

Multiple sclerosis



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Introduction

Multiple Sclerosis (MS) is an inflammatory disease of the central nervous system, a leuco encephalomyelitis. Inflammatory disease of the central nervous system meets acute infectious diseases; outside the abscess, it is mainly encephalitis and myelitis virus (herpes, measles, CMV.) related to the presence of the virus in nerve cells; reaching in good standing (general?) gray matter, these are polio-encephalitis and polio-myelitis (see acute anterior poliomyelitis). - post-infectious encephalomyelitis, that follow a viral infection (vaccinia, measles, mumps, chicken pox.) after the viremia phase contemporary with the immune response and in connection with it; they reach the white matter; They are leuco encephalitis peri-venous (edema, inflammation, demyelination in perivascular cuffing) moving monophasic manner. This simple opposition is insufficient - there very late viral encephalitis, associated with the persistence of a virus, persistent latent for years, in neurons and glia, before declaring as subacute sclerosing pan-encephalitis (measles virus, rubella); -Some viruses papova up (owed to the year immune deficiency) glial cells in tea white matter, give has progressive multifocal leukoencephalopathy, subacute. Experimentally, it can produce leuco encephalo-myelitis injection in certain strains of mice with a suitable adjuvant, of an extract of myelin (or basic myelin protein, or of certain peptides of this protein); is the model of experimental allergic encephalitis said (EAE). Can also be produced by intracerebral injection of some viruses (Theiler, mouse hepatitis) disease in two, polio-encephalitis by viral infection and buffy encephalitis autoimmunity [1].

As part of the inflammatory pathology of MS has a special place because of its frequency (the main non-tumor disease of the nervous system in young adults), its prolonged course of decades, causing over time deficits increasingly disabling. Experimental Allergic said Encephalitis (EAE). Can also be produced by intracerebral injection of some viruses (Theiler, mouse hepatitis) disease in two, polio-encephalitis by viral infection and buffy encephalitis autoimmunity. As part of the inflammatory pathology of MS has a special place because of its frequency (the main non-tumor disease of the nervous system in young adults), its prolonged course of decades, causing over time deficits increasingly disabling. Experimental Allergic said Encephalitis (EAE). Can also be produced by intracerebral

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Definition of the multiple sclerosis

The nosological definition of the MS remains anatomical; The description of the hurts was worth him its name (Charcot on 1868). It is a demyelination, a destruction of the myelin in the white material of the encephalon and the moëlle, respecting axons (myéline-axonal dissociation, at least relative). It is a demyelination inflammatory as show it the young hurts where the oedema and the inflammatory infiltrate accompany the active destruction of the girdles of myelin. In the less young hurts, it is in the periphery of the hurts, in their zone of progress that we find the inflammation: T-cells (CD4), then macrophages and lymphocytes B; Secretion of immunoglobulins (IGM then IGG). The former hurts are the seat of a proliferation astrocytaire which characterizes the hardening of the nerve tissue; What Charcot described, it is the scar stage of the hurts. These demyelinating hurts have a singular distribution and a topography, not spread, neither in tablecloths, nor in muffins, but in plates. The zones of demyelination are confined, in the cup rounded off or ovalaires, in the space egg-shaped, well confined, with a clear border with regard to the healthy myelin. These plates are multiple and spread in the central nervous system (encephalon, optical nerve, moëlle épineière), being able to affect any sector of the white material, but with sites of preference: brainstem, periventricular zones.

Epidemiology

She shows an uneven geographical distribution of the disease with zones of high prevalence (around 100 for 100.000 inhabitants) in Scandinavia, the Scotland, the Northern Europe, in Canada and in the North of the United States, the zones of average prevalence (around 50), Central and western Europe, the South of the United States, the zones of low prevalence (lower than 20), around the Mediterranean Sea and in Mexico.

The disease is exceptional in Africa in the population black. Thus, a first look reveals a North-south gradient, wet moderate climates in the hot countries, but this principle of latitude is not general application: Japan, the North-south extension of which amounts to that of the U.S.A. is uniformly in low prevalence; The distribution of the SEP in the southern hemisphere does not present a gradient so simplistic. On the other hand, in the zones of strong and average prevalence, we observe an uneven distribution, with homes, in a city or a rural area (it is the case in Brittany) [5,6]. The analysis of these data leads to suppose the interference of two factors at least: a factor of environment and a genetic factor. The hypothesis of a factor of environment is pressed in particular on the study of the migrations of populations between zones of uneven prevalence: very in broad outline, those who migrate after the age of 15 years have the risk of the region of origin, those who migrate before the age of 15 years have the risk of the region of arrival, as if a decisive event occurred, only, in certain parts of the country, in the adolescence (several years before the clinical beginning of the disease). In favour of a genetic factor, besides the low prevalence of the SEP at the Japanese, we raise the rarity to Black Americans in the North as in the South of the USA. The intervention of the genetic heritage is confirmed by the enumeration of families multi-case more frequent than would not give the fate we estimate the risk at 2 p. 100 for the brothers and sisters of a patient, that is a factor 50 with regard to the unrelated subjects. The Canadian study of the twins among which the one is expanding the disease shows for dizygotes a concordance of 2 p. 100 (as all the Germans), and for the monozygotes of 40 p. 100. This reveals another time the importance of the genetic factor in the determinism of the disease and the fact that it is not a hereditarily passed on disease (100 p. 100 of the concordance to monozygotes). There is a susceptibility of genetic origin, but which is not enough so that occurs the disease [7-10].

Genetics

The notion of genes of susceptibility rests on the connection between SEP and HLA system today. Numerous and confirmed studies showed, at the patients reached of SEP, an overrepresentation (an excessive presence with regard to the comparable witnesses) alleles A3 and B7, and especially DRW15, DQ6 (corresponding to the group DW2 in lymphocytic culture mixed). These results, found in the European countries and in North America, appear to correspond mainly to the genetic contribution of the populations of Scandinavian origin. The connection to HLA is found everywhere, but the overrepresented alleles are not everywhere the same (examples of Italy of the North, the parsis in India). If the presence of certain alleles on the 2nd locus of histocompatibility is responsible of, or associated with a bigger susceptibility in the SEP, this genetic factor is not certainly the only one (nor even possibly the most important). Other genetic studied polymorphisms (immunoglobulins, receiver of the cell T, proteins of the myelin) did not at the moment succeed to untangle the certainly multi-genetic transmission of the susceptibility.

Etiology

The distribution by sexes indicates a feminine ascendancy: more or less 3 women for 2 men. The study of ages shows that the clinical beginning is situated between 20 and 40 years in 70 p. 100 of the cases. There are early debuts: - 2 p. 100 before 10 years - 13 p. 100 between 10 and 20 years and SEP of late occurrence: - 11 p. 100 between 40 and 50 years - 3 p. 100 between 50 and 60 years - And even 0,3 p. 100 after 60 years. The argument of a usual age of appearance must in practice be put in perspective.

Private hospital - principles of the diagnosis

The diagnosis of SEP rests on the highlighting by the discussion of the clinical and electro-physiological syndromes observed of more than a lesional site, an obligation to hold several locations and on the report of an evolution which goes on or revives beyond a few months. The most characteristic is the remittent evolution by successive pushes (65 p. 100 of the cases), who decline in a few weeks, with or without aftereffect, to give way to a calm or a stability until the following push. Sometimes after a phase of remittent evolution of a few years begin a worsening of progressive continuous type (remittent-progressive forms). The remittent evolution is not a necessary argument: we estimate to 13 p. 100 the progressive continuous S.E.P. straightaway, form from what the profile is very different and the severe forecast.

Modes of the beginning

The hurts which express themselves in the first one can sit in very diverse points of the central nervous system, pulling a revealing, noisy or discreet, very variable symptomatology of a case in one other. The first demonstrations can be (to quote only the most frequent): - a paraparesis, - a brachial monoplegia or crurale, - an imbalance in position standing (cerebellar? Proprioceptive? Vestibular?)- sensory disorders, paresthesias, dysesthésies, bum around of cutaneous hypoesthesia, - a retrobulbar optical neuritis, - more rarely the paralysis of a cranial nerve, a facial paralysis, Claude Bernard-Horner's syndrome. The diagnosis rests at this stage, in front of an apparently isolated functional demonstration, on the complete neurological examination looking for the objective signs of a lesion of other location, by using some study of the PEV, PE, PEA, possibly by waking by the test of the hot bath of the subdued symptoms. The MRI, when it shows, with a clinical syndrome uniloculaire, multiple hurts of the white material, also contributes to return the likely diagnosis. Only the evolution, indicating that it is not a monophasic acute process allows an asserted diagnosis.

Usual established Paintings

At the end of a variable time, typically after a succession of regressive pushes, the SEP pulls a permanent overdrawn picture. Because of the preference of the hurts for certain beams, a clinical picture can be described which, without to be unambiguous or compulsory, finds itself in a majority of cases. Spasticity (in position up); Deficit (in position up); Moderate deficit of

raccourcisseurs; Exaggeration of the BELCH, polycinétiques, spread (this exaggeration finds itself in upper limbs and comes along précocément with an abolition of the reflex of the veil); Sign of Babinski. With a discreet paraparesis in the neurological evaluation, we can observe a loss of command of lower limbs in the walking, true abasie.

A cerebellar note

Which deteriorates the disorders of statics and approach? She is obvious for upper limbs, boorishly incoordonnés, until make the writing impossible.

A proprioceptive note

Ataxia; Sign of Romberg; Certain patients have to check by the view the position of their feet.

A nystagmus

The most frequent are the rotatory side nystagmus in both side positions of the look and the multi-directional nystagmus. To note particularly the monocular abductor nystagmus of eye, testifying of an inter-nuclear ophtalmoplégie. Other rarer nystagmuses are possible.

A dysarthria

Where join to diverse degrees a cerebellar component and a paralytic component.

Anomalies of the fundus oculi

Paleness of the temporal segment of the papilla, united or bilateral, scar of an optical neuritis the interrogation of which sometimes finds the acute phase, as the history of an episode of spontaneously regressive unilateral amaurosis. In the phase séquellaire, an optical neuritis can pull only a minimum of functional deficit, but in contrast, at some patients, the amaurosis is one of main factors of incapacity.

Other

To this set syndromique, himself very multi-form according to the relative severity of the various constituents, other symptoms can be added which modify the presentation.

Pains

The SEP is an invalidating chronic disease but typically it does not ache. The exceptions take the form of shooting pains of lower limbs, to which we can move closer to the sign of Lhermitte caused by the fast passive flexion of the neck (sign of infringement cordonale later) of diffuse shooting pains in the low part of the body - of facial pains, either to type of Neuralgia of the triplet (in discharge), or of continuous type (with or without hypoesthesia of the same territory) - of joint and muscular pains bound to the spasticity.

Disorders of the functioning's sphincter and sexual

Usual at a late stage, after numerous years of evolution, they appear at some patient's précocément and dominate then the picture. They deserve a particular attention, a functional balance sheet, mesures rééducatives, pharmacological treatments.

Psychological troubles

On thymic and emotionally, there is described a condition of indifference, serenity discordant with respect to the real life of discomfort (Spes sclerotica); This state exists but it is rare and more often a depression more or less rebellious observed, exacerbating the social and occupational disability. Cognitively, moderate disability is common but not constant: inattention, memory, synthesis, flexibility. The forms dementia are exceptional, such as changes marked by confuso-mania with recurrent outbreaks.

Seizures

Few in all, they are often generalized as partial; Well controlled by tea treatment they can go out after-party has few months gold years and allow stopping anticonvulsants.

Dystonic phenomena

In a disease that does not include extrapyramidal symptoms, they can be summarized in a contracture (with tetaniform paroxysms) of a limb or side of the body.

Device signs

MS is a central demyelination; This formula explains lesional any symptoms. However, late in evolution can be observed areflexia in certain territories and localized neurogenic atrophy (lesion of motor root fibers in their intramedullary trip? Stake peripheral myelin destruction in the process?).

Vegetative signs

Very rare are the disorders of T.A., of the pulse, the vasomotricity. The hyperthermia accompanies episodes of coma which, after regression, can repeat several times.

Clinical Forms

The polymorphism that associations between the previously described syndromes can make allows to pass quickly on the chapter of the symptomatic forms by mentioning only some atypical or little usual aspects: - hemiplegic or sudden, pseudo-vascular forms, very rarely yoker, is progressive feigning one T.C ., - Medullary forms, under the form of a syndrome of Brown-Sequard, either a cross-functional myelitis, or a hardening combined of the moëlle, the paraplegia in flexion or the syndrome of the terminal cone. In the I.R.M. certain cases of purely medullary hurts cannot contain abnormal images at the level of the encephalon, - cerebellar forms with lack of coordination of the trunk, the head and the members; They are quickly invalidating forms to young subjects. Evolutionary forms: the profile of the evolutionary curve distinguishes, we said it, remittent, remittent-progressive and progressive forms straightaway. The rhythm of the pushes and/or the progress of the deficits individualizes: - grave acute forms which install very fast severe handicaps, completed and reshaped in a few months of continuous pushes.

After one or two years they can finally stabilize by leaving an overdrawn state major. -Average forms, which, in 10 years,

reach the level 4 on a scale in 10 points (keep an activity in spite of severe difficulties), in 20 - 25 years, the level 6 (= limited and difficult travels). -Mild forms, in the form of pushes spaced out, made by sensory declining disorders every time without aftereffect, either beginning in a anyway but stopping showing itself later 2 in 3 pushes. -There are even dumb, completely latent, bare forms of autopsy (to a subject died from other affection). It is necessary to hold the extreme diversity of these evolutions, very different from the unambiguous image of fast incapacity usually attached to the SEP (and which corresponds to the grave forms: approximately 10 out of 100 of the cases).

The mild forms must be known because, for them, no therapeutic risk is justified. Some indications allow to plan in which evolutionary form we are confronted: - the interval between the first one and the second push - the number of pushes in the first 2 years - Date of entrance to a continuous progress - the level achieves on the scale of incapacity in 5 years, in 10 years. But it is never a question that of probability, the forecast which can be denied, for the better when a very long complete stabilization intervenes (even during a continuous progress), for the worse when after years of stability without no demonstration of the disease arises a grave push which increases seriously the deficit séquellaire. There are faded forms of SEP. If he is usual to see the interval lengthening between the pushes with years, to observe after 10, 15- or 20-years long periods of stability, it happens to meet patients of 70 and more years old, to whom the disease shows no activity for 20 years and which the neurological status will not change anymore.

Positive Diagnosis

He rests on the private hospital, the electrophysiology, the evolution. The study of the LCR can complete the criteria of the diagnosis, what is important when it is lacking or the proof of a distribution multi - loculaire or the evolution polyphasique in relapses. She shows: - the rise of the protéinorachie, always moderated (0,60), fickle, - the abnormal presence in the electrophoresis of IgG, produced in the nerve tissue, by secretion intra - thécale. The character of (inflammatory) exudate and no of transudate (permeability in blood proteins) is demonstrated by the comparison of the (normal) rate of the albumin with regard to the IgG; Comparison made according to diverse formulae of which the Index IgG of Link. -The distribution oligoclonale IgG. This anomaly is the most constant (> 90 p. 100 of the cases), the most characteristic (she observes only in acute encephalitis or certain chronic infections), but she is not revealed by most of the electrophoresis of routine. -A hypercytose, almost always moderated (< 20 cells). She is very often lacking, in particular in the first years. Dependent on the seat of the active hurts, she has no meaning forecast.

E.I.R.M.

It is the only method which shows the hurts in the central nervous system. She confirms from the beginning some SEP what Ana Path. showed late: the large number of hurts (disproportionate with what indicates the private hospital) and

their seats of preference. The presence in the white material of zones of hypersignal in T2 does not distinguish: the oedema, the demyelination, calcifies her (former plates) and, in this one the possible axonal necrosis. The use of the gadolinium, marking the zones of abnormal vascular permeability, shows the hurts - some plates, or the periphery of some plates, in ring - at present in service. For the diagnosis, the MRI is outstandingly sensitive, but specific no. The multiple and confined images of hypersignal are frequent after about fifty, especially at the persons suffering from high blood pressure. Such images observe in the disease of Behcet and in the vasculites of SNC. Some young subjects present a small number of hypersignals confined except any visible (?) pathology. Nevertheless, the MRI brings, in front of a neurological episode of location one-of-a-kind to a young subject, the argument of spread multiple hurts. THE repeated MRI taught us on the natural history of the hurts a lot.

In SEP of average gravity, remittent or remittent-progressive evolution (both types are here inseparable), there is 8 times more locatable events by the MRI than by the private hospital (appearance of new plates, increase or decrease of certain zones of hypersignal, taken by gadolinium). THE MRI confirms the isolation of the mild forms (small-sized, relatively few images) and the individuality of the progressive continuous forms (small number of images of hypersignal, or not evolutionary, some voluminous images).

Differential diagnosis

Easy as far as there is a usual picture, making associations syndromiques rather particular, the diagnosis can also be of the most difficult because numerous cases move away from the type by their picture and by their evolution. The errors are frequent, by excess and by default. They concern: - tumors and angiomas of the brainstem - the deformations of the crânio-encephalic hinge and the tumors of the foramen magnum - the disease of Friedreich - the neuro-inanimate syndromes - the subacute myelopathies and particularly the artério-venous shunts (with recurrent evolution) - The acute encephalomyelites spread (the evolution of which however is monophasic). In the group of the diseases which cause hurts multi - loculaires of SNC, we still find: - the disease of Whipple, - angéites, disease of Behcet and uvéonévrites, the disease of Gougerot-Sjögren. -The sarcoidosis - the cancerous metastases - the progressive multifocal leuco-encephalopathy. Contrary to the SEP, the previous affections come along, to varying degrees, with a general inflammatory syndrome.

In front of the fluctuating, fuzzy, temporary character, of certain demonstrations of the beginning (weakness, imbalance), the diagnosis of hysteria is sometimes held; Mutually demonstrations of hysteric nature can be taken for evolutionary pushes to a patient carrier of a SEP. Nosological limits The definition of the SEP being pathogenic but anatomical and restful not on the notion of multiple confined inflammatory demyelination, there are entities which can be considered either as anatomical forms of the disease, particular by the seat, the

volume of the hurts, their necrotizing trend, or as different diseases, such as the optical Neuro-myelitis of Devie, or the inflammatory form of the disease of Schilder. What we indicate as progressive forms today straightaway the SEP will appear maybe one day as other disease.

Physiopathology

It is false to say that we ignore everything of the cause of the SEP. There is not most probably a cause one-of-a-kind; But we rather have to admit a multi-factorial disease certain factors of which begin to be untangled, others no.

The auto-immunity

We have an experimental model of the SEP: the experimental allergic encephalitis in its chronic forms with relapses (in man, the neurological complications of the antirabid vaccination of type pastorien make a true experimental disease very close to the EAE). This disease caused by an injection one-of-a-kind of extract of myelin (or other better defined antigens: the basic protein and its peptides encéphalitogènes; The protéolipid) with the additive of complete Freund, shares with the SEP the fact: - to be an inflammatory demyelination - To evolve in a remittent and prolonged way - to depend on genetic limitations, being possible only in certain origins of mouse - to be bound to the major complex of histocompatibility. She differs from it however because the hurts are essentially perivenous, no in plates, and especially that no antigen was able in the SEP to make the proof absolved from its role immunogenic (responsible for an auto-immunity).

The history of the development of the hurts is very stackable to the detail between the animal model and the SEP. The first phenomenon is the infringement of the endothelium of the vessels of SNC, with break of barrier, passage of globulin, lymphocytes and secretion of lymphokines. Contemporary of this beginning of the disease or the departure of a push, we note in the circulating blood a fall of cells CD8 (which among others have a function suppressive). These first events are going to lead locally the activation of the macrophages which separate and destroy the girdles of myelin, as well as the attack of oligodendrocytes (in which intervenes the complement). The push is a local phenomenon, where every lesional site evolves or remains faded in a independent way. The interferon gamma is one of main activators of the development of the hurts. If we cannot, in the absence of responsible antigens, to assert an autoimmune disease, at least as it was created for the SEP the concept of disease with immunological mediation.

The genetics

The susceptibility in the SEP appears, we saw him, determined by the genetic heritage of each. This datum is completely compatible with the intervention of immunological mechanisms, because the main immune responses are commanded and modulated by various genes. In this context, the presence of certain alleles HLA is not maybe a simple marker of susceptibility; The molecule can intervene by itself in its

presenter's role of antigen to the receiver of the cell T. Anyway, other genes still intervene in the susceptibility; They are the object of an active search.

The factors of environment

So that a susceptibility becomes a disease it is necessary the meeting of determining factors or at least déclanchants. For a very long time the possible role of virus was suspected and every progress in the knowledge of the biology of the viruses and the classes of viruses was immediate tested with regard to the SEP. The description, disputed, of epidemics of SEP would be an indirect argument for the intervention of a virus. Occasionally an isolation (myxovirus, coronavirus, retrovirus) from a case refreshes this old discussion. We do not know a virus of the SEP today. We have no arguments to support the hypothesis of capable multiple viruses of déclancher in a non-specific way pathogenic immune response.

Treatment

We are in a pivotal period between a stubborn skepticism and therapeutic tries which multiply. The tries raise difficult problems which were analyzed well to be surmounted: - the sought objective, defining the measured parameter (the duration of the pushes and the symptoms estimated at 1,2,3,4 weeks; The number of annual pushes; The increase of the level of incapacity after 2 years of treatment). The natural history of the disease imposes rigorous conditions to hope to obtain significant results, - staff and durations of experiment, - the choice enters a try on determined types of SEP and an evaluation on cases all - venants, knowing that we think twice to propose the inclusion, with a luck on two to be under placebo, to patients who quickly deteriorate their incapacity, - and knowing that for the mild forms there will be no difference between the handled patients and the witnesses, - the ethical problems such as the preservation during several years, under placebo of a serious illness (the comparative test of two treatments risking more not to be decisive and rarely lending itself to a double insu). The duration of a try and the staff of the inclusive patients can be largely reduced by measuring on repeated MRIs the number of the events which occur. We estimate an effective action on the disease but not the utility for the sick. Considering these difficulties and uncertainties which they pull, some conclusions are possible. The therapeutic conducts which ensue from it are to be seen again according to the results of current tries.

Corticoids

They are active medicine on the pushes (less sustainable, less grave). THE ACTH (nature or of synthesis) is replaced by the prednisone and its by-products. In this indication, we prefer the brief and intense cures (1 gram a day of injectable methylprednisolone during 3 days) today. We discuss the utility to make follow this flash of a cure from 15 to 20 days of prednisone per os. The continuous grip of corticoids per os does not give considerable advantages; She does not protect against the return of the pushes. She exposes to all the complications

of the corticosteroid therapy (among which the amyotrophy, the excess weight and the osteoporosis). She must be disadvised.

The immunosuppressive

The Azathioprine (Imurel)

Administered in long tea term (3 mg / kg / day orally), it has little intolerances and virtually no serious accidents. The possibility of obtaining a prolonged stabilization (in average forms) is apparent in practice, but not definitively proven.

Cyclophosphamide (Cytoxan)

Influence spontaneous worsening of the disease. It is used in treatments intravenously. It can stop or slow down a serious evolution. Besides complications (digestive, bladder), there is a risk oncogenic (dose-dependent, requiring to stop this when the total dose administered peaked).

Tea mitoxantrone

Did not the same risk oncogenic but cardiopulmonary toxicity. Results of has controlled motocross in very progressive forms of MS has demonstrated its ability to stop tea inflammatory process one MRI and 80 % reduction in tea case of relapses with has consequent improvement of disability over has six-month period.

Stupid man-interferon

Its action on the disease is regarded as established by the US and Canadian trial. It has capacity for a period of 3 years 30% reduction of the frequency of relapses and reduce the progression of the lesion load on MRI. Its effectiveness on disability is being evaluated. It is administered either subcutaneously (1 injection every other day) or IM (1 injection / week.) The side effects are dominated by a flu-like illness in the hours following the injection. It is the only drug to have obtained marketing authorization for MS. Its high cost approximately 8000 F per month.

Rehabilitation

Even with an evolutionary disease which can put in danger of the patiently won results, she deserves to be permanently practised. She is difficult when exist several associated deficits (pyramidal + cerebellar + proprioceptive). It is necessary to warn against the dumping in hot water (the thermal rise deteriorates the disorders of conduction in fibers démyélinisées). The vésico-sphincter reeducation can transform the social handicap at certain patients. The intervention of the occupational therapists facilitates the preservation within the framework of life usual.

A patient reached the MS develops disorders necessitateswork with speech therapist. -tea respiratory problems: there is has sound Pneumo lack of coordination, decreased tea amplitude of respiratory movements and has fast pace and disturbed. cerebellar -L'atteinte shows a slower flow rate, an abnormality of the laryngeal operation, dysarthria, hoarseness, an abnormality of the joint, of phonetics, a pseudo stuttering. Yew hyperkinesia can be observed hoarseness, has screaming stamp of transmission interruptions.

-We observed disorders of orofacial sphere with swallowing disorders: different time swallowing are not causing has clear accumulation of food residues gold salivated in tea pharynx, nasal regurgitation, false passages. At cognitive disorders there is a persistence of language: patients complain of seeking their words. There are also memory problems - very detrimental for patients who are still working - and attention disorders. In work with has speech therapist will help to achieve better control tea flow and improved articulatory distortions. Rehabilitation can only be implemented if the patient agrees, is working and is motivated. During rehabilitation we will consider -tea breath work: trying relaxation exercises, exercises control and strength of tea respiratory breath and sound Pneumo coordination exercises -tea work of tea palate: it is to strengthen tea muscles of soft tea palate to allow better differentiation of oral and nasal phonemes.

Examples: yawning mouth wide open, lips swell and explode them against each other, pronounce an "a" mouth wide open. The work of the musculature and coordination organs of phonation. The purpose of these exercises is to improve the accuracy and speed of the movements of the lips, tongue, cheeks and jaws: 2 cheeks inflate like balloons, or pursing his lips to keep air out and tap with the index swollen cheeks inflate one cheek and then the other, back in the cheeks aspiring; move the right jaw on the left, back and forth The work of the joint: it starts by determining the phoneme working, working praxis then envisages joint isolated phoneme. syllable repetition is then proposed to the patient containing the phoneme worked in different positions, repeating logatomes, words, sentences. For vowels can provide differentiation exercises. tea work flow, rhythm and melody: it contemplates tea repetition of words and sentences, text reading, conversation and then going to work one intonation sentences, texts, poems, sing songs known, perform skits.

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