A REMARKABLE CASE OF PACHYMENINGITIS HYPERTROPHICA PRESENTING SPINAL BLOCK AND FROIN'S SYNDROME.

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INTRODUCTION.

IN another paper²⁴ I published the histories of two patients in whom an obstruction of the spinal canal, caused by tumour, could be demonstrated by the existence of Froin's syndrome (xanthochromia and massive coagulation of the cerebrospinal fluid). In both cases the presence of spinal block was confirmed by the result of röntgenography. In the first patient injection of air by means of lumbar puncture led to an exact determination of the level of the tumour, as the air was seen to be confined underneath the obstruction and to form a distinctly less opaque column, which reached as high as the ninth thoracic vertebra. At the operation an extramedullary intradural fibroma, which was seated at this level, was removed and afterwards the patient recovered nearly completely, presenting only a very small remainder of the paraplegia, which for several months before the laminectomy had been a total one. In the second patient lipiodol was used as a contrast object; it was injected by the lumbar route, the patient's pelvis being raised. It stopped at the level of the tenth thoracic vertebra, which when laminectomy was performed appeared to be affected by a sarcoma. Neither the operation nor X-ray treatment, which was then tried, had any success. Without having presented even a temporary improvement of the total transverse lesion of his spinal cord, the patient died a few months after the operation.

When in 1903 Froin for the first time called attention to xanthochromia and massive coagulation of the cerebrospinal fluid, he thought that the syndrome was found only in cases of tumour either of the spinal cord itself or of its membranous or bony surroundings. But later on it appeared to develop sometimes in cases of spinal block of the most varied kind. Mingazzini¹¹ mentions its presence in Pott's disease, in luxation or fracture of the vertebræ and even in cases where no real blocking of the spinal canal exists. Babonneix and Voisin¹ emphasize its frequency in pachymeningitis hypertrophica cervicalis. This disease consists in a hypertrophic inflammation of the dura mater, which owing to the extreme degree of thickening of that membrane in its progression always leads to a total obstruction of the spinal canal. Though the inflammatory process usually has its localisation at the level of the cervical enlargement, it may be found in any other part of the spinal cord and in that case gives rise to very uncommon clinical symptoms, so that the diagnosis becomes very difficult or even impossible. The spinal cord itself rarely remains unaffected;

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in many cases holes in its substance are seen, which much resemble the anatomical changes found in syringomyelia. The pathogenesis, however, of these cavities is not known as yet, though Müller¹² mentions that it is probably not the same as in ordinary syringomyelia.

The following case, which is interesting from its many clinical and anatomical peculiarities and by the fact that it presented Froin's syndrome, may be put on record.

CASE HISTORY.

M.d.V., postman, age 22 years, came into the clinic in the beginning of March 1924. In his previous history nothing remarkable was found.

Present Illness.—About the middle of January, 1924, he was aware of a slight numbness of the toes, first in his left foot and a few days after in his right. Soon the numbness, connected with a prickling sensation, rose to the ankles, the knees and even to the gluteal region, but he could do his work well and did not pay much attention to his troubles. In the beginning of February he suddenly got a sharp pain in his back and his chest, which was a little better when he was walking. At the end of the month he began to complain of severe sphincter troubles: he could no longer pass urine spontaneously and had to be catheterised. Defaecation too was difficult. A few days later the patient could not get up from his bed: the legs had become progressively more and more powerless, though the pains in back and chest became rather less.

Somatic examination did not reveal any deviation in any of the internal organs. The urine contained some leucocytes but no albumen.

Examination of the vertebral column and nervous system.—Mobility of the vertebral column rather good; no deformation. Pressure on the fifth dorsal spinous process, and to a less degree on the sixth, was painful. Skull and cranial nerves did not present anything abnormal, nor did the arms. The muscle strength of the trunk was reduced; the upper abdominal reflexes were diminished, the middle and inferior reflexes absent. Both legs were paretic; the muscle tonus was very poor; the kneejerks and Achillis tendon reflexes were diminished; Babinski's reflex was not obtained. Sensation was diminished for all qualities below the groins, so that for tactile and painful stimuli a total anaesthesia, for thermal stimuli a hypaesthesia was found. Above this field of diminished sensation, up to the umbilicus, a marked hyperaesthesia and hyperalgesia were present, but at a higher level an anaesthetic zone of about 10 cm. breadth was present again, which was limited both above and below by a small zone of hypaesthesia. Above this zone a field of hyperaesthesia and hyperalgesia existed, extending as far as the third rib.

During the next few days the patient had complete retention. The urine was removed twice a day by catheter. He was somewhat feverish, but did not present alarming symptoms. In X-ray photographs of the vertebral column nothing abnormal was seen. After a week both legs had become totally paralysed; the hyperalgetic field on the abdomen disappeared. A few days after the patellar and Achillis tendon reflexes were no longer elicited. The upper abdominal reflexes too had disappeared.

The diagnosis of a transverse lesion of the spinal cord was readily made, but it was more difficult to determine its nature. The rather acute though insidious beginning, the rapid progression, the ascending character of the sensory level, and the fever seemed to point to a myelitis. A vertebral lesion could not be diagnosed, though the painfulness of the fifth dorsal spinous process perhaps seemed to point in that direction, for the X-ray photographs of the vertebral column did not reveal anything that could be regarded as a sign of disease. Besides, the flaccid paraplegia of the legs could not well be explained

if there was only a disorder in the cervical and upper dorsal cord ; it must have been seated at a much lower level as well. A tumour therefore did not seem very probable, but it was felt that lumbar puncture might point more in that direction.

The latter was performed because the patient began to complain of pain in his neck and presented some neck rigidity. The pressure of the cerebrospinal fluid was low ; the fluid itself was distinctly yellow and clear, and within two hours it presented massive coagulation. Pandey's and Nonne's reactions were highly positive ; no sugar appeared to be present. The fluid contained very few erythrocytes and a single leucocyte. The yellow colour appeared not to originate from hæmoglobin, but from lutein. No micro-organisms could be cultivated on ascites-agar. A second lumbar puncture a week after gave the same result.

Gradually formication in both hands developed and the muscle strength of the arms diminished somewhat. Now and then attacks of hiccough occurred, which were very distressing. They were, however, rather easily controlled by the administration of $\frac{1}{4}$ mgr. of atropine sulphate.

It was now clear that an obstruction of the spinal canal was highly probable. As no vertebral lesion of any kind was present, it was considered to be due either to a tumour or to an inflammatory process within the spinal canal itself. In the last few weeks the condition had progressed and the motor as well as the sensory troubles had reached the arms. This course seemed to speak in favour of an inflammation, for it is not easy to see how a tumour can give rise to such a development on the clinical side. But a mere myelitis on the other hand is not likely to cause an obstruction of the spinal canal. So the uncertainty was not done away with and the further clinical course did not bring much more light.

The patient gradually got worse and the attacks of hiccough became more and more frequent. By the end of March the hyperalgesic zone had reached the clavicle and only a few cm. lower a total anaesthesia and analgesia existed. From the beginning of April the patient had nearly every day a rigor with a rise of temperature to 40°, 41° or even 42° C. Between those acute elevations the temperature fell again to the normal level. The hiccough in the meanwhile became more and more intractable towards the end of April drugs no longer influenced it. In the beginning of May, 1924, he died.

The autopsy was made by M. Mieremet. In the region of the lesser curvature of the stomach a large ulcer was found. It appeared to be very recent, so that it could not have any bearing on the etiology of the spinal cord affection. Apart from a purulent cystitis and pyelonephritis, which were to be expected, no disturbances of the internal organs were present.

The spinal cord was very difficult to remove ; in the dorsal region it was very soft and adherent to the dura, and could not be separated without tearing the pia. A yellowish mass protruded which gave the impression of being pus. Microscopical investigation, however, showed that it did not contain leucocytes or microorganisms, but was only an indefinable mass of detritus. When incisions were made at various levels of the spinal cord a cavity appeared to be present in the deepest part of the left dorsal column which as far as could be made out microscopically did not communicate with the central canal. It extended from the third cervical segment to the lumbar region, though in the dorsal cord it could not be traced because of the total destruction there present. The white matter in the dorsal and lateral columns in the cervical and lumbar segments seemed to be somewhat degenerated. The meninges were much thickened.

M. Mieremet kindly gave some pieces of the spinal cord to the laboratory of our clinic, where they were cut and stained with hæmatoxylin-eosine and by Van Gieson and Weigert-Pal methods for microscopical investigation. The meninges appeared to be highly thickened and to consist for the greater part of granulation tissue in which many exudation cells and a marked hyperæmia pointed to the inflammatory character of the process. No gummata or endarteritis could be found.

DISCUSSION.

Thus a diagnosis of pachymeningitis hypertrophica had to be made. This affection is extremely rare and very few elaborate descriptions of it are known. Müller¹² says: "Leider sind bisher nur ganz vereinzelte Fälle ätiologisch, klinisch und histologisch mit moderner Methodik untersucht, vor allem mit Hilfe der Lumbalpunktion und der Komplementbindungsreaktion auf Syphilis." As in this case all these methods of investigation have been applied, I believe it may contribute a little to increase of our knowledge of the disease.

I.—ETIOLOGY.

As far as etiology is concerned, it may be taken as an example and proof of the fact that syphilitic infection is by no means essential in the production of the disease. For no signs of this condition were found, either in the history or in the actual clinical state of the patient: Wassermann's reaction in the blood serum was negative, while in a xanthochromic and highly albuminous cerebrospinal fluid it is of no value. In both cases of tumour mentioned above it gave doubtful results. One cannot, therefore, regard as an omission the fact that it was not performed in the fluid in this case. Microscopically no syphilitic alterations could be demonstrated.

Gross⁸ mentions a case of pachymeningitis hypertrophica of tuberculous nature, but he emphasizes the rarity of a localisation only in the meninges without the slightest affection of the vertebræ. Still, tuberculosis is one of the most well-defined etiological factors of the disease known. Rümke and Goudsmit¹⁵ report a case in which the inflammation was also probably due to a tuberculous infection, though no tubercles could be demonstrated in a piece of dura removed at operation. Other factors mentioned by various authors are, according to Cassirer⁶, cold, overstrain, trauma, alcohol; and, according to Babonneix and Voisin¹ alcohol, lead poisoning, rheumatism; but in most cases syphilis seems to be the main cause. It was so, *e.g.*, in a case of Souques, Blamoutier and de Massary¹⁹, which presented a positive Wassermann's reaction in blood serum and cerebrospinal fluid while the patient recovered almost completely after treatment with bismuth preparations. A very remarkable case is reported by Weigeldt²². It concerns a patient who, more than two years after production of lumbar anæsthesia (novocaine) for a pelvis operation, developed a total paraplegia of the legs. He died and the autopsy showed a complete obliteration of the lumbar theca, which had in all respects the appearance of a pachymeningitis hypertrophica. Weigeldt knows of three other patients in whom a lumbar anæsthesia was followed by obliteration of the subdural space and of two in whom an intradural injection of neosalvarsan had the same result. All these patients recovered; the obliteration was determined by means of lumbar puncture. The last two cases are less instructive, for here the syphilis of course may have been the cause of the affection.

From all the various etiological factors which have been demonstrated or supposed we may conclude that irritation, by preference chronic, of the spinal meninges mainly contributes to the beginning of the disease. In our case we have to ascertain if a chronic irritation of any kind can be demonstrated. Other evidence of it than a very busy life, however, is not obtainable. Possibly the great amount of walking that the patient had done contributed somewhat to irritation of the spinal meninges.

II.—SYMPTOMATOLOGY.

The clinical aspect of this case was different from the usual. In most cases the alterations are localised entirely or mainly in the cervical enlargement and this localisation gives rise to rather typical symptoms, which were first described by Charcot and Joffroy. The first stage is one of bilateral root pains and paræsthesiæ in the arms and is followed by a stage of segmental paralysis in which flaccid paralysis of the arms occurs with typical attitudes of the hands (*mains de prédicateur*) because of the affection mainly of the roots of origin of ulnar and median nerves, and finally by a stage of affection of motor tracts, producing a spastic paralysis of the legs. The authors gave the name of pachymeningitis hypertrophica cervicalis to the disease, as no other localisation was known to them. Subsequently it appeared, however, that other localisations did occur. Cassirer⁶, Babonneix and Voisin¹ and Müller¹² mention the possibility of localisation in any part of the spinal cord and they emphasize the difficulty of making the diagnosis when the affection is seated in the dorsal segments. Confusion with tumour is nearly inevitable, especially when no distinct bilateral root pains are present, as in our patient. Westerhuis²³ mentions a case in which by means of 'lipiodol ascendant' and 'lipiodol descendant' he demonstrated an obstruction of the spinal canal the inferior pole of which was seated at the level of the tenth and the upper at the level of the sixth thoracic vertebra, without being able to determine the nature of the process. This was only possible after microscopical investigation of a small piece of tissue removed at operation. The laminectomy did not bring about any improvement.

In our case the existence of an obliterating process involving several segments was also clear enough, and for this reason an operation was thought useless. But before the autopsy the correct diagnosis was not made. We apparently had to deal with an ascending affection: the movement upward of the sensory level and the appearance in the course of the disease of slight pareses of the arms proved this. The hiccough attacks must probably be regarded as a symptom of irritation, the pathological process finally having reached the fourth cervical segment, which innervates the diaphragm. As the fever was of a septic type it seemed to point to an infective disease. It will perhaps be possible to make the diagnosis of a pachymeningitis hypertrophica in the future, when an obliterating process is found that extends over many segments and presents signs of ascending or descending and a temperature course that points to an infection.

Finally, I wish to discuss a peculiarity of the paraplegia. A glance at Fig. 5 will make clear that a total interruption of the spinal cord in the dorsal part was present. The existence therefore of a flaccid paraplegia of the legs without an affection of the anterior horns in the lumbar segments is not surprising. It is on the contrary so common, that it has given rise to the conception of Bastian's so-called law, according to which a flaccid paraplegia necessarily should follow a total interruption of the spinal cord. Brouwer⁴, on the contrary, having shown that during the long continuance of such an interruption the tendon reflexes may come back and spasticity appear, thinks that in those cases which at the beginning seem to follow Bastian's law we are dealing with a process of diaschisis, which after a shorter or longer time will disappear. Several other authors have recorded cases in which a total interruption in the dorsal or cervical segments of the spinal cord was followed by only temporary loss of reflexes. I am inclined to believe, therefore, that in our patient the affection had not lasted long enough for the diaschisis phenomena to disappear, but that, had he lived longer, the tendon reflexes of the legs would have returned.

III.—HISTOLOGY.

Only the histological aspect of the case is left for discussion and for that purpose I shall give a more detailed description of the microscopical preparations than I did when mentioning the results of the autopsy.

In the upper part of the cervical cord the first thing that strikes the eye is the cavity already alluded to. It really appears neither to communicate with the central canal nor to have a wall of its own, but to be surrounded by a zone of necrotic tissue that occupies a part not only of the posterior columns but of the posterior horns as well (Fig. 1). This

Fig. 1. Upper part of cervical cord. Stain: Van Gieson.

tissue consists of hardly recognizable debris with indistinct cell nuclei and here and there a single lymphocyte. Further examination of the section shows that the ganglion cells of the anterior horns are rather well conserved. Here and there in the gray as well as in the white substance some small infiltrations consisting of mononuclear cells are to be seen. The white substance presents a marked degeneration in all the columns, least noticeable in the anterior pyramidal tracts. As to the meninges, they appear to be much thicker than in normal circumstances and in places fill up the whole subdural space. They consist of a vascular and hyperæmic granulation tissue in which numerous fibroblasts, plasma cells and lymphocytes are to be seen. At some points obliterated blood vessels filled with fibroblasts can be distinguished and at others there are small hæmorrhages. Layers of older connective tissue can in some places be made out (Fig. 2).

Fig. 2. Part of the granulation mass in the meninges of about the same section as fig. 1. Stain: hæmatoxylin-eosine.

Fig. 3. Inferior part of cervical cord. Stain: Weigert-Pal.

Lower in the cervical cord the necrosis round the cavity is less extensive, though as distinct as in the former sections. The cell nuclei have disappeared almost entirely, an occasional small round nucleus being seen amid the tissue debris. Especially in the neighbourhood of the necrotic region small infiltrations are visible, consisting of mononuclear cells (Fig. 4). Here, too, the white substance presents a considerable degeneration

Fig 4. Infiltration of mononuclear cells in the neighbourhood of the necrotic tissue in the same part as fig. 3. In the right upper corner of the figure a part of the necrotic tissue surrounding the cavity. Stain : haematoxylin-eosine.

in all the columns (see Fig. 3). The meninges are less thickened at this level, but apart from that have quite the same aspect as in higher sections. The same granulation tissue with fibroblasts, plasma cells and lymphocytes is found here, and also a strip of older connective tissue at one or two places.

The middle segments of the thoracic region present the greatest destruction possible (Fig. 5). In the sections of this region nothing can be distinguished of the proper spinal cord substance; it is reduced to a necrotic and indefinable mass of tissue fragments, much of which has fallen out during the preparation of the section. The meninges are greatly thickened, their internal layer is necrotic and hardly distinguishable from the spinal cord substance, but gradually the granulation tissue becomes recognizable in the outer layers and it appears to be of the same nature as in sections at a higher level. Hyperaemia, fibroblasts and lymphocytes are present here as well, and strips of older connective tissue are not missing. The external layer of the section consists of membranous connective tissue tightly matted together with the granulation mass in the subdural space.

The lumbar part of the spinal cord appears to be destroyed to a much less extent (Fig. 6). It looks much like what was found in the cervical segments. The same cavity in the posterior columns can be distinguished, filled with necrotic debris and without the well defined limits to the zone of necrosis that in the cervical cord were seen to surround it. Here and there a small infiltration of mononuclear cells is found (Fig. 7). The ganglion cells of the anterior horns are perfectly well conserved. Degeneration is present in

Fig. 5. Thoracic section of the spinal cord. Stain : Van Gieson.

Fig. 6. Lumbar section of the spinal cord. Stain : Van Gieson.

Fig. 7. Mononuclear infiltrations in the lumbar segments. Stain: Van Gieson.

all the columns. The meninges have the same aspect as in the other sections. Though not as much thickened, they consist of quite the same granulation tissue, interrupted in some places by a strip of older connective tissue.

IV.—CAVITY-FORMATION.

The description of the microscopical preparations may have made clear that though a general resemblance to syringomyelia may be found in the existence of a lengthy cavity almost throughout the whole extent of the cord, it can be distinguished from genuine syringomyelia very well. For in our case the cavitation does not originate from a primary gliosis arising in the region of the central canal, as is usually the case in ordinary syringomyelia, but takes its origin in decay of tissue, as may be concluded from the zone of necrosis that is found to surround it everywhere and from the fact that it is filled with necrotic fragments in the lumbar segments. The occurrence of cavities in cases of pachymeningitis hypertrophica is mentioned by several authors and they all ascribe it to necrosis. But the cause of the necrosis is not easy to determine. It is not probable that propagation of the inflammatory process from the meninges to the spinal cord substance should be made responsible for it, for, in the first place, we cannot understand why the inflammatory spread should be confined in the greater part of the cord to a small region almost in the middle of the section; we should expect it on the contrary in a marginal zone. And in the second place the cavity and its surroundings present very few signs of inflammation: except for some small infiltrations here and there, only uncomplicated necrosis can be found. It seems much more probable that the necrosis must be ascribed to want of blood supply and that it is of ischaemic nature. In the meningeal granuloma many obliterated blood vessels are present, so it is not surprising that the blood supply of the cord has become

deficient. According to Pitres and Testut¹⁴ the cavity lies exactly in the region of the terminal ramification of the posterior peripheral artery, which is a branch of the perimedullary arterial circle found in each segment. I admit it is not immediately comprehensible why in every segment the same region must have become ischæmic, since each has its own arterial supply. Perhaps the consideration that the region of the terminal ramification of the posterior peripheral artery is one of the parts farthest from the supplying arterial circle may contribute to the explanation of the facts. For when by the blocking of afferent and perhaps also of efferent blood vessels the blood-pressure in the supplying arterial circle is lowered, those parts will necessarily first be deprived of blood in which the arterial pressure has been lowest from the beginning. Now the lowest arterial pressure is found in the arteries which are most remote from the supplying branch.

Concerning the point of origin of the inflammation, recent authors differ from Charcot and Joffroy. The latter thought that the affection originated in the dura mater, but Müller¹², Cassirer⁶ and Babonneix and Voisin¹ suggest that in many cases it arises in the leptomeninges or even in the superficial layers of the cord itself and spreads only secondarily to the dura mater. From study of our preparations we may reach the same conclusion, for in all the sections the greatest anatomical changes were found between the dura and the cord, whereas the dura itself, as well as the cord substance, shows distinct signs of inflammation only in the thoracic region. Most of the changes in the spinal cord are due to necrosis as a consequence of deficient blood supply.

V.—SYNDROME OF FROIN.

Since this case, as well as the cases of tumour mentioned at the beginning of the paper, showed xanthochromia and massive coagulation of the cerebrospinal fluid, it may be of interest to conclude with a short discussion of the significance of this syndrome. Froin thought it was a reliable sign for the diagnosis of blockage, but it has also occurred in patients in whom no process obliterating spinal passages has been found. De Sanctis¹⁶ mentions the occurrence of the syndrome, for instance, in various kinds of meningitis. I saw it myself in a young man who had abruptly become ill, presenting the aspect of an acute catatonia, but after three days neck-rigidity and Kernig's sign developed. A lumbar puncture was made: the cerebrospinal fluid was intensely yellow and coagulated within 15 minutes. It did not contain red blood corpuscles and only 3 white blood corpuscles per cubic mm. The globulin reactions were strongly positive. The next day he died; at the autopsy the existence of tuberculous meningitis was demonstrated.

Though not confined to cases of spinal block, the syndrome of Froin is very rare in other conditions. Its diagnostic value is therefore considerable, if in cases of block it occurs with great frequency. But it is exactly on this point that the opinions of various authors differ. Dandy⁷ saw xanthochromia

only once in a series of 36 cases of tumour, Gross⁸ three times in 21 patients with spinal block, one of whom only presented massive coagulation of the cerebrospinal fluid at the same time. In agreement with most German authors he thinks the syndrome very rare and therefore of much less value than Nonne's syndrome—a large amount of globulin coupled with a normal or subnormal number of cells. He found it 13 times in his series. Bingel^{2, 3} however, in three cases of tumour found xanthochromia and massive coagulation in one and in two a perfectly normal fluid. In a case of caries with spinal block he found xanthochromia but no coagulation, while the globulin reaction was feeble. French authors on the contrary find the xanthochromia syndrome much more frequently. Guillain, Alajouanine, Mathieu and Bertrand⁹ in a case of cauda perithelioma; Guillain, Alajouanine, Périssin and Petit-Dutaillis¹⁰ in a case of fibrogloma of the eleventh dorsal segment mention its occurrence, and curiously enough they found in both cases xanthochromic cerebrospinal fluid above the tumour, though in the last case it was present below the growth as well. Souques, Blamoutier and De Massary¹⁹ found the syndrome in pachymeningitis hypertrophica. Babonneix and Voisin¹ say in this last disease it is nearly always present. In Dutch literature xanthochromia is mentioned in three cases of tumour, described by Brouwer and Oljenick⁵, by Pameyer¹³ and by Von Ziegenweidt²⁵, but in Pameyer's case it was not combined with massive coagulation. In six cases described by Dutch authors (another case of Brouwer and Oljenick⁵, De Vries²¹, Stenvers²⁰, Pameyer's second case¹³ and two cases of Sillevius Smitt and Bok^{17, 18}) the syndrome was entirely absent. Nonne's syndrome was present in four of these cases and in Westerhuis' case of pachymeningitis hypertrophica²³. But in two cases of tumour the cerebrospinal fluid was almost normal. Together with personal cases I have collected the following data relating to cerebrospinal fluid syndromes in spinal block patients.

Nonne's syndrome	18 cases.
Xanthochromia	15 cases.
Normal or almost normal fluid	4 cases.

I consider that xanthochromia is frequent enough to have considerable diagnostic value, although Nonne's syndrome seems to be a little more common.

I wish here to point out that according to De Sanctis¹⁶ the value of xanthochromia remains the same whether massive coagulation is present or not, for when absent the latter usually appears when one drop of blood serum is added. The coagulation is due to a certain amount of fibrin and it cannot appear when no fibrin ferment is present. By means of a drop of serum this ferment is added and at once the fluid coagulates.

The yellow colour must not be ascribed to hæmoglobin, since this substance is often missing in xanthochromic fluid, but to lutein, the same colouring matter that causes the yellowish tinge of blood serum: it can always be demonstrated in the cerebrospinal fluid when xanthochromia is present. It has been found in all our patients. As to the cause of the syndrome Babonneix and Voisin¹

think it must be sought in the lowered pressure of the fluid below the block. This causes a local oedema and transudation of blood plasma into the dural theca. I believe, however, this theory is incorrect, as the xanthochromia may be found above the obstruction and in cases where no spinal block is present at all. It is more reasonable to make irritation of the meninges responsible for it, as a result of which they become more permeable for components (fibrin and lutein) of the blood plasma.

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