, and we let her think over the information that she got from us.

Finally, she accepted a disease-modifying treatment and she got interferon-beta-1b at 250 µg, S.C., once every two days. Her tolerance to this drug was very good and at clinical level we noted a progressive reduction of her handicaps with a reduction of her EDSS score at 2.0 with a return to work as usual.

This patient's regular clinical, biological and MRI follow-up during 5 and half years showed the following evolution:

- appearance two years and nine months later of a second attack of her demyelinating disease with some difficulties walking and micturition problems (neurogenic bladder); the MRI of her spinal cord showed a mild increase of the size of her already known demyelination plaque at cervical level that now was Gd+ and her cerebral MRI showed the appearance, for the first time, of demyelinating plaques at cerebral level – this all indicates that this patient's demyelinating disease demonstrates a dissemination in the time and a dissemination in the space and thus her first demyelinating episode has been transformed into *a clinically, biologically and radiologically confirmed multiple sclerosis*; as consequence, this patient had a treatment by I.V. perfusion of methylprednisolone in a dose of 1000 mg for three consecutive days; the disease-modifying treatment by interferon-beta-1b at 250 µg has been maintained; finally, after less than one month this patient clinical state returned to the normal and she returned to work as usual;
- a 3rd attack appeared at four years and 2 months after the beginning of the problem and it has been treated in the same way with methylprednisolone I.V. for a short period of time; on the contrary, the disease-modifying drug has been changed to dimethyl fumarate;

– at nine years and four months from the clinical beginning of this patient's demyelinating disease of the central nervous system she is in a good health state, her EDSS score is 1.5 and she takes her dimethyl fumarate, she still works, and she has a normal everyday life.

In order to know more

The first demyelinating episode

The first demyelinating episode can be defined as he first clinical expression of a demyelinating disease of the central nervous system which medical history, clinical examination and paraclinical examination results do not allow establishing a diagnosis of multiple sclerosis according to the actual McDonald criteria for this disease.

The clinical presentation of the first demyelinating episode can be

– either monofocal (when all the clinical signs can be explained by the presence of a single demyelinating lesion),
– or multifocal (when the clinical signs cannot be explained by a single demyelinating lesion of the central nervous system, for example the presence of a hemiparesis and of a visual disorder).

The clinical evolution of a first demyelinating episode can be very different:

– some on these patients will return to normal clinical state and they will never more have neurological disorders of inflammatory nature;
– some of these patients will remain with a certain degree of neurological deficit(s) without any new attacks;
– approximately 50 % of these patients will develop multiple sclerosis.

Nowadays there are no exact criteria that allow predicting with exactitude who from the patients with a first demyelinating episode (FDE) will evolve to a multiple sclerosis. From what is known it can be said that the patients with FDE who are at high risk to develop a multiple sclerosis in the months and years to come are above all the patients who

– present a intrathecal synthesis of proteins and
– present at their brain MRI and spinal cord MRI T2-weighted lesions; in addition, more there are such lesions, higher risk is.

Because there is a significant risk for conversion of at least half of cases with a first demyelinating episode into a multiple sclerosis, the patients with a first demyelinating episode are proposed a disease-modifying therapy as if they were patients with multiple sclerosis.

The Lhermitte's sign

The Lhermitte's sign (also known as « the barber's chair sign ») presents itself as a sensation of electric discharge that is triggered by the flexion of the patient's neck and that starts from his neck and then immediately propagates alongside his upper limbs, his trunk, and – eventually – his two lower limbs.

It is an expression of the suffering of the axons of the posterior columns of the cervical segments of the spinal cord and it can be observed in case of:

– its inflammatory demyelination;
– its traumatic lesion(s),
– cervical spondylotic myelopathy,
– vitamin B12 deficit…

It has been named to keep the memory about the French neurologist Jean Lhermitte.

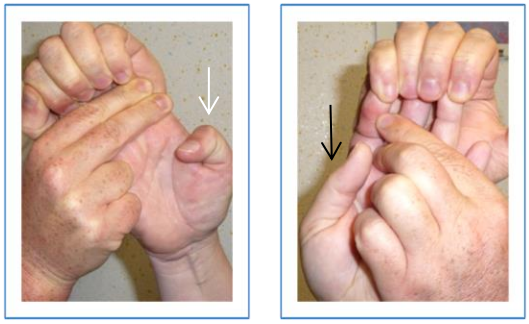
Jean Lhermitte

Jean Lhermitte (20.01.1877 – 24.01.1959) studied medicine in Paris where he also worked during most of his carrier. He was specialist both in neurology and in psychiatry.

He left his name in several signs and syndromes: most of all in the famous sign of Lhermitte, but also in the Lhermitte's peduncular hallucinosis and in the Lhermitte – Duclos' syndrome.

The Wartenberg's sign for pyramidal lesion

The Wartenberg's sign for pyramidal lesion is represented by the flexion of the interphalangeal articulation of the thumb and by the flexion and the adduction of the proximal phalanx of the thumb during the patient's voluntary effort to flex the distal phalanges of his fingers II, III, IV and V against the examinator's resistance (in a normal health the thumb does not move).



The Wartenberg's sign of pyramidal lesion in our patient. The test is done as described above. On the 1st picture (our patient's right hand) the flexion of the interphalangeal articulation and the flexion and the adduction of the proximal phalanx of the right thumb during the patient's voluntary effort to flex the distal phalanges of the rest of the fingers of his right hand are clearly seen. The patient's right thumb is indicated by a white arrow. At the level of this patient's left hand (2nd picture) the search of the Wartenberg's sign for pyramidal lesion is negative (the patient's left thumb is indicated by a black arrow).

The Wartenberg's sign of pyramidal lesion is the equivalent of the Babinski's sign at the level of the upper limbs.

Robert Wartenberg

Robert Wartenberg (19.06.1886 – 16.11.1956) was a German neurologist who has been born in Grodno in the Russian Empire and who later he got U.S. American citizenship.

He has described:

- the Wartenberg's disseminated neuropathy;
- the syndrome of cheiralgia paraesthetica of the radial nerve (the Wartenberg's syndrome);
- the Wartenberg's first sign that represents the position of abduction of the little finger in case of ulnar nerve paralysis;
- the Wartenberg's second sign (the equivalent of the Babinski's sign at the level of the upper limbs);
- the Wartenberg's phenomenon that accompanies the corneal reflex and that consists of the deviation of the mandible towards the opposite direction when the cornea is touched at the same time when the ipsilateral eye closes.

He has invented the Wartenberg pinwheel that serves for studying the sensory functions and most of all for searching the level of a sensory disturbance, as for example in case of a spinal syndrome.

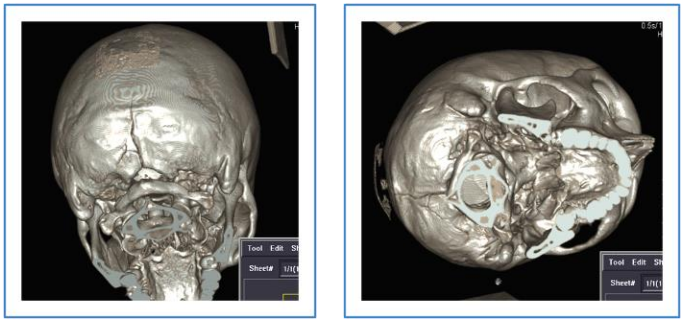
132. The head trauma (1st case)

This 30-years-old man who was in a perfect health up to now, has just been admitted in our hospital after a fall from a height of 3 meters. In fact, he has fallen on asphalt on his back and on the posterior part of his head.

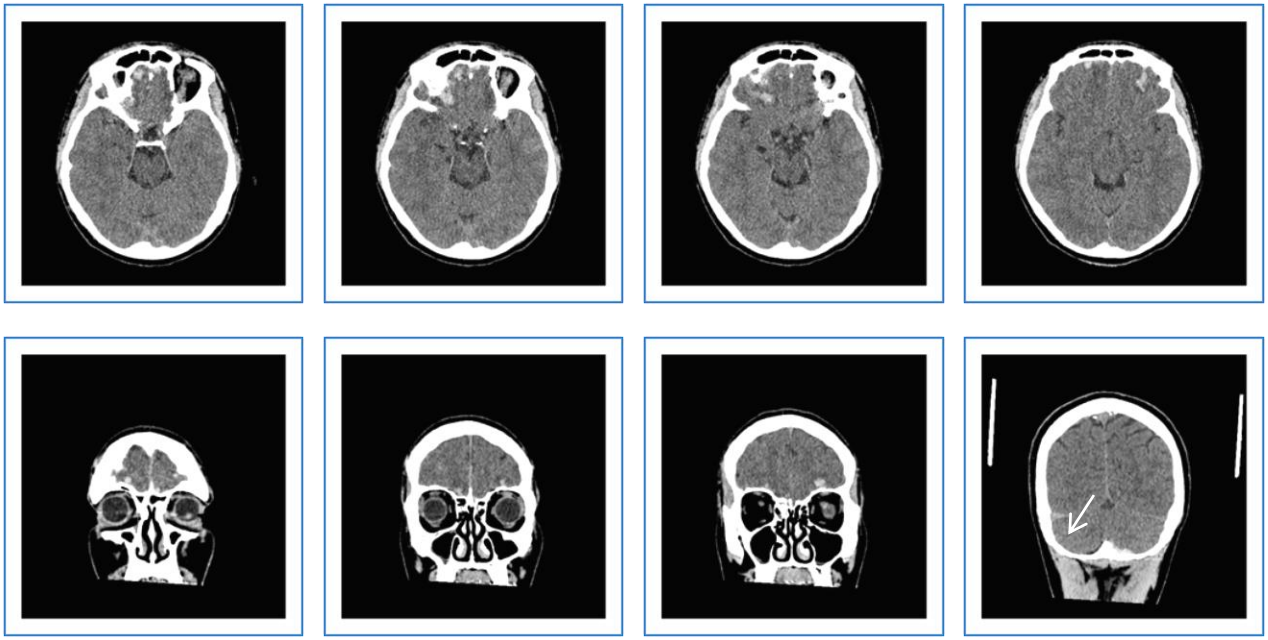
Now he complains both of headaches (as if his head was in a helmet) and of diffuse pains in his back.

His somatic examination reveals a superficial injury of his scalp in the occipital area that has just been sutured. His neurological examination is normal: he has neither motor deficits, nor sensory disturbances, nor memory disorders, his CGS is 15 points.

The logical next page is to do a head scan to search for possible consequences of this fall in the skull and / or cerebral parenchyma. Here is its result:



The study of the skull reveals the presence of a comminute fracture in the occipital region.



The study of the cerebral parenchyma shows the presence of bleedings according to the « coup – contrecoup » mechanism: a minimal subdural haematoma in the right occipital region (the arrow in the 8th image) and multiple haemorrhages in the cerebral parenchyma in both frontal lobes (the hyperdense zones that can be seen in both frontal regions in all other images).

So, this is a clinical case of an important head trauma with multiple contusions of the cerebral parenchyma. This patient's case has been discussed with our neurosurgeons and the therapeutic approach to it has been decided jointly:

The patient was admitted in the continuous medical surveillance unit in order to keep a close eye on the possible development of acute or subacute complications of this trauma.

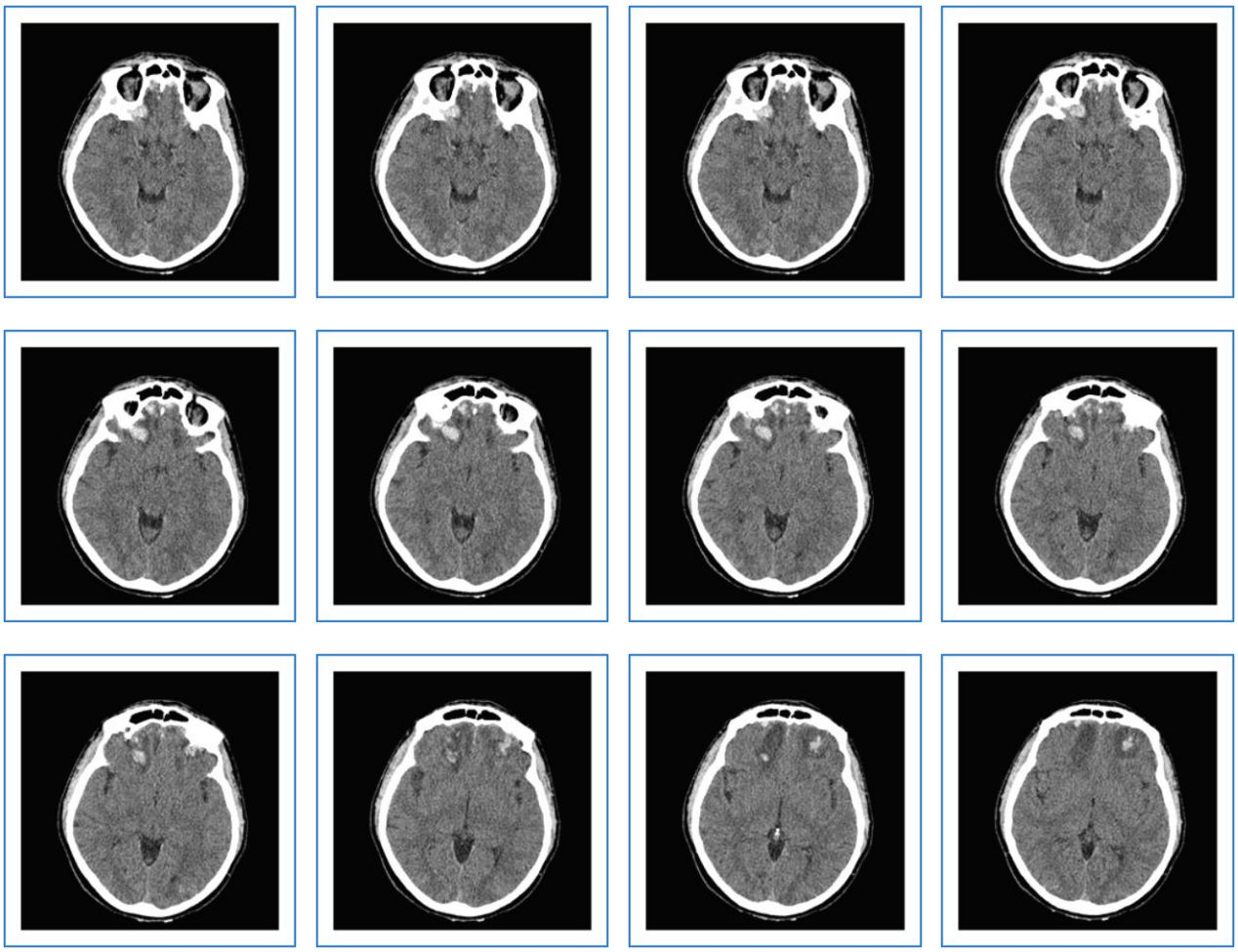
His treatment consisted of:

- analgesics, first I.V. and later per orally,
- an antibiotic (one gram of amoxicillin t.i.d. per orally) as primary meningitis prevention even though the patient had neither otorrhea, nor rhinorrhoea,
- a corticosteroid (for the reduction of the oedema around the zones of bleeding into the brain parenchyma) with a proton pump inhibitor per orally and
- rehabilitation for the primary prevention of the possible complications of the decubitus and for a progressive return to a normal mobilisation.

Because of pains and of a functional impotence in his left elbow this patient had also an X-ray of his left elbow followed by an orthopaedic consultation. This proceeding allowed excluding a pathology in this location and the patient quickly regained the normal mobility and use of his left elbow.

The first 5 days of his treatment have been marked above all by very important headaches with a quite slow diminution of their intensity. His vital parameters remained normal, and he had neither focal neurological deficits, nor behavioural disturbances.

At the 5th day of our patient's in-hospital stay a control head scan has been carried out and its result gave the explanation why he was complaining so much of headaches:



Presence of an important oedema around the zones of bleeding into the cerebral parenchyma with beginning of a resorption of the extravasated blood.

Considering this patient's stable clinical state at the evening of the 5th day of his in-hospital treatment he has been transferred into a standard neurological ward.

And, so is it the real life, in the morning of this patient's 6th day of in-hospital treatment we had an unpleasant surprise: coming to the workplace in the morning, the first thing that the on-duty nurse told us was that this patient has even more headaches and since the early morning he feels really sick.

This patient was now complaining of headaches of 10/10 according to the VASP and of a mild intolerance to the light. His neurological examination showed only one pathological particularity: the bilateral presence of the Kernig's sign.

These new data make us at first place think of an acute (bacterial) meningitis because of an osteo-meningeal breach. So, the immediate realisation of lumbar puncture is a must, there is no place for any discussion and for any postponing of this diagnostic examination.

This lumbar puncture was done in the next few minutes in an atraumatic way. Here is the result of the cerebrospinal fluid analysis:

Studied parameter	Test tube 1	Test tube 2	Test tube 3
Opening pressure of the CSF	повишено		
Macroscopic aspect	кървав	кървав	кървав
Leukocytes	3680 / mm³	4350 / mm³	4920 / mm³
Erythrocytes	60000 / mm³	40000 / mm³	30000 / mm³
Differential leukocyte count	polynuclear cells: 80 %, lymphocytes: 5 %, monocytes: 15 %		
Proteinorachia	0.90 g/l		
Glycorachia	2.00 mmol/l for a capillary glycaemia of 6.00 mmol/l		
Chlorurachia	117 mmol/l		
Bacterial flora	no visible germs		
Bacterial culture	negative in 48 hours		
Mycological culture	negative in 48 hours		

Already during the preparation of the lumbar puncture, having in mind what the supposed diagnosis was, we have prepared an I.V. antibiotic perfusion with four grams of cefotaxime (supposing that most probably this was a bacterial meningitis by pneumococcus). This perfusion has been immediately started in the moment when the needle for the lumbar puncture has been withdrawn.

The patient was once again transferred into the continuous medical surveillance unit and once we had the results of his lumbar puncture we could establish the diagnosis of a **decapitated meningitis** (bacterial meningitis, most probably by pneumococcus that has been « beheaded » by the per oral treatment by amoxicillin that did not suffice for the meningitis prevention but that has weakened its clinical presentation and that has contributed to the fact that there was no bacterial growth from the obtained cerebrospinal fluid).

The dose of the cefotaxime was of 200 mg per kilogram body weight in 24 hours divided into four equal doses done by I.V. perfusion every 6 hours. This dose is a *meningeal dose* (adapted for the treatment of the meningitides), and the duration of this meningeal treatment was 14 days.

In addition to the antibiotic, the patient also got a symptomatic treatment: analgesics, laxative, rehabilitation, so he managed to recover completely and finally he returned home.

In the meantime, an ENT consultation did not reveal any particularities in this patient.

One week later the patient had also a neurosurgical consultation with the prescription of a scan of his skull for the search of possible meningeal breaches. This search for meningeal breaches gave a negative result and the decision that has been taken was to refrain from a neurosurgical treatment.

This patient continued his usual everyday life, he got vaccinated against pneumococcus and against haemophilus influenzae b (even though it is well-known that these vaccinations do not provide an absolute protection against these bacteria in case of meningeal breaches).

In order to know more

Therapeutic attitude in case of acute meningitis

The (suspicion of) acute meningitis is one of the real neurological emergencies and that's why is such a case no time must be lost neither for the establishing the diagnosis, nor for the beginning of a curative treatment as the price here is no more and no less than the patient's life.

Except there is a well-founded suspicion of a syndrome of intracranial hypertension the LP is done immediately, without head / brain imaging studies: the goal is to gain time.

The other factors to consider are the patient's coagulation parameters (major thrombocytopaenia? VKA?) and the local state of the skin and of the subcutaneous tissues at the location where the LP will be done.

Once the lumbar puncture is done a probabilistic antibiotic therapy in meningeal doses must be started: the best attitude is that the patient has already an I.V. line and that the I.V. antibiotic perfusion is already ready to use.

The antibiotic therapy can be corrected / optimized in a second time; in any case this attitude saves lives (I have personally observed a clear-cut and immediate improvement of the general state and of the neurological state in a patient with acute purulent meningitis thanks to the application of such a therapeutic approach).

The decapitated meningitis

The term «decapitated meningitis» describes a bacterial meningitis that has (had) an antibiotic treatment insufficient to cure it but sufficient not to allow the identification of the pathologic agent as the CSF cultures do not give any bacteria growth. In such cases other methods for the identification of the responsible pathogen must be used, e.g., search for soluble bacterial antigens, PCR.

How to differentiate a traumatic CSF from a truly haemorrhagic CSF

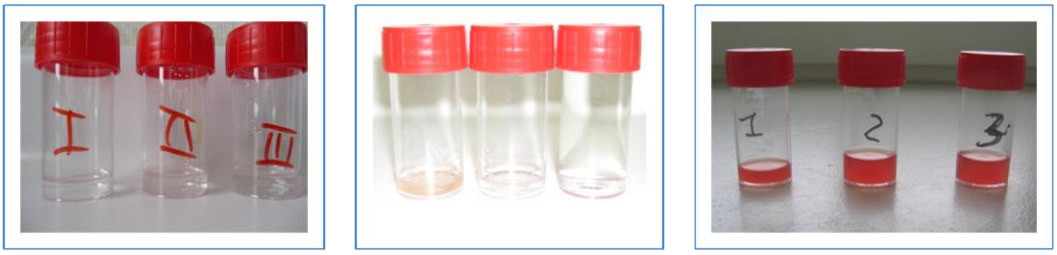
The normal cerebrospinal fluid has a completely limpid colour.

This normal limpidity can disappear in case of different pathologies (traumas, infections, tumours etc.) and the pathologic colour can orient towards the type of underlying pathology.

A special case of a potential diagnostic difficulty is represented by the traumatic lumbar puncture. Such a LP does not obligatory mean that it has been realized in an incorrect manner as there are many patients who are very difficult to puncture (arthrosis and / or scoliosis, past surgeries) or, sometimes, it happens to puncture one of the veins that are situated in the spinal canal.

In case the CSF has a red colouration, we must differentiate between a traumatic LP and a LP in case of real post traumatic or spontaneous bleeding inside the head and / or the spinal canal:

- if the LP is traumatic the intensity of the bleeding decreases with every new test tube and if there has been a real bleeding it remains the same in all test tubes;
- in case of a real bleeding the obtained SCF does not coagulate (the fibrinogen is consumed at the place of the bleeding), but in a case of traumatic LP the obtained CSF may coagulate if there is enough blood inside the obtained specimen.



On the left: normal CSF and a non-traumatic LP; in the middle: normal CSF and a traumatic LP; on the right: CSF in a case of spontaneous subarachnoid bleeding (example from our document 010).

136. A special type of acute diarrhoea to know well (1)

This 85-years-old woman (body weight 59 kg and height 166 cm) has been admitted to the hospital 17 days earlier because of an acute febrile confusion.

Thanks to the quick and complete initial diagnostic work-up she was diagnosed with acute meningitis by group C streptococci and an appropriate antibiotic treatment has been started a few hours after her hospital admission (continuous i.v. perfusion of cefotaxime in a dose of 300 mg/kg b.w. per 24 hours, i.e. 18 grams per 24 hours). So, this is an antibiotic treatment by a 3rd generation cephalosporin in a dose for meningitis with the amount of the total dose calculated according to the patient's bodyweight.

The duration of this antibiotherapy has been determined to be 14 days according to the official recommendations for this type of pathology and for this type of pathogenic microorganism. In our patient's case the antibiotic treatment has been done during 15 days.

So, on the 16th day after the beginning of the diagnostic and the therapeutic care for this patient we are at one day after the end of the antibiotic treatment. The patient is without fever since the third day of her hospital treatment and her general health is excellent.

But, during the clinical visit in the morning of the 17th day of her hospital stay the patient tells us that since the last evening she has abdominal pains and that she has had 5 stools (so in a lapse of time of 12 hours).

At that moment we have a feeling of "déjà vécu" and we suspect a specific pathology.

Which one?

In these circumstances, *the first diagnosis that we should think about is* ***the pseudomembranous colitis***, because the clinical settings are typical for the onset of this type of diarrhoea: beginning of diarrhoea in a patient who has been given high doses of antibiotic during several days.

So, we must act quickly. Four things are needed right now:

- a stool sampling for stool analysis;
- the beginning of an antibiotic treatment that is specific for this disease;
- the establishing of a preventive contact isolation for this patient and
- a consultation by a gastroenterologist.

All these measures have been implemented in our patient's case.

The stool analysis showed the presence of the toxin A of Clostridium difficile.

The chosen antibiotic treatment was metronidazole by the mouth at 500 mg every eight hours.

The preventive contact isolation has also been set up.

The gastroenterologist consulted this patient and did a sigmoidoscopy that showed the presence of pseudo membranes in the colon and in this way presented one more argument in favour of the clinical diagnosis of a pseudomembranous colitis.

The metronidazole has been an efficient treatment and at the 3rd day after its introduction the abdominal pains have disappeared and since the 4th day the stool was neither too soft nor too frequent. It has been continued for a total of 10 days and thereafter this patient could return at her home in a normal health.

There is an important particularity in this patient's diagnostic and therapeutic work-up that should be noted: the initial biology showed a hyperglycaemia of 1.55 g/l (8.3 mmol/l) and the follow-up confirmed the elevated glycaemic values between 1.50 and 1.80 g/l, the glycosylated haemoglobin was at 7.6 %. So, a diabetes mellitus type 2 has been discovered and, after a specialized consultation, a specific treatment by appropriate diet and Insulin S.C has been started.

In order to know more

The pseudomembranous colitis

The pseudomembranous colitis is known since 1893 when the typical clinical picture of this disease has been described.

The causative agent of the pseudomembranous colitis is Clostridium difficile. This is a Gram positive anaerobic motile bacterium that is discussed more precisely in the section "In order to know more" of our second case of pseudomembranous colitis.

The typical clinical picture of the pseudomembranous colitis consists of:

- abdominal pains that could be very violent;
- appearance of a diarrhoea (the diarrhoea is being defined as the emission during 24 hours of at least three liquid or soft stools which total weight is more than 300 grams);
- appearance of fever.

The biological analysis shows an inflammatory syndrome and, eventually, dehydration.

The stool analysis shows the presence of leukocytes (in approximately half of the cases) and of the toxins A and / or B of Clostridium difficile.

In its typical clinical form the pseudomembranous colitis appears **during peroral or intravenous antibiotic treatment or up to two weeks after the end of this antibiotic treatment**. The risk to develop a pseudomembranous colitis is bigger if this antibiotic treatment is by quinolones, cephalosporin and / or clindamycin, i.e. with antibiotics to which Clostridium difficile possesses a natural resistance.

The other predisposing factors for the appearance of this type of acute diarrhoea are the age > 65 years, the hospitalisation, the medications that inhibit the production of the gastric acid and the eventual factors that modify the intestinal motility and the intestinal flora.

The therapeutic care consists of bedrest, sufficient hydration (either peroral, or i.v., or by both ways) and an appropriate antibiotherapy.

The antibiotics that are efficient for the treatment of the pseudomembranous colitis are:

- the metronidazole either by the mouth or i.v.; its usual doses, no matter in which way it is given, are of 500 mg every 8 hours for 10 days;
- the drinkable vancomycin that is kept as treatment for the pseudomembranous colites of moderate and severe degree; the other options for its application are the recurrences of this disease and its application in the pregnant women (the metronidazole could cause malformations); the standard dose is 500 mg every 6 hours for 10 days;
- the fidaxomicin: fewer side effects, to be used if the other two antibiotics are inefficient.

It is contraindicated to prescribe anti diarrheal drugs in case of pseudomembranous colitis because the slowdown of the intestinal transit increases the concentration of the toxins of Clostridium difficile in the intestines and, in this way, it exposes the patient to a high risk of eventual complications.

The cholestyramine is a bile acid sequestrant that binds the Clostridium difficile toxins and it can be an efficient addition to the antibiotic treatment.

The I.V. perfusions if polyvalent immunoglobulins could be helpful in cases of patients with pseudomembranous colitis who have an immune system deficiency.

The feces transplantation is an innovative method in the cases in which the antibiotics are inefficient and in the cases with multiple recurrences of this disease.

The possible complications in case of pseudomembranous colitis are the toxic megacolon, the perforation of the colon, an eventual peritonitis, the sepsis.

The recurrences of this disease are not rare – about 20 % after a first antibiotic treatment.

Book 2

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Table of contents – Summary

103	**A few tingling of the forehead and on the cheek**	*A clinical case of osteoma of the skull: medical history, clinical presentation, results of the head scan and of the head MRI, results of the bone scintigraphy and of the bone biopsy.* *The osteoma; the Gardner's syndrome.*
104	**The multiple sclerosis: the beginning of a typical clinical case (1)**	*A clinical case of multiple sclerosis with typical beginning: medical history, clinical presentation, standard and specialized blood analyses, brain MRI and spinal cord MRI, result of the lumbar puncture, treatment, evolution over the next 7 years.* *Blood and cerebrospinal liquid analyses in case of demyelination disease of the central nervous system; the McDonald's criteria; the Barkhof and Tintoré's criteria using the central nervous system MRI results in order to determine the dissemination into the time of the demyelinating lesions; the multiple sclerosis: aetiology, genetics, environmental factors.*
105	**A multiple sclerosis with an aggressive beginning**	*Clinical case of multiple sclerosis with aggressive beginning: medical history, clinical presentation, standard and specialized blood analyses, lumbar puncture, ENMG, brain MRI and MRI of the spinal cord, treatment, clinical follow-up.* *The EDSS, the multiple sclerosis with an aggressive beginning.*
106	**The same problems recur quite often in real life… A not so easy case of spontaneous subarachnoid bleeding**	*A clinical case of spontaneous subarachnoid bleeding: medical history, clinical examination, main rule in case of patients with headaches of sudden onset; aetiologic diagnosis, treatment.* *Possible complications in case of spontaneous subarachnoid bleeding; spasms of the cerebral arteries in case of spontaneous subarachnoid bleeding; the scale of the World Federation of the Neurological Surgeons (WFNS); the Fisher's modified scale and the risk of ischemic stroke.*
107	**The anisoreflexy: a clinical sign that is always pathological**	*A clinical case of anisoreflexy as indication for the presence of an organic problem into the central nervous system: medical history, clinical examination, brain scan, scan of the cervical segments of the spinal column, ultrasound examination of the cerebral arteries, cerebral MRI, aetiologic diagnosis, treatment.* *The deep tendon reflexes and their anisoreflexy.*
108	**In case of head trauma: always suspect lesions of the cervical segments of the spinal column**	*Two clinical cases of head traumas with concomitant lesions of the cervical segments of the spinal column, aetiological diagnosis, treatment.* *Principle of application of the imaging studies in case of head and brain traumas.*
109	**The symptomatic peripheral facial palsy (2)**	*Three clinical cases of symptomatic peripheral facial palsy: 1. Tumour of the cerebellopontine angle, 2. Complex malformation of the development of the branchial arch, 3. Barotrauma of the facial nerve.* *Tumours of the cerebellopontine angle, the barotrauma, the Boyle – Mariotte's law.*
110	**A patient who paralyses in front of us**	*A clinical case of acute axonal motor form of the Guillain – Barré's syndrome: medical history, clinical presentation, result from the lumbar puncture and the ENMG, standard and specialized (antibodies against Campylobacter jejuni) blood analysis, curative treatment, rehabilitation, clinical follow-up and ENMG follow-up.* *Acute motor axonal neuropathy.*
111	**The ventriculoperitoneal shunt: sometimes there are complications**	*A clinical case of obstructive hydrocephaly after haemorrhage into the brain parenchyma after spontaneous rupture of a cerebral artery: medical history, clinical presentation, result of the brain scan, result of the endoluminal treatment of this aneurysm, implantation of a ventriculoperitoneal shunt, complications on the part of this shunt.* *The ventriculoperitoneal shunt: different types, indications, possible complications; the ventriculoatrial shunt.*
112	**The symptomatic trigeminal neuralgia**	*A clinical case of a symptomatic trigeminal neuralgia: medical history, clinical examination, brain scan, blood analyses, malignant non-Hodgkin lymphoma, specific curative treatment and non-specific symptomatic treatment, final result of the medical cares.* *The malignant non-Hodgkin lymphomata: types, risk factors, clinical picture, type A and type B symptoms, aetiological diagnosis, stages, treatment.*
113	**The vitamin K antagonist overdose (2)**	*A clinical case of vitamin K antagonist overdose: medical history, clinical presentation, blood analyses with determination of the serum vitamin K concentration, treatment.* *Medical problems in patients treated by vitamin K: asymptomatic overdose, haemorrhages, traumas – head traumas and other types of traumas.*

134	**The carotid body tumour**	*A clinical case of accidental discovery of a carotid body tumour: medical history, clinical presentation, ultrasound examination of the extracranial segments of the cerebral arteries, therapeutic approach.* *The carotid body and its tumours: medical history, clinical picture, paraclinical examinations, differential diagnosis, different therapeutic approaches.*
135	**The febrile neutropenia: is this not a problem for the neurologist?**	*A clinical case of febrile neutropaenia: medical history, clinical presentation, blood analyses, symptomatic treatment and aetiological treatment, final result.* *The neutropaenia: definition, degrees, causes; the MASCC scale; the febrile neutropaenia: definition, aetiology, therapeutic approach, algorythm for stopping the antibiotic treatment in case of febrile neutropaenia.*
136	**A special type of acute diarrhoea to know well (1)**	*A clinical case of pseudomembraneous colitis as consequence of an antibiotic treatment: medical history, clinical presentation, treatment, final result.* *The pseudomembraneous colitis: definition, risk factors, typical clinical presentation, possible complications.*
137	**Recurrent acute pyelonephritis**	*A clinical case of recurrent acute pyelonephritis: medical history, clinical presentation, biological examinations, scan of the abdomen and of the pelvis, antibiotic treatment and symptomatic treatment, endoscopic treatment, final result of the therapeutic care.* *Acute pyelonephritis and chronic pyelonephritis: definition, aetiology, clinical presentation of the acute pyelonephritis, symptomatic treatment and aetiologic treatment, possible complications, risk factors; prevention of the pyelonephritides, the ureteral double J stent.*
138	**The melaena: it should be known well**	*A clinical case of melaena: medical history, clinical presentation, drug treatment and invasive treatment, final result of the treatment.* *The melaena: definition, clinical presentation, significance of the melaena for the neurologist.*
139	**The Murphy's sign**	*A clinical case of recurrent acute cholecystitis: medical history, clinical presentation with the Murphy's clinical sign, abdominal scan, treatment, final result of the therapy.* *The Murphy's clinical sign; the Murphy's ultrasound sign; the predisposing factors to the cholecystitides.*
140	**The Rovsing's sign**	*A clinical case of acute appendicitis: medical history, clinical presentation, result of the ultrasound examination of the abdomen and of the pelvis, threatening perforation, in case of acute appendicitis, treatment and final result of the therapeutic care.* *The Rovsing's sign, Niels Thorkild Rovsing; the McBurney's point; the Lapinski and Jaworski's sign (the Obraztsova's sign); the acute appendicitis: historical overview, Claudius Amyand, classical clinical presentation, diagnosis, ultrasound examination, scan of the abdomen and of the pelvis and blood analyses in case of suspicion of acute appendicitis, prognosis and treatment of the acute appendicitis; the acute appendicitis and the neurology.*