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THE CONCEPT OF ASSIMILATORY SYSTEM OF PAULESCU DATING FROM 1912

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VII – THE LIVER

CHAPTER V
VASCULAR HEPATOPATHIES

There are two types of these hepatic disorders: arterial and venous.

ARTICLE ONE – ARTERIAL HEPATOPATHIES

§ 1 – HEPATIC ARTERY

Paludic and syphilitic arteritis
Obliteration and aneurism of the hepatic artery

Paludism, syphilis, tuberculosis etc. sometimes affects the hepatic artery, causing circumscribed lesions which result in the obliteration of its line or a dilation or aneurism of the vessel. Such diseases are generally met in young age.

Aneurisms have the size of a peanut, an egg or even a child's head (WALLMANN). Their walls have variable width, adhere to adjacent tissues and contain stratified clots. They may compress the portal vein and the bile ducts up to the point where they generate ascites and jaundice; they can also compress the duodenum and the stomach thus causing digestive disorders. They break frequently either in the peritoneal cavity or in the gallbladder, causing rapid death.

Aneurismal tumour manifests through neuralgic pains, pulsatile and intermittent, of such intensity that they have often been considered hepatic cramps. Sometimes they are accompanied by hematemesis and melena. Seldom jaundice and ascites occur.

The aneurism bag is usually large enough to move the liver; however, it is difficult to detect it given its in-depth localization.

The evolution of this disease is slow and intermittent; its duration is between one and several years; the regular ending is death dueto hemorrhage generated by rupture of the bag, syncope or, if the artery is obliterated, hepatic insufficiency.

Setting the diagnosis is among the most difficult, because in general the physician does not think of aneurism of the hepatic artery, but bile cramps. Nevertheless, it can be detected if the liver is moved and the pains are completed by hematemesis and melena.

The prognosis is serious.

In case of syphilis, the treatment consists of mercury massages and 2–4 g of potassium iodine a day. In case of paludism, iodine treatment is also recommended. If it is an aneurism, subcutaneous gel injections must be administered in order to coagulate the blood in the bag and prevent its rupture[2].

Liver arteriosclerosis

This disorder is a manifestation of herpes (see vol. II pag. 15); it is very rare as compared to renal, cardiac or brain arteriosclerosis.

The walls of the hepatic artery and of their branches have the internal surface dotted with yellowish spots which narrow the vessel. Here and there, in the places with the elastic tunic alters, the light expands. Moreover, a sclerosis tissue grows in the adjacent parenchyma and forms fibrous, retractile stripes on the arterial trajectory.

Another consequence of liver arteriosclerosis is hemorrhage which is relatively rare at the level of this organ due to the fact that the vessels are supported by conjunctive tissue in the portal spaces. We present a personal example: a 47-year-old woman, seized in May by apoplexy with right hemiplegia, died in September of cholera. The necropsy revealed, besides an intra-cerebral hemorrhagic focus and generalized arteriosclerosis, which also affected the hepatic artery, a 3 cm dark brown blood clot, in progress of transformation, located in the right hepatic lobe close to the upper surface.

Liver arteriosclerosis usually goes unnoticed, hidden by more visible phenomena of the kidneys, heart or cerebellum.However, a certain degree of meteorism, progressive weight loss, slow digestion, poorly colored feces, hemorrhages and other accidents of hepatic insufficiency are symptoms which can be attributed to liver arteriosclerosis.

The evolution of this diseases is slow and insidious and its duration unknown.

It is very difficult to diagnose this disease and the prognosis is serious because of anemia and degradation common in these disorders.

The treatment is not different from that for arteriosclerosis; it consists of prescribing potassium iodine and arsenic from the very beginning of arterial alteration. Subsequently, in case of uremia and hepatic insufficiency, purgatives and diuretics must be used.

Thrombosis and embolisms of the hepatic artery

Etiology and pathogenicity – The celiac trunk and the hepatic artery alter during paludism, syphilis, tuberculosis and herpes (arteriosclerosis) and when the alterations narrow down and block them, thrombosis occurs.

Embolism, independent of any arterial lesion, results from the emigration of a fibrous cyst or a fragment of atheromatous plaque (originating in the pulmonary veins, heart or a point of the arterial system) which stop in the hepatic artery, obliterating it.

Pathologic anatomy – The right angle formed by the celiac trunk with the aorta and the low caliber of the hepatic artery help us understand the rarity of the embolisms of these vessels. In what follows, we present two personal cases[3]. The former refers to a 50-year-old man, with mitral insufficiency and dilation of the heart cavities. His condition improved under the influence of foxglove, but towards 2 p.m. he suddenly felt in the right hypochondrium pains of an excessive intensity, which made him cry. Soon, the abdomen meteorised, the feces discolored, the face features altered; bilious vomiting occurred, then the patient displayed states of delirium, somnolence and died 12 hours later. The necropsy revealed a large liver, filling

[2] LANCEREAUX şi PAULESCO, *Bull. Acad. de méd.,* 22 iunie 1897, pag. 804.
[3] E. LANCEREAUX, *Traité des maladies du foie,* Paris, 1899, pag. 541.

a significant part of the abdominal cavity. With reduced consistency, this organ was suffocated by blood which was streaming from the section, with the aspect of a sponge soaked in liquid. In the hepatic artery we found a fibrous plug of cardiac origin, very tough, which blocked it on its entire diameter. The celiac trunk and the other arteries were free and intact.

The latter case probably suggests thrombosis. It refers to a 65-year-old man, suffering from generalized arteriosclerosis and necrosis of both legs. He was seized by violent abdominal pains, agitation and delirium, then he fell into a semi-comatose condition and died 2 days later. The liver, highly congested and filled with blood, was very friable and the tissue dispersed under the action of a gush of water. The hepatic artery was sclerosed, dilated and contained a small adherent cyst ending in a peak.

Experiments proved the functional importance of the hepatic artery in liver nutrition.

Symptomatology – The beginning of this disease is dramatic and is accompanied by strong pains, of excessive intensity, in the right hypochondrium. The liver rapidly increases in volume and becomes painful when pressed. Sometimes, a sensation of vomiting occurs. The face features alter, eyes sink into sockets, extremities turn cold; the patient is weak, display delirious conditions and succumbs to coma.

The evolution is faster in embolism than in thrombosis, particularly if the obliteration is complete; the duration varies from several hours to 2–3 days.

Semiology and treatment – Diagnosis is easy, given the coexistence of above-mentioned phenomena with cardiac and arterial alterations.

The prognosis is among the most serious.

There is no treatment. The physician shall have to calm down pains with morphine.

Albuminoidal liver degeneration

(Syn. *Amyloid degeneration. Hepatic leukocytosis*)

This disorder consists in a deposit on the walls of hepatic arterioles, capillaries and other organs of a homogeneous and transparent albuminoidal substance, which has the property to turn brown-red under the action of iodate water. Relying on this reaction, VIRCHOW mistook this substance foramyloid carbohydrates and inappropriately calls it *amyloid.* We called this disorder albuminoidaldegeneration or hepatic leukocytosis.

Etiology and pathogenicity – The usual causes of leukocytosis are paludism, syphilis and especially tuberculous suppuration of bones and joints (coxalgia, the diseases of Pott); exceptionally, it is met in the case of old purulent ulcers of the legs, in old bronchitis etc. It affects both sexes, at any age, particularly in adolescence.

Pathologic anatomy – The liver is highly tumid and sometimes weighs 2, 3, 4, 5 kg and more. Its surface is smooth and shining, it has the consistency of soap and a reddish or grey color, which is not uniform on the entire surface. The sharp edge is dense and rounded. In section, the organ has the aspect of a piece of lard and contains little blood; it is tough and resistant, yet friable. Touched with a iodine solution, it acquiresa red hue which, when adding sulfuric acid, turns violet and, more rarely, blue. The bile ducts are free and the gallbladder contains a small amount of watery bile.

The microscopic analysis reveals that the walls of the arterioles and capillaries are narrow and homogeneous; they narrowdown the vessel caliber and end up devoid of appreciable structure. The venous branches are little changed. Hepatic cells are compressed, atrophied and, in some cases, impregnated withthe same substance as the vessels. However, this cellular alteration is rarely uniform, some parts of the liver being preserved because of the albuminoidaldegeneration.

The spleen, whose relative tumefaction can exceed that of the liver, the kidneys, the lymphatic ganglions,the intestinal mucous membrane are generally similarly altered.

Symptomatology – This disease has an insidious start. In a more advanced stage, it translates into the increase of the hepatic dullness, which exceeds the costal margin and descends to the naveland lower. Palpation reveals that the liver surface is smooth and firm and the anterior edge is narrow and rounded. The same examination procedures show that the spleen is very tumid and its surface is smooth.

At the same time, the patient complaints of a sensation of fullness in the right hypochondrium and displays anorexia, tympanism, sometimes mucous vomiting and persistent diarrhea.

Jaundice and ascites are exceptional and refer to the compression of the hilum organs by tumid ganglions.

The urine, considerable at the beginning, more reduced later, is frequently albuminoidal and symptoms of uremia are common.

Patients lose weight and stop eating; they have a pale, yellowish color similar to wax, and discolored mucous membranes; they are completed by a swelling of the teguments and, more rarely, edema. Finally, hemorrhages, particularly epistaxis, occur accompanied by phenomena of hepatic insufficiency and the patient succumbs to coma.

The evolution of this disease is slow and its duration can spread onto severalmonths or even years. Healing is rare, since it is difficult to remove the substance which impregnates the tissues and the ending is almost always fatal.

Semiology – Diagnosis relies on the condition of the liver and the spleen, albuminuria, digestive disorders during prolonged suppuration or a disease such as tuberculosis, syphilis etc.

The prognosis is serious; it becomes lethal when the disorder is old and spreads to the kidneys.

Prophylaxis and treatment – The prophylaxis of hepatic leukocytosis consists of prolonged suppurations, tuberculosis and syphilis.

The treatment is almost inexistent. The physician shall prescribe hydrotherapy; when applicable, as is the case of syphilis and paludism, iodine-based substances and sulfurous waters or sodium chlorides can be partially helpful. Opiates and astringent substances shall be used against diarrhea and diuretics and purgatives against uremia.

ART. II – VENOUS HEPATOPATHIES

A – THE PORTAL VEIN

The portal vein is in contact with pathogenic agents absorbed in the intestine, which are toxic substances and microbes. At the exterior, it can suffer from the action of mechanic agents, capable to compress and slow down the blood stream.

These agents determine phlebitis alterations which we shall study in two categories: *mechanic and toxic phlebitis and microbial pilephlebitis.*

Mechanic and toxic pilephlebitis

The mechanic obstacles (tumours, ganglions, peritonitis, cirrhosis) which compress the portal vein and slow down blood circulation simultaneously irritate the walls of the vessel, causing local blood coagulation and the formation of a thrombus.

Alcohol and wine are one cause of portal vein phlebitis, if we consider the relative frequency of this disorder in alcoholic cirrhosis.

In the irritated spot, the endothelial cells of the internal tunic, but also the elements of the other tunics of the veingrow. The internal surface of the vessel becomes rough, unequal and the blood flow is slowed down by this obstacle; a solid thrombus grows, adherent to the wall, which forms concentric layers, which become the deposit of calcium salts (calcification). The vein has the aspect of a solid cord; it is resistant, thicker than usual and has a grey color. The clotblocks the vessel diameter partially or totally and spreads to its main branches.

The liver is sometimes affected by enolic cirrhosis; other times, it is toughened and atrophied by the withdrawal of the portal vein branches.

The spleen is tumid by blood stasis; the digestive tube is congested and retracted; the peritoneum, hyperemiated, contains abundant ascites.

The clinical phenomena have an insidious beginning. Ascites is the first and primary symptom; it occurs suddenly and soon reaches a considerable extent, so that it requires frequent repeated punctures, which rapidly exhaust the patients. Subcutaneous abdominal veins dilate considerably, from the lower parts of the abdomen to the axillae, being similar to those noted in the obstruction of the vena cava.

Live examination reveals that it is first tumid, then reduced. The spleen is large.

We note anorexia and disgust for food; after a while, viscous vomiting and mucous and bloody diarrhea occur. There are also stomachand intestinal hemorrhages, which sometimes avoid ascites, reducing the pressure excess of the portal vein. The urine is scarce. Jaundice and fever are absent.

The patient loses weight rapidly; his legs are swollen and he succumbs to marasmus.

The evolution of this disease is continuous and progressive, its duration being of several months; the ending is fatal as a result of ascites (if punctures are not performed on time if ascites is too much withdrawn), considerable hemorrhages, hepatic insufficiency.

Setting the diagnosis of this form of piphlebitis relies on specific symptoms which distinguish it from various types of cirrhosis. The epithelium of bile ducts can sometimes enter the portal vein and causes the formation of a thrombus. The obliteration of the lower vena cava is followed by the dilation of superficial abdominal veins and edema of the legs, in piphlebitis, in exchange, such phenomena follow ascites.

The prognosis is among the most serious.

There is no cure. The physician shall fight diarrhea and hemorrhages through astringents and ergotine. He shall perform paracentesis as soon as there is danger of chocking and shall only partially empty the abdominal cavity in order to avoid syncope.

Microbial piphlebitis

Paludism and syphilis sometimes affect the portal vein, giving rise to phlebitis which resembles the one caused by mechanic and chemical agents (see above).

Suppuration and gangrene microbes generate*purulent piphlebitis*that shall be described below:

Ulcerous lesions of the stomachand intestine, particularly those of the cecum appendix, are the regular causes of purulent and gangrenous piphlebitis. In a personal case[4], for instance, caused by a needle which perforated the cecum, suppuration affected even the hepatic veins; the right pleura contained a very fetid liquid; the corresponding lung and the brain also contained gangrenous abscesses.

A hepatic calculus, a hydatyd cyst opened into the bile ducts, the suppuration of the bile, of mesenteric ganglions and especially of bile ducts near the portal vein can also generate purulent piphlebitis.

The portal vein, partially altered, has narrowed walls and contains a purulent clot, little adherent, made of red cells and leukocytes. The liver also hosts multiple gangrenous abscesses; sometimes, suppuration reaches the pleura, the lungs and other organs.

The beginning of purulent piphlebitis is usually masked by an initial disorder: appendicitis, ulcers of the stomach or intestines, suppuration of the spleen, mesenteric ganglions etc.

Usually this disorder manifests through pains in the epigastrium, the right hypochondrium or the cecum region, depending on whether the portal vein is affected at the level of the trunk or branches. Pains are accompanied by fever in remittent crises, with intense shivers and abundant perspiration.

The abdomen is affected by meteorism and becomes painful; the liver and spleen increase volume; these are followed by lack of appetite, greenish vomiting, bilious or bloody diarrhea, very rarely ascites with dilation of superficial abdominal veins and, if phlebitis is caused by the propagation of suppuration of bile ducts, mild jaundice with discoloration of the feces. At any rate, teguments turn pale, the features alter, weakness and weight loss increase; hemorrhages, mainly epistaxis and petechias appear at the level of the legs, urine becomes scarce; these are accompanied by phenomena of pulmonary, pleural, articular suppuration; finally, nervous accidents (delirium, somnolence) lead to coma and death.

[4]E. LANCEREAUX, *Traitéd'Anat. path.,* vol. II, pag. 962.

The evolution of this diseases is progressive and sometimes lasts for a few days, other times extends to several weeks. The ending is usually death, as an effect of purulent infection, more rarely hepatic or renal insufficiency.

Setting the diagnosis of purulent piphlebitis follows the symptoms, whose complex is pathognomonic. This disease distinguishes from liver abscess, with which it often coexists through signs of venous obstruction.

The prognosis is very serious.

The prophylaxis consists in laparotomy as soon as a purulent focus is formed in the abdomen.

There is no cure. However, pain must be fought with opiates, fever with quinine and aspirin, diarrhea with bismuth subnitrate.

B – THE HEPATIC VEINS

The hepatic veins, which continue the portal vein, display more rarely alterations similar to those of this vessel. We shall study *mechanic and toxic phlebitis and microbial phlebitis.*

Mechanic and toxic phlebitis of hepatic veins

This disorder seems to have the same causes as adhesive piphlebitis. In a case reported by FRERICHS, a 45 old worker, a heavy drinker of plum brandy, was seized by jaundice and ascites and succumbed to coma. When we examined the liver, we noted that the orifices of hepatic veins were narrowed by clots and one of them was completely obstructed by blood concretion. Some branches of the portal vein contained thrombi.

The symptomatology of this morbid condition resembles that of piphlebitis of the same origin. The hepatic tissue is congested, even sclerosed.

It is difficult to set the diagnosis and the prognosis is very serious.

There is no cure.

Microbial phlebitis of hepatic veins

There are cases of hepatic phlebitis caused by syphilis. For instance, there is the case noted by GÉE of a 16-year-old child, edemized for 2 months, who died of an abdominal puncture. The liver was congested, hardened, sclerosed and dotted with small nodules; several hepatic veins had orifices blocked by adherent cysts. Thoracic and epigastric veins were dilated.

Purulent phlebitis is more frequently met than syphilitic phlebitis.

It is consecutive to the propagation of the abscess located in the liver to the neighboring hepatic veins and has mainly arterial origin; it can also be caused by ulcerations of the intestine, even abscesses of the pelvis.

The internal surface of the veins becomes tough, unequal and covered with fibrous deposits which soon turn to pus. Carried to the brain with the blood, the pus is sent first to the lungs or pleura, which suppurates; then it spreads to the entire organism, causing a purulent infection or pyemia.

The symptoms consist of intense fever, remittent or irregular, with violent shivers and abundant perspiration followed by a condition of excessive prostration, agitation and delirium. The teguments are discolored and acquire a yellow lead hue; finally, the patient succumbs to coma.

The cure, little effective, isthat of pyemia and consists of cold baths and quinine in considerable doses.

Passive liver hyperemia
(Syn.: *Cardiac liver*)

We shall study here a very important disease from the point of view of diagnosing heart diseases, being the *criterion of asystole,* namely cardiac insufficiency.

Etiology and pathogenicity – Any obstacle in the circulatory tree, from the hepatic veins up to the aortic orifice, generates stases in the hepatic veins; this is the result of the obstruction of the inferior vena cava over the diaphragm, the narrowing and insufficiency of the cardiac valves; the dilation of the right

heart, arterial cardiopathies and emphysema, tuberculosis and sclerosis of the lungs, dilation of the bronchi, some old pleurisy, deviations of the spine, vicious thoracic conformations etc.

The pathogenicity of this stasis is easy to understand; the blood existing above the obstacle, for instance in the aortic narrowing, dilates the cavities of the left heart, the vessels of pulmonary circulation, the cavities of the right heart, the vena cava and the hepatic veins; then the capillaries of the hepatic lobes and finally the branches of the portal vein. The compressed hepatic cells atrophy while the conjunctive tissue gradually expands and can cause, in certain cases, a sclerosis of the organ.

Pathologic anatomy – The volume of the liver grows; its capsule extends, it becomes violet and acquires a firmer consistency. When sectioned, black blood drains and the surface of the section presents a mosaic made of two colors (a red violet area around central veins and a lighter, yellowish area at the periphery of the lobes) which determined an English author to compareit with nutmeg (nutmeg liver).

After a period, liver becomes slightly granular, with increasingly hard consistency, and the volume tends to diminish. Thus, the process has two stages: one of hyperemia, another of hyperemia and sclerosis with slight retraction of the hepatic tissue.

The microscopic analysis reveals that at the beginning the lesion is formed by blood accumulation in the central veins and capillaries of the lobes; later on, as the branches of the portal vein dilate, the walls of the central veins narrow down and the neighboring conjunctive tissue proliferates. The hepatic cells of the lobe focus, compressed and atrophied, display protein and fat granulations. The cells at the periphery of the lobes keep their integrity. The portal spaces remain intact.

The spleen and pancreas, tumid and congested at the beginning, harden and retract. The injected digestive tube presents a grey pigmentation of the mucous membrane; the walls seem narrow and shortened. The mesenteric ganglions are pigmented. The kidneys are tumid and violet, hard and retracted. The legs, the trunk, the upper limbs and the peritoneal, pleural, pericardial cavities have an edematous infiltration or a citrine exudate of variable size.

Symptomatology – The first symptom and one of the most important of passive liver hyperemia is the tumefaction of this organ, revealed upon palpation and percussion. The anterior edge of the gland goes down under the costal margin, exceeding the navel and reaching the iliac crest. Its surface, smooth and with various degrees of toughness, provides pulsations, felt upon palpation, synchronous with ventricular systoles.

At the same time, liver becomes *painful* when pressed. The pain is very intense and, if the liver is suddenly pressed under the costal margin, it brings patients on the brink of crying and contracting their abdominal muscles to protect themselves. This pain decreases when the liver becomes sclerosed.

The patient complains of a very uncomfortable feeling of weight in the right hypochondrium and the epigastrium. He also complains of lack of appetite, nausea, eructation, slow digestion, diarrhea etc. The teguments, cyanosed, acquire a yellowish color, but the urine does not contain any bile.

In a more advanced stage, ascites occurs, made of a serous-hemorrhagic liquid which is sometimes bloody. The urine, rare, concentrated, very colorful, often contains albumin.

These disorders are preceded and accompanied by accidents specific to cardiac or pulmonary diseases, which represented the starting point; the patient gasps, the pulse is weak and irregular, the face, swollen and violet, the limbs and often the trunk arefull of edemas.

Under the circumstances, the amount of urine drops; the patient is somnolent, is seized by delirium and succumbs to coma.

The evolution of this disease, although progressive, follows asystole, with increases and remissions. The duration, usually long, is several months or years. The usual ending is death, as an effect of uremia and initial cardiac or pulmonary disorder; rarely, there is hepatic insufficiency, when the cells are destroyed and jaundice occurs.

Stasis hepatic hyperemia can cure when the circulating obstacle is suppressed, but this situation happens very rarely because of the persistence and gravity of the lesions which represent its starting point.

Semiology – This disease is diagnosed based on two main symptoms: regular liver tumefaction and intense pain, determined by percussion and sudden pressure; they are completed by accidents related to cardiac, pulmonary, thoracic lesions. It is easily distinguished from cirrhosis, lymphoma and albuminoidal liver degeneration.

This form of hyperemia is the best symptom we have to identify the functional condition of the heart, because, being better than a heart murmur which is granted too much importance, it allows us to determine the scope of this form of insufficiency.

The prognosis is serious.

Prophylaxis and treatment – The prophylaxis of this diseases consists of the removal of the circulatory obstacle generating it. This indication is envisaged by the treatment, which must be dynamic and administered from the very beginning. Revulsive drugs, potassium iodide in cardiac diseases, opiates and cold hydrotherapy in pulmonary diseases, corsets and methodical exercise in deviations of the spine bring many benefits.

Subsequently, the physician can only fight liver hyperemia and stimulate the action of the heart.

Leeches applied to the anus, cups applied in the epigastric region and especially repeated purgatives (podophyllin, evonimine, rhubarb, calomel, neutral saline substances) are the first recommendation. An exclusively dairy diet, combined with coffee, tea, alcohol, theobromine, caffeine, has the advantage of increasing diuresis.

At the same time, we use macerated foxglove in infusion or as pile (associated with violets and scammonies, 5 cgr.) from which we shall administer five up to eight a day, for four-six days, repeated when necessary.

If foxglove cannot influence the heart, we shall prescribe drastic purgatives (scammonies, turpeth, resin, in tablets or as German brandy or irrigation) and we shall not hesitate in administering these drugs every two or three days, if diarrhea persists.

CHAPTER VI
NERVOUS HEPATOPATHIES

The liver has two types of nerves: branches of the pneumogastric nerve and branches of the sympathetic nerve which originate in the solar plexus. The functional lesions or alterations of these nerves generate various and multiple disorders. In turn, liver lesions can influence the nervous system and cause reflex disorders.

Hepatic cough – Sometimes, when the liver is tumid following enolic cirrhosis and especially paludic cirrhosis, amyloid degeneration, stasic hyperemia, hydatyd cyst etc., in certain positions such as laying or standing, the patient presents s a contraction of the pneumogastric branches which manifest through crises of dry cough.

Usually there are no nervous lesions.

The patient, who does not cough before going to bed, is seized by it immediately as he lies down, especially on the left, accompanied by a feeling of discomfort, weight in the right hypochondrium and simultaneously, a tinkling in the throat which causes crises of dry violent cough, which only cease if the patient changes position. More rarely, the cough occurs when the patient is standing.

This cough, which ceases when the liver tumefaction decreases, sometimes extends for several months and years. A young woman, affected by an enormous liver hydatyd cyst, had a dry noisy cough which stopped immediately after the puncture of the cyst.

The hepatic origin is recognized due to the fact that the cough occurs only in certain positions, being often preceded by a feeling of discomfort, it is dry and there is no concomitant, pleural, pulmonary, bronchial, tracheal or laryngeal lesion.

The prognosis is serious, since this cough can determine the dilation of the right side of the heart or aggravates it when it occurs.

The treatment consists of fixing the liver with an appropriate bandage; morphine, heroin and quinine (1 g in 2 tablets, administer at night, during supper) also calm down this cough, but their use cannot prolong indefinitely.

Asthma crises sometimes originate in the liver. For instance, LANDAU consulted a woman who had been having asthma crises for 6 years and who stopped having any after the surgery of a large liver hydatid cyst.

Hepatic neuralgia – Hepatalgia manifests as intermittent crises at relatively regular intervals, in nervous, hysteric or herpetic persons; it is followed by jaundice which sets it apart from bile cramps.

We present one of our cases[5]. A 35-year-old woman, in good condition, when returning to the country, was seized in the right side by very intense pains which irradiated towards the stomachand shoulder. These pains occurred in crises and stopped only to reappear the next day, approximately at the same time. Treated with quinine, they disappeared after four days never to appear again.

This form of neuralgia has multiple occasional causes; in general, it is the effect of a toxic, microbial or constitutional disease or the consequence of a local lesion of the nerves or spine. It is met in (lead cramp, with greenish vomiting), paludism, hysteria and herpes, syphilis (crises called gastralgic), mechanic irritations of the hepatic nerves etc.

Hepatic neuralgias translate into sharp intense pains in the right hypochondrium and the epigastrium, irradiating to the right shoulder and repeating at short intervals. They are accompanied by a feeling of choking, eructation, food or bilious vomiting. Sometimes, there are convulsive spasms of the hands and legs. The face turns red, the conjunctive mucous sometimes turns yellow, the upper region of the abdomen and the corresponding portion of the back are sensitive, the liver becomes tumid and breathing movements speed up.

These crises last for two or three days, after which they stop only to reappear, irregularly or periodically, a few days, months or weeks later. In a woman, crises reappeared on a regular basisduring menstruation (FRERICHS).

Once the crisis is gone, the patient recovers rapidly.

The pains of hepatic neuralgia are very similar to those of bile cramps, so that sometimes there is a confusion between the two diseases. Sometimes, the absolute intermittence of the crises, the periodicity and absence of consecutive jaundice are signs of hepatalgia.

The prognosis is generally benign, this disease being short, without any threat to life.

The treatment varies with the cause. Quinine (1–2 gr.) is effective in case of paludism or herpes. Antipyrin, opiates, injected morphine are recommended when the pains are very intense, mainly in syphilis crises. Hydrotherapy can prevent the recurrence of accidents; finally, a certain diet is recommended, a dairy based one being effective in this case.

Emotional or spasmodic jaundice

Following a strong or sudden emotion (anger, fear, violent pain), mild jaundice occurs. Other times, jaundice is late; it only occurs after a few days from the emotion and coincides with an infectious disorder of digestive function (gastric disorder); in this case, its features and duration are similar to catarrhal jaundice, from which it is difficult to distinguish.

This type of jaundice preferably affects young emotional persons, particularly hysteric women (out of 9 personal cases, 6 are women), patients with herpes, alcoholics. An unmarried young woman acquired jaundice shortly after she discovered that she was pregnant (NORTH). When a raging dog darted at him, a young monk was so frightened, that he lost consciousness and soon turned yellow (VILLENEUVE).

ODDI's research provides the key to this phenomenon. He noted that after the ablation of the gallbladder, the choledoc dilates and turns into some sort of bile reservoir; he also noted at the orifice of this channel a layer of smooth fibers, independent from the intestinal muscle, which form a sort of sphincter which regulates bile drainage and which prevents the intestinal content to enter the bile ducts. The spasmodic contraction of this sphincter generates emotional jaundice, while paralytic relaxation creates jaundice which follows gastric disorders.

Spasmodic jaundice usually occurs suddenly, within 24 hours at the latest after a strong emotion. Teguments turn yellow and even acquire the hue of saffron; the urine, dark, contains bile pigments; the feces are grey and whitish.

These are accompanied by lack of appetite, nausea and sometimes muscular pain, weakness and a bad general condition. The liver remains normal and the spleen keeps its regular volume contrary to catarrhal jaundice.

[5]E. LANCEREAUX, *Traité des maladies du foie et du pancréas,* Paris, 1899, pag. 593.

These phenomena stop a few days later; in a 22-year-old young man who had jaundice which occurred the next day after a violent crisis of rage, urine pigmentation and discoloration persisted for only 5 days.

The evolution of this form of jaundice is continuous and its duration, usually several days, rarely exceeds 15 days. It is longer if emotional jaundice is completed by infectious or catarrhal jaundice. The disease cures.

The diagnosis of emotional jaundice relies on so-called pathognomonic signs. Catarrhal jaundice is preceded by digestive disorders, is accompanied by tumefaction of the liver and spleen and lasts over 15 days, which distinguishes it from emotional jaundice.

The prognosis is not serious; if, in an exceptional case, there are any symptoms of serious jaundice which add to those of emotional jaundice, it means that an infection completed the nervous disorder.

The treatment consists of administering antispasmodic drugs and opiates. The patient shall rest, shall take cold baths and shall be administered cold water irrigations. A dairy-based dietis convenient, given the lack of appetite and avoiding dangers of gastric disorder, with subsequent catarrhal jaundice.

Nervouspolycholia – In the morning or during daytime, after eating, some persons have liquid, abundant and greenish feces, without any significant disorder. The beginning of a hepatic colic crisis is often marked by abundant bilious vomiting. This accident is caused by an excess of bile, determined by a reflex act which originates in the arrival of food in the stomach or the presence of a stone in the bile ducts.

Sometimes, polycholia is direct and results from a lesion of the nerves (peritonitis, tuberculosis of suprarenal glands, lead colic); under the circumstances, the abundant bile is eliminated through vomiting. Other times, polycholia refers to disorders of the spine or cerebellum (gastric crises of syphilis, meningitis, uremia) which are also accompanied by abundant bilious vomiting.

The substances that are vomited and those eliminated through the feces contain a high amount of pigments and bile salts.

Hepatic movements

(Syn.: *Hepatic ectopia and mobility*)

Hepatic ectopia, also known as *mobile liver and fractured liver*, is more common in women than in men. It is usually noted in herpetic persons who suffer from dyspepsia, constipation, hernia, moving kidney, gastroenteroptosis etc. and refers to a trophic disorder of the fibrous parts of the abdomen. The relaxation of the abdominal wall in women with several children, repeated pregnancies, the abusive use of the corset are additional causes of liver ectopia.

The narrowing and reduced resistance of the fibrous tissue of the abdomen is the only visible lesion. The liver, displaced, is maintained by elongated and lax ligaments.

The symptoms consist of plenitude, a sensation of weight, contraction in the right hypochondrium, often accompanied by irritating pains, especially following efforts, irradiating along the right intercostal spaces. The exam reveals in the hepatic region a mobile, smooth and unpainful tumour which extends downwards through a sharp edge up to the navel, ilium or pubis. It can drain upwards; it leaves the normal liver localization free, where percussion reveals a tympanic sound instead of dullness.

Hepatic ectopia is not always easy to diagnose and sometimes ii is mistaken for renal ectopia or abdominal tumour.

The prognosis is not very serious.

The treatment consists of wearing a well-made belt, in order to avoid the feeling of weight and contraction. If it does not relieve the patient's painenough, surgery is necessary tofasten the liver to the abdominal wall.

With spontaneous hepatic ectopia we associate the displacements suffered under the influence of *mechanic causes*, located in the thorax or abdomen, such as: pulmonary emphysema, pleurisy with drainage, pneumothorax, thoracic tumours, deviations of the spine – pregnancy, ovarian cyst, fibrous uterine bodies, tumours of the mesenteric muscle, kidneys, spleen, gastro-intestinal tympanism etc.

The treatment is the same as for spontaneous ectopia.

We must add a few words on liver displacements caused by abusive use of a very tight corset. Under its action, the liver is deviated downwards and leftwards and seems to have an increased volume. It surface has a series of parallel striations resulting from an annular constriction of the abdomen and often a portion of the right lobe is separated from the remaining gland through a ridge, at the contact withthe costal margin.