ДРУЖЕСТВО НА НЕВРОХИРУРЗИТЕ В БЪЛГАРИЯ
BULGARIAN SOCIETY OF NEUROSURGERY

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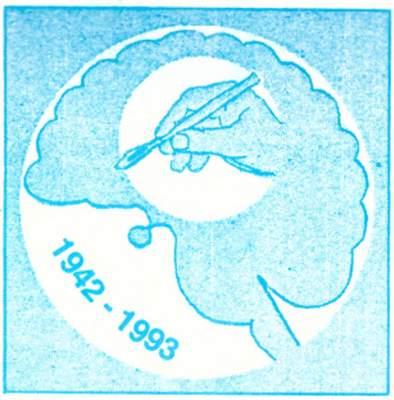

BULGARIAN NEUROSURGERY

1/93

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—ГРАФИК—

CONTENTS

УВАЖАЕМИ КОЛЕГИ,

Преодолявайки много трудности ние поставяме началото на първото у нас научно периодично издание по неврохирургия. Убедени сме, че това ще бъде значителен принос за развитието на неврохирургията в България в теоретичен, организационен и клинико-практически аспект.

Поставяме си за цел да бъдат публикувани материали, третиращи актуални и важни проблеми на неврохирургията, завършени научни изследвания в същата област, казуистки и литературни обзори. Ще се постараем също така да Ви информираме за проведени и предстоящи научни форуми по неврохирургия у нас и в чужбина.

Идеята ни е изданието "Българска неврохирургия" да излиза в две книжки годишно не само у нас, но и в чужбина. Именно за това амбицията ни е занапред статиите да се публикуват едновременно на български и английски език и да бъдат достъпни за по-широка читателска аудитория. Стремежът ни ще бъде към високо научно ниво, което ще постигнем като предявяваме високи изисквания към авторите, които ще предоставят научните си трудове за отпечатване.

Редакционната колегия ще приветствува най-горещо сътрудничеството на всички български автори за участие както с научните си трудове, така и с активна финансова подкрепа чрез спонсориране, за да може това издание да продължи традицията на нашата неврохирургична клиника и да се утвърди като съвременно неврохирургично четиво с високо научно съдържание.

От редакционната колегия

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PINEAL REGION TUMORS – CHALLENGES AND PROBLEMS OF TREATMENT

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A number of challenges and controversies are the concomitants of each step in the development of the diagnosis and treatment of pineal region tumors. For more than 80 years these problems have been widely discussed in medical literature; however, the challenges and part of controversies persist even to this day. The basic challenge is connected with the location of these tumors in the center of the brain and the approach to them, as well as their proximity to structures of great functional importance. Of no less importance is the fact that we still do not know everything about the functions of the pineal gland or about the so-called "pineal organ".

In a historical view, R. Descartes considered the pineal gland as the seat of the soul; the Indian philosophers viewed it as the center of vital energy and supreme spiritual forse. The pineal gland was also considered as the "third eye" (42, 48).

Our knowledge today accounts for the possibilities of the pineal gland to translate photic, sensory and neural information into a neuroindocrine response. More than 30 components have thus so far been isolated from the gland. There is growing evidence that among this components there is one of low molecular weight and a marked antitumor effect (10, 67).

The controversies started from the very beginning when C. Howell (39) presented a paper to the Royal Society of Medicine on February 24 the 1910 on tumors of the pineal gland including autopsy findings from three patients, two of whom were operated thought the posterior fossa. Sir V. Horsley (38) took part in the discussion and he reported: "With regard... to the possibility of doing anything surgically" – he was bound to confess that the surgical results so far, were far from, favorable. He thought that this might be due to the fact that he had approached the lesion subtentorially. He suggested that all future operations for the removal of these tumors, should be supratentorial.

The various controversies could be best understood in the context of the changes in neurosurgical opinions in the different periods. The classical scheme "clinic-investigations – treatment" had undergone different changes depending on the achievements of medical and technical scienses.

It is well known fact that neurological symptoms are nonspecific and that they can rarely determine, with certainty, the exact topic of the lesion. That was the reason why many patients (prior to the introduction of modern diagnostic methods) were operated with a wrong preliminary diagnosis. (1, 5, 37).

With the introduction of different diagnostic methods (ventriculography, angiography, brain scintigraphy, CT and MRI), possibilities were created for determining exactly the topic of the lesion, what kind of lesion, interrelations with surrounding structures, the degree of vascularisation, and its relationships with the deep venous system. Sometimes there is a possibility to determine even the morphological nature of the lesion. Unfortunately the exact histological diagnosis of the tumor is not always possible to determine (4, 15, 17, 29, 30)

The therapeutic attitude towards these tumors has created the greatest challenges, controversies and problems. What was the response of neurosurgeons to these challenges? (Tab. 1 and 2)

Table 1

CLINICAL SIGNS AND SYMPTOMS
SUSPECTED PINEAL LESIONS

↓

INVESTIGATIONS

↓

NEURORADIOLOGICAL

↓

NEUROENDOCRINOLOGICAL

↓

MARKERS

↙ ↘

LESION **NO LESION**

Table 2

Kind of lesions in the pineal region

PINEAL REGION LESION(WHAT KIND OF LESION)	
1.TUMOR	5.A V M
2.CYST	6.ANEURYSM
3.PARASITE	7.HAEMATOMA
4.GRANULOMA	8.OTHERS

BENIGN (10-50 %) ; MALIGN (50-90 %)

The first steps were to create and introduce different surgical approaches: F. Krause (44) and Brunner and H. Rorschach (12) proposed infratentorial-supracerebellar; posterior transcallsosal introduced by W. Dandy (20) in 1921 which has since been extensively described by O. Foerster (28). In 1931 Van Wagenen (65) described a transventricular approach. F. Heppner (32) and later J. Poppen (56) introduced an occipital-transtentorial approach. G. Horrax (36) applied occipital resection for these tumors. In 1910 L. Pussep (57) attacked a pineal tumor

trough a suboccipital flap and by transsecting the transverse sinus and the tentorium.

The results of the first attempts to remove directly pineal region tumors, however, were desperate. W. Russell and E. Sachs (59) in 1943 reviewed the 32 patients whose pineal tumors are treated by direct attack. They found that there were only 3 survivors in the immediate postoperative period which amounts a to a more then 90% mortality rate.

Suffice it to mention the words of the most eminent pioneers of neurosurgery. H. Cushing (19) in 1932 stated that he "never had succeeded in exposing a pineal tumor sufficienly well to justify an attempt to remove it". W. Dandy (21) in 1936 wrote: "Although an operative approach to the pineal region was proposed by me in 1921, it was not until a decade later (1931) that the first pineal tumor was sucsessfully extirpated. A disastrous toll of seven fatal issues during this long period seemed to indicate the futility of further efforts".

Even more pessimistic were the words of L. Davidoff (1967), (22): "The direct surgical attack on these tumors was in the past and still is, by and large, a harmful procedure"... and "the radical surgical treatment of tumors originating in the pineal region after 50 years more or less of bold aggressive surgeons to extirpate them is even to this day dismal by unsuccessful. "

The high operative mortality and morbidity, as well as the high percentage of malign tumors in this region – more than 75% prompted most authors to advocate surgical conservatism and direct surgery was replaced by drainage procedure and blind radiotherapy. This method of treatment has been associated with an overall mortality of less than 5% and a 5-year survival rate of 60% to 75% (7, 14, 23, 26, 33). Surgery was reserved to those tumors that failed to respond to radiotherapy.

Despite the better results od conservative treatment, a number of autors (50, 53, 55, 58, 63) opposed this type of treatment:

- Because they considered that the percentage of benign and radio- resistant tumors is not so negligible – (30% 50%).
- Because often malign tumors are surprisingly well encapsulated, permitting total gross excision.
- Because a course of blind radiotherapy may lead to serious side effects and a harmful effect on the developing brain, as it is well that more than 50% of this tumors occur under age of 20.
- Because radiotherapy for malign, quickly growing pineal tumors, is more effective after total gross or partial removal.
- Because surgery after radiotherapy is difficult and hazardous.

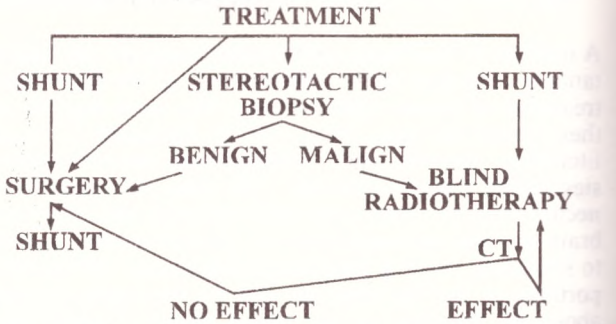
Practical experience and these discussions showed that in the choice of a correct therapeutic approach – surgery or radiotherapy – of key importance is our preliminary knowledge about the nature of the tumor. Experience shows the lack of reliable criteria to make an accurate histological diagnosis on the basis of neuroradiological studies, even when applying the most up-to-date brain imaging techniques.

It is worth remembering that pineal region tumors are rare – 0. 4 – 1. 5 % of all intracranial tumors but in the pediatric popultion their frequency is considerably higher and varies between 3% and 13% (2, 27, 31, 35, 51, 52, 58). They are a small group but they exhibit an exceptional variety which is confirmed by author's data – more than 30 histologically different tumors. Thus for example, M. Edwards et al. (27) has established 17 different types of tumors out of 36 cases. A number of authors (23, 26, 34) emphasize the high frequency (over 50%) of germ – cell tumors; others (49, 50, 53, 66) have observed them very seldom. In our material of 30 verified tumors, the germ-cell tumors are only 4 (13, 3 %).

Another factor that should be remembered is that in this region, besides tumors, there is a wide variety of vascular pathology – AVM, aneurysms, arterio-venous malformation of the great vein of Galen, cysts of different kinds, parasites, granulomas, hematomas, etc. (3, 6, 13, 46, 47, 66, 67). (Table 3).

Table 3

Scheme of different possibilities to treatment of pineal region tumors



Because of these great variety, some neurosurgeons have advocated stereotactic biopsy in order to predict the nature of the tumor and the type of therapy to be used. At the same time they showed that the stereotactic method can also be used also as a means of treatment – aspiration of the cyst or introducing radioactive substance in the tumor for interstitial radiotherapy (9, 18, 25, 43). However, the controversies persist here too. Many authors think that the stereotactic technique carries the risk of hemorrhage because of the close proximity of many and large veins. Besides, the pathological nature of the whole tumor which is very often of mixed histology is difficult to estimate with a stereotactic biopsy because of the inadequacy of the tissue volume (a small piece of only 1 or 2 mm 3 of tissue may be taken). Stereotactic biopsy also carries the risk of dissemination (16, 50, 54).

Thank to modern technical achievements – the opreration microscope, the microsurgical instrumentarium and the recent development of anesthesiology and intensive care, neurosurgeons have been able to approach pineal tumors with great deal of safety and with excellent results.

With the help of microsurgery in 1971, B. Stein (61) and K. Jamieson (40) revived the subtentorial supracerebellar and occipital transtentorial approaches.

It was introduction of microsurgery that marked the beginning of a new stage – that of direct surgery of these tumors.

A number of authors (11, 41, 50, 55, 62) started operating pineal tumors directly with zero or very low operative mortality. And it was now that the ideas of the pioneers in neurosurgery were implemented and their efforts were crowned with success.

Different criteria have been established for the application of one approach or another. These criteria are based on the location of the tumor as regards the tentorium and the deep venous system. However, we think that experience and personal preferences are instrumental in the choice of the ap-

proach. It is well known that some authors (8, 11, 45, 62) operate pineal tumors by applying just one approach. Despite the different preferences, the approaches most widely applied today, are indoubtedly the occipital transtentorial and the subtentorial supracerebellar ones. Other approaches besides these mentioned, were also introduced, such as the lateral subtentorial supracerebellar (64), subchoroid in the tumors in the medial and posterior parts of the third ventricle (24), combined sub – and supratentorial (60) and parieto – occipital approach (8).

Bulgarian neurosurgery followed all these changes in the strategy to these tumors. Until 1981 our attidude was mainly conservative – radiotherapy, and in the cases of obstructive hydrocephalus – drainage (ventriculo-cisternostomy) or shunt operation.

After 1981 we changed our policy. Microsurgery was applied in 30 patients. In 26 of them we applied occipital or parieto – occipital transtentorial approach, and in 4 posterior transcallosal. In 11 patients we achieved total or gross total removal, in 13 partial resection, and in 6 only biopsy. In the postoperative period 4 patients died (13, 3%) (Tables 4 and 5).

Table 4

Histological verification on 30 pineal region tumors

PINEAL TUMORS (HISTOLOGICAL VERIFICATION ON 30 TUMORS)			
I. TUMORS OF PINEAL PARENCHIMAL CELL ORIGIN	-		7
1.PINEOCYTOMA	-	4	
2.PINEOBLASTOMA	-	3	
II. TUMORS OF GERM-CELL ORIGIN	-		4
1.GERMINOMAS	-	2	
2.EMBRIONAL CARCINOMA	-	1	
3.TERATOMA	–	1	
III.OTHER CELL ORIGIN	-		14
1.ASTROCYTOMA	-	9	
2.OLIGODENDROGLIOMA	-	1	
3.MENINGIOMA	-	4	
IV.CYSTS	-		5
1.ARACHNOID	-	3	
2.EPIDERMOID	-	1	
3.EPENDIMAL	-	1	
TOTAL	-		30

In conclusion I can say that the recent advances in diagnosis, intensive care and microsurgical techniques were the key factors which have changed our strategy. Today, the direct approach to pineal region tumors is a method of choice allowing for total or maximal resection of the tumor, a highly reliable histological diagnosis and the possibility to apply the most appropriate complex of treatment with a view to radio – and chemotherapy.

Table 4-A

VASCULAR MALFORMATION IN THE PRINEAL REGION

ARTERIOVENOUS MALFORMATIONS	-	5
AVM OF GREAT VEIN OF GALEN	-	5
ANEURISM OF PCA	-	1

Table 5

TYPE OF SURGERY IN 30 PINEAL REGION TUMORS

TYPE OF SURGERY

TOTAL OR GROSS TOTAL REMOVAL	-	11
PARTIAL REMOVAL	-	13
BIOPSY	-	6
PREOPERATIVE SHUNT	-	15
POSTOPERATIVE SHUNT	-	4

Through part of the problems and controversies have been solved, the challenges are still there because they are the challehges to be met by an exceptionally difficult surgery.

REFERENCES

1. Бурлуцкий, А.П., Опухоли шишковидной железы. Медгиз, М. 1962
2. Габровски Ст. в кн. Тумори на централната нервна система в детска възраст (ред. П. Вецка), М.Ф., С. 1987, с. 88-96.
3. Габровски Ст. II Нац. конгрес рентгенол, радиол., радиобиол. 1-3 юни 1990, Смолян . Резюмета с. 95.
4. Коновалов, А.Н. Ж. Вопр. Нейрохир, 47, 1983, 2:3-11.
5. Корнянский, Г.П., Вопр. Нейрохир. 13, 1940, 1:32-44.
6. Сальман, А.Я. Вопр. Нейрохир., 13, 1949, 1: 30:37.
7. Abay, E. O. et al. J. Neurosurg., 55, 1981, 6: 889-895.
8. Ausman, J. I. et al.: In surgery in and around the Brain Stem and the Third Ventricle (Ed. M. Samii), Springer Verlag, Berlin, 1986, pp. 326-328.
9. Backlund, E.-O.: Adv. Tech. Stand. Neurosurg., Vol. 6., 1979: 13-19.
10. Bartch, H. et al.: Oncology, 49, 1992, I: 27-30.
11. Brotchi, J. et al.: Acta Neurochir., suppl. 53, 1991: 144-147.
12. Brunner, C., H. Rorschach: Cor. Blatt Schweiz. Arste, 1911, may: 642-643.
13. Burres, K., R.Hamilton: Neurosurgery, 4, 1979, 2: 264-268.
14. Camins, M. B., E. B. Schlesinger: Acta Neurochir., 40, 1978, 3-4: 131-143.
15. Chang, T. et al.: AJNR, 10, 1989, 5: 1039-1044.
16. Chang, C. G. et al.: Neurosurgery, 8, 1981, 4: 656-668.
17. Clarke, J. C.: Brit. J. Radiol., 61, 1988, 6: 953-955.
18. Conway, L. W.: J. Neurosurg., 38, 1973; 3:453-360.
19. Cushing, H.: Intracranial Tumors. C.C. Thomas, Springfield, 1932
20. Dandy, W.: Surg. Gyn. Obstet., 53, 1921, 2: 113-119.
21. Dandy, W.: Arch. Surg., 33, 1936; 1:19-46.
22. Davidoff, L. M., Bull. N. Y. Acad. Med, 43, 1967, 4: 537-561.
23. DeGirolami, U., H. Schmidek: J. Neurosurg., 39, 1973, 4: 455-462.
24. Delandsheer, J. M. et al.: Neurochirurgie, 24, 1978, 6: 419-422.
25. Dempsey, P. K., et al.: Acta Neurochir., 116, 1992, 1-2: 14-22.
26. Donat, J. F. et al.: Arch. Neurol., 35: 1978; 11:736-740.
27. Edwards, M. S. B. et al.: J. Neurosurg., 68, 1988; 5:689-697.
28. Foerster. O.: Wien klin. Wschr., 41, 1928: 986-990.
29. Futrell, N. at al.: AJNR, 2, 1981, 5: 415-420.
30. Ganti, S. R., et al.: AJNR, 7, 1986, 1: 97-104.
31. Graf, N. et al.: Med. Welt, 40, 1989, 8: 778-785.
32. Heppner, F.: Zbl. Neurochir., 19, 1959, 3: 219-224.
33. Hitchon, P. W. et al.: Neurosurgery, 13, 1983, 3: 248-253.
34. Hoffman, H. J. et al.: Concepts pediat. Neurosurg., vol. 4, 1983, pp. 360-386.
35. Hoffman, H. J.: Progr. exp. Tumor Res., vol. 30, 1987, pp. 281-288.
36. Horrax, G.,: Arch. Neurol. Psychiat., 37, 1937, 2: 385-397.
37. Horrax, G.,: Arch. Neurol. Psychiat., 64, 1950, 2: 227-242.
38. Horsley, V.: Proc. R. Soc. Med., 3, 1910, Febr.: 77.
39. Howell, C.M.H.: Proc. R. Soc. Med., 3, 1910, Febr. 65-77.
40. Jamieson, K. G.: J. Neurosurg., 35, 1971, 5: 550-553.
41. Jooma, R., B. E. Kendall: J. Neurosurg., 58, 1983, 5: 654-665.
42. Kappers, J. A.: In The Pineal Organ (eds.: A. Oksche, P. Pévet), Elsevier, Amsterdam, 1981, pp. 3-21.
43. Kitchen, N. et al.: Brit. J. Neurosurg., 6, 1992, 3: 265-269.
44. Krause, F.: Chirurgie des Gehirns und Ruckenmarks, Urban und Scvhwarzenberg, Berlin, 1911.
45. Lapras. C.: In Diagnosis and treatement of pineal region tumors (Ed.: E. A. Neuwelt), Williams & Wilkins Balt., 1984, pp. 289-299.
46. La Torre, E. et al.: J. Neurosurg., 49, 1978, 5: 610-613.
47. Miyake, S. et al.: Surg. Neurol., 37, 1992, 4: 293-299.
48. Moreau, J. J. et al.: Anat. Clin., 7, 1985, 1:3-13.
49. Moringlane, J. R. et al.: Adv. Neurosurg., vol. II, 1983: 270-281.
50. Neuwelt, E. A., H. H. Batjer: Contemp. Neurosurg., 4, 1982, 1:1-5.
51. Packer, R. J. et al.: Pediatrics, 74, 1981, 1: 97-102.
52. Pecker, J. et al.: Surg. Neurol., 12, 1979, 4: 341-348.
53. Pendl, G.: Pineal and Midbrain Lessions. Springer-Verlag, Wien, 1985.
54. Peragut, J. C. et al.: Neurochirurgie, 33, 1987, 1: 23-27.
55. Pluchino, F. at al.: Acta Neurochir., 96, 1989, 1-2: 26-31.
56. Poppen, J. L.: J. Neurosurg., 25, 1966, 6: 706-710.
57. Pussep, L.: Neurol Zentralblat., 33, 1914, 560-563.
58. Raimondi, A. J., Tomita: Child's Brain, 9, 1982, 4: 239-266.
59. Russell, W. O., E. Sachs: Arch. Path., 35, 1943, 869-888.
60. Sekhar, L. N., A. Goel: Surg, Neurol., 37, 1992, 3: 197-201.
61. Stein, B. M.: J. Neurosurg., 35, 1971, 2: 197-202.
62. Stein, B. M.: In: Surgery of the Third Ventricle (Ed.: M. L. J. Apuzzo), Williams & Wilkins, Balt., 1987, pp. 570-590.
63. Todo, T. et al.: Neurosurgery, 29, 1991, 1: 101-105.
64. Van der Bergh, R,: Clin. Neurol. Neurosurg., 92, 1990, 4: 311-316.
65. Van Wagenen, W. P.: Surg. Gynec. Obstet., 53, 1931, July-Dec.:216-220.
66. Vorcapic, R. ,G. Pendl: Neuropediatrics, 18, 1987, 4: 222-226.
67. Wetterberg, L.: In Pineal Organ (Eds.: A. Oksche, P. Pevet), Elsevier, Amsterdam, 1981, pp. 345-354.

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SURGICAL REMOVAL OF A FALX-OSTEOMA IN CASES OF PERSISTING HEADACHE. (PRELIMINARY REPORT)

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The calcifications of the falx and the falx-osteomas are discussed as two consecutive stages of a single process. Some authors even didn't make difference between both of them (3). In fact, this condition is relatively frequent and is demonstrated as a calcium salts sedimentation. Considerably rarely, dural bone metaplasis occurs, leading to neoformation of bone tissue, formed by compacta and spongiosa. This condition is considered as "falx-osteoma". On X-ray film calcifications of the falx or falx-osteomas are observed in 20 % of people at over 50 years of age. In a study of almost 3000 skull films of patients of all ages, was observed that the calcifications of the falx cerebri is present in 7 % of cases (7). In 6 % of children over 3 years of age a falx calcification is observed (4). At CT-scan, these are found in nearly half of the cases over 20 years of age, due to the increased sensitivity of the examination method.

The reason for these formations is not clear as well as factors leading to bone metaplasis. They are more frequent by metabolic diseases, leading to hypocalcaemia. However, not in all cases with calcifications such a disease is found. Some authors refer it to an atavistic mark (some maritime mammalians normally have stuff plate, separating the two hemispheres – seals, walruses (1, 2). Others suppose disembryogenetic reasons – residual embryonic cells from the primary mesenchima – Russel et al. (6). Conforming to the latter, is also the publication of Bakaert 1983, who reports about intracranial osteomas, located intracerebrally without any connection with the dura mater. Some authors consider the falx-osteoma as a rare tumor of the falx, like the chondroma and the osteochondrosarcoma. (5).

In falx-osteomas, the clinical condition is symptomless, but in rare cases it can be demonstrated itself as a volume process. There are single reports that they can cause irritating changes of vessel or nerve character.

The falx itself is innervated by the three main branches of the fifth cranial nerve. The ophthalmic nerve innervates the basal areas of the falx, near inferior saggital sinus by small dural branches, in its anterior parts and by rami tentorii in the posterior parts. Before leaving the cranial cavity, the maxillary nerve gives rise to a little branch – ramus meningeus medius – which innervates the upper parts of the falx. Extracranially, before dividing into sensory and motor branches a small ramus rises from the mandibullar nerve. It enters the cranial cavity by the foramen spinosum together with the middle meningeal artery and innervates the upper, middle parts of the falx. The areas innervated by the falcian branches rising from the maxillary and mandibular nerves are overlaying each other.

CLINICAL CASES:

We present two cases of patients with persistent headache in which we have removed osteomas of the falx.

V. A. V (Nr. 114/88). Female 38 years old, complained from a severe headache, localized in the two temples, which after one or two hours from the beginning involves the head as a whole. In cases of physical or mental efforts, the pain increases. There was no connection between the pain and the menses. Subsequently the headache was followed by nausea, vomiting, face paleness, sweating under arms, mouth dryness, tension in the ears, chewing muscles stiffening, neck rigidity and photophobia. Home treatment with analgetics, corticosteroids, vasoactive drugs and acupuncture was without effect. Since 2 – 3 months the pain was unbearable. She was enable to work and sleep. The pain was so severe, that she began to express suicide ideas. For this reason she was permanently under family control. The somatic and neurologic status was normal. X-ray and CT scan investigations of the head showed a left sided calcification in the anterior part of the falx. Bilateral carotid angiography was without pathological changes. The patient was operated by left frontal craniotomy. Penetrating between the left hemisphere and the falx a total osteoma with dimensions 5 X 3 centimeters was removed. The fixation point of this formation on the dura mater was cross cut from front to the back and from down to up between the superior and inferior saggital sinuses (having in mind the falcine nerve fibers directions). The histological result: Nr. 51/1988 " Component bone formed mainly by spongious bone tissue ".

On the next day after the operation, the headache sharply subsided and on the third completely disappeared. The patient was observed for a period of 8 months. She was in a very good general condition and without headaches. After that the contacts were interrupted.

B. D. P. (Nr. 461/92) Female 38 years old, complained for eight years of headaches, primarily in the frontoparietal region. For the last 3 – 4 years the headaches had increased mainly with diffuse character; It appeared 3 – 4 times monthly, with a duration of 3 – 5 – 6 hours. When it occurred during the night, she couldn't sleep, she had nausea, sweating, eyes tension. The attacks sometimes were unbearable, up to "getting mad". During the last 6 months she was unable to work. She didn't dare to travel in the country and be absent from home for a longer period. The headaches are without connection with the menses or other outside factors. The somatic and neurologic examinations showed no deviations. Blood and urine examinations were normal. Brain mapping with low amplitude E. E. G. – "Intermittent focus of delta waves bifrontaly". X-ray and CT-scan of the head demonstrated a 6 X 3 centimeters falx osteoma in the frontoparietal regions occupying the both sides of the falx. Bilateral carotid angiography was normal.

The patient was operated by biparietal access, mainly in the right. The falx-osteoma was totally removed together with the fixation spot to the falx. Histological result Nr. 274/92 – Pieces of component bone (osteoma). Postoperatively a light left sided hemiparesis was observed, which disappeared after a week. The headaches sharply decreased. Control examination after 5 and 7 months – the patient declares that herself confidence is normal.

She is fully active in everyday life, travels by herself outside the town. Once or twice monthly after physical or mental overload an extremely light diffuse headache was appeared. This one certainly was not of the same kind as preoperatively and was influenced favorably by walks or by a tablet of Analgin.

DISCUSSION:

This surgical intervention for stubborn and strong headache was applied for the first time in our neurosurgical activity. We can not mantain only on two cases for the connection and character of these headaches in patients with falx-osteomas. Even though by the cases, described there is a coincidence of enough clinical symptoms.

We can't discuss the long term results from such surgical intervention, only on two cases. It is necessary to observe a greater number of patients. The paper has a preliminary character.

REFERENCES

1. Aschoff.: Pathol Anatomie.v.2 Nerven-system./ P.Ernst. 1923
2. Klumair, J.: Ueber die Haufigkeit und Bedeutung des Falx-Knochen.Radiol.Austria,16,1966,221-22
3. Kopilov,M.B.: Basics of the x-ray diagnostics of the diseases of the brain. M.Medizina, 1968
4. Manelfe,C. et al: Tumeurs sous-tentorieles de la ligne mediane. Dans "Traite de Radiodiagnostic" tome 14-1, p. 252
5. Osonoff,M.B. and Burrows,E.H.: Intracranial calcification of the brain – in "Radiology of the scull and brain" Newton and Potts eds. Chapter 41 pp 828 – 829
6. Russel,D.S. and L.J. Rubinstein: Pathology of brain tumors of the nervous system.Baltimore 1972,Williams & Wilkins eds.
7. Taveras,J.M. and E.H. Wood – Diagnostic Radiology 2nd ed., Williams & Wilkins Cc,Baltimore, 1976.

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DISTAL ANEURYSMS OT THE ANTERIOR CEREBRAL AND MIDDLE CEREBRAL ARTERIES

Report of 5 cases and literature survey

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The aneurysms located distally to the anterior communicating artery (AcoA) are usually designated as distal aneurysms (DA) of the anterior cerebral artery (ACA). They are further subdivided to: infracallosal (located at the area of emergence of frontpollar artery), DA of pericallosal-callosomarginal branching point (or at the genu of corpus callosum), and DA or ACA sensu structu at the branching points of anterior, middle and posterior internal frontal arteries, paracentral and parietooccipital arteries. The DA of the middle cerebral artery (MCA) are located at the secondary or/and tertiary bifurcations or trifurcations as well as more distal branching points.

The DA of ACA accounts for 2% to 5% in the intracranial aneurysms in most surgical series (5, 8, 9, 11, 16, 25, 27, 28). The percentage of DA of MCA seems to be mentioned by Yasargil and Smith (1982) only.

Most of DA of ACA are associated with multiplicity (9, 16, 25, 26, 28), and anomalies and variants of ACA and its branches. The natural history of these lessions is complicated by poor outcome (4, 15), and defferent surgical approaches have been utilized for their management (16, 26).

The DA of ACA and MCA are infrequent lesions and any contribution to their clinical presentation, surgical anatomy and management is considered by us to be enough relevant to be reported.

CLINICAL MATERIAL AND METHODS.

Five patients admitted in the Dept. Neurosurgery (Med. Academy between 1989-1991) and having DA or ACA and MCA are presented. The clinical presentation operative treatment and results are shown on table 1 and table 2.

DISCUSSION

The rate of DA of ACA is between 2,3% (26) and 9,2% (16), the most frequently reported figures being between 3% and 6% (11, 23, 25). Our findings are similar – 5%.

In most of the surgical series both sexes are equally represented (8). All our patients are female, which is most probably due to the limited number of patients included in our study. Several autors have emphasized that 20% to 50% of patients having DA of ACA have another aneurysms (16). The highest incidence of multiplicity is shown by Yasargil (1984) – 52,2% and Wisoff and Flamm (1987) – 55%. This fact makes the complete angiografic investigation mandatory. Most of the patients with DA of ACA have their first clinical manifestation at 5-th or 6-th decade of life (8). Our patients suffered their SAH earlier. This might be explained by the limited number of cases and the tendency to increase the age limit for angiography and surgery in Japan, USA and Western Europe. The most common location of the DA of the ACA is at the bifurcation of ACA into pericallosal and callosomarginal branches – 71% according to Kuwabara et al. (1984). The same autors found that the infracallosal aneurysms represent 15% of 191 cases that were included in their study. It has been repeatedly emphasized that the DA of the ACA are frequently associated with anomalies of the ACA (16, 23, 25). Details about the normal anatomy anomalies and their clinical and surgical significance can be found in comprehensive studies of Critchley (1930), Baptista (1963), and Perlmutter and Rhoton (1978). The most common variants associated with DA of ACA are azygos artery, distal AcoA or supreme AcoA, the pericallosal artery supplying both hemispheres and e.t.c. (7, 16). In one of our cases the aneurysm was based on supreme AcoA, and in the remaining cases the ACA were of uneqal diameters and with bihemispheric branches of pericallosal and/or callosomarginal arteries. The nonsurgically treated cases have dismal prognosis (4, 12, 15). The SAH caused

by the rupture of these aneurysms is often associated with intracerebral hematomas, leading to destruction of the commissural fibres at the genu of corpus callosum and resulting in severe morbidity. According to Fankhauser and Zander (1978) 22% of the patients with ruptured DA of ACA have contralaternal hemiparesis, 10% weakness of the contralateral leg, homolateral leg weakness in 8%, 8% were admitted in comatous state and 5% have akinetic mutism. Transient of permanent psychoorganic syndrome is a relatively frequent complication of the ruptured DA of ACA (11). One of our patients had signs of mild psychorganic syndrome.

The most frequently used surgical approaches to the DA of ACA are:

1. The pterional (frontotemporosphenoidal) approach recommended by Yasargil (1984);
2. The right subfrontal approach advocated by Laitinen and Snellman (1960);
3. The right frontal parasagittal approach (11, 16, 25).

In 2 of our cases Yasargil's pterional approach was used, which enables the surgeon to have early controle of both A2 segments of ACA. However it requires prolonged dissection in the interhemisheric fissure and more retraction on the frontobasal areas. The same is apparently valid for the subfrontal approach. The parasagigittal interhemispheric approach requires small frontal craniotomy and more limited dissection into the interhemispheric fissure, after creating of at least 2-3 cm space between the bridging veins draining into the SSS. If one frees the veins from arachnoid for 2 – 2,5 cm the retraction on the frontomedial areas is very slight and there is no need to sacrifice the veins. However the dissection into the interhemispheric fissure is difficult due to the arachnoid reaction to the SAH. Moreover the surgeon has to approach the aneurysm before having identified the afferent and efferent branches, or to try to dissect them into the immediate vicinity of the neck. It is advisable to try to identify first the frontopolar artery or another branch on the medial surface of frontal lobe and to follow this until the trunk of ACA is reached. It is our experience that one should not abandon the dissection of already identified branch when it penetrates the depth of the sulcus since this might lead to confusion about the identity and direction of branches. After having identified the proximal to the aneurysm ACA one has to free the neck from the arachnoid adhesions. At this stage the surgeon faces specific problems. The neck has to be freed from loops of the efferent arteries (pericallosal and callosomarginal and their branches) forming sometimes complex network. Additional difficulties might be created by atherosclerotic plaques at the neck that might involve the main branches. Wisoff and Flamm (1987) have emphasized the main sources of error during dissection are misenterpretation of the right ACA for left or vice versa, misidentification of the callosomarginal artery as pericallosal and misconception of the distal and proximal directions. Once the proximal controle over the ACA is insured a possible rupture is easy to put under controle. Snykers and Drake (1973) observations on permanent occlusion of the callosomarginal and/or pericallosal artery have led them to conclude that in most of cases the resulting neurologic deficit is transient.

The outcome from surgery is related to the preoperative condition of patients (16, 19, 25, 26, 30) and is generally favorable without mortality and severe disability. However patients with severe preoperative neurological dificit, decreased level of consciusness, associated medical problems, CT-data for parenchymal hematomas, massive subarachnoid blood accumulations, ischemic areas in the brain have incomplete neurological recovery. There is still some controversy on the timing of the surgery. Sindou et al. (1988) advocate delayed surgery, where as Ohno et al. (1990) have demonstrated good results following early surgery and after carefull selection of the patients suffering SAH. All of the patients reported by us were in grade I or II according to Hunt and Hess grading scale, and this point can not be discused.

In one of the presented cases there was rupture during dissection around the neck and temporary clip had to be applied on the ACA. This patient had transient postoperative psychorganic syndrome (confusion, disorientation, memory loss), contralaternal hemiparesis, but they resolved within two weeks. Another patient suffered transient increase of the preexisting psychorganic syndrome which might be explained by distrurbances of venous outflow, and/or retraction of the medial frontal lobe since no temporary clipping was used.

Some of aneurysms of the MCA are traumatic or mycotic. In our patient (10, 22, 23) there was no history of trauma or infectious disease. Traumatic aneurysms represent pseudosac resulting from laceration of the arterial wall and transformation of the periarterial hematoma into aneurysm. Most probably they are the result of rapid acceleration deceleration forces leading to injury of the ACA on the free edge of the falx.

Traumatic aneurysm have no necks unlike berry aneurysms which frequently requires parent vessel occlusion and end anastomosis. Moreover they have typical time of clinical manifestation (10, 22, 23).

The DA of MCA are rare. According to Yasargil and Smith (1982) and Yasargil (1984), they represent about 5% of the MCA aneurysms. In the large series of Suzuki et al. (1984) and Pasztor et al. (1986) this particular location has not been discussed. One can not draw conclusions from a single observation but nevertheless some points deserve to be mentioned. The dissection of the secondary and tertiary branches of the MCA should be started from the MI segment. If the surgeon begins the dissection from insulooper-cular segments of MCA there is a possibility of misinterpretation of the identity and direction of the individual arteries due to their complex and tortuous course. Moreover misconception of the anatomy might be created by the arachnoid reaction after SAH, which makes the dissection difficult and requires coagulation of Sylvian vein tributaries to decrease the retraction of the opercular areas. Some of the DA of the MCA are bacterial (2% to 6,7%) according to some reports (13, 24), and 3% of the endocarditis are complicated by aneurysms. Moreover they are as a rule multiple, and their rupture lead to intracerebral hematomas. They usually respond to antibiotic treatment (13, 23, 24).

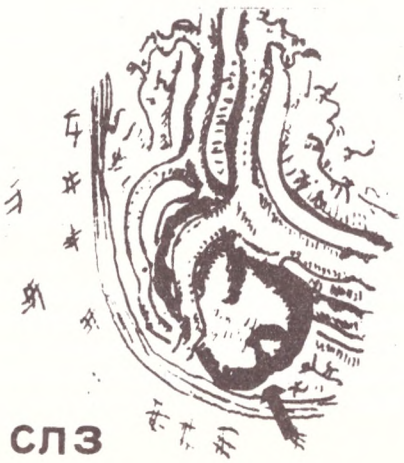
It might be concluded that the surgical treatment of DA requires perfect knowledge of the surgical anatomy and identification of the parent vessels in the early stages of dissection in order to minimize the retraction and avoid sacrifice of important vessels.

Table 1

NAME	AGE	SEX	SAH	STAGE (Hunt& Hess) OPERATIVE APPROACH	EARLY POSTOPE-RATIVE PERIOD	CT	LATE POSTOP-ERATIVE PE-RIOD	
N.B.S.	30	Female	1	I	pterional transsylvian	without neurological complications	-	-
T.I.T.	36	Female	2	II	parasagitt. inter-hemispheric	with complica-tions	postoper. ischemia in left internal capsule	without neurologi-cal deficit
R.I.I.	43	Female	2	I	pterional inter-hemispheric	without compi-cations	ischaemia in right frontal area	without neurologi-cal deficit
K.M.V.	44	Female	1	I	subfrontal	without compli-cations		
P.C.S.	51	Female	1	II	pterional inter-hemispheric	with transient neurological deficit	small ischaemia in left frontal area	transient psychor-ganic syn-drome

Table 2

FIGS. 1 TO 5 DEMONSTRATE THE SURGICAL FINDINGS. THE ARROWS POINT TO THE ANEURYSMS



REFERENCES

1. Baptista, A. G.: Neurology, 13: 825-835 (1963).
2. Becker, D. H., Newton, T. H.: Neurosurgery, 4: 495-503 (1979).
3. Critchley, M.: Brain, 53: 120-165 (1930).
4. Dandy, W. E.: Intracranial arterial aneurysms. New York Hafner 1969 (cit. K. S. Mann et al., 1984).
5. Fankhauser, H., Zander, E.: Schweiz. Arch. Neurol. Neurochir. Psychiat., 123: 331-357 (1978).
6. Gibo, H., Carver, C. C., Rhoton, A. L. Jr., Lenkey, C., Mitchell, R. J.: J. Neurosurg., 54: 151-169 (1981).
7. Huber, P., Braun, J., Hirschmann, D., Agyeman, J.F.: Neuroradiology, 19: 143-147 (1980).
8. Kuwabara, S., Ishikawa, S., Andoh, S., Matsumoto, S., Sekimoto, H., Kemura, Y, Yamane, K., Takahashi, M.: Neurol. Med. Chir., 24: 580-590 (1984).
9. Laitinen, L., Suellman, A.: J. Neurosurg., 17: 447-458 (1960).
10. Laun, A.: Unfallheilkunde, 81: 482-491 (1978).
11. Mann, K. S., Yue, C. P., Wong, G. et al.: Surg. Neurol., 21: 261-266 (1984).
12. Mc Kissock, W., Paine, K. W. E., Walsh, L. S.: J. Neurosurg., 17: 762-776 (1960).
13. Morawetz, R. B., Karp, R. B.,: Neurosurgery 15: 43-49 (1984).
14. Nakstad, P., Norues, H., Hauge, H. N.: Neuroradiology, 28: 335-338 (1986).
15. Nichioka, H.: J. Neurosurg., 25: 574-592 (1966).
16. Ono, K., Monma, S., Suzuki, R., Masaoka, H., Matsushima, Y., Hirakawa, K.: Neurosurgery, 27: 907-913 (1990).
17. Pasztor, E., Vaida, J., Juhasz, J., Toth, Sz. Orosz, E., Horvath, M.: Acta Neurochir., 82: 92-101 (1986).
18. Perlimutter, D., Rhoton, A. L.: J. Neurosurg., 49: 204-228 (1978).
19. Sindou, M., Pelissou-Guyotat, I., Mertens, P., Keravel, Y., Althayde, A. A.,: Surg. Neurol. 30: 434-440 (1988).
20. Suzuki, J., Yoshimoto, T., Kayama, T.: Neurosurg., 61: 17-23 (1991).
21. Snyckers, F. D., Drake, C. G.: S. Afr. Med. J., 47: 1787-1791 (1973).
22. Tsubokawa, T., Kotans, A., Sugawara, T., Moriasu, N.: Neurol. Surg., 3: 663-672 (1975).
23. Weir, B.: Aneurysms affecting the nervous system. Williams & Wilkins, Baltimore (1987) pp 159-174.
24. Weir, B.: Special Aneurysms (Nonsaccular and Saccular). Infective. In: Cerebral aneurysms (ed. B. Weir) pp. 159-172. Williams & Wilkins, Baltimore (1987).
25. Wisoff, J. H., Flamm E. S.: Neurosurgery, 20: 735-741 (1987).
26. Yasargil, M. G.: Surgery of intracranial aneurysms and results. In: Microsurgery, Vol. II. Clinical Considerations Stuttgart, Georg Thieme Verlag, pp. 224-231 (1884).
27. Yasargil, M. G., Smith, R. D.: Management of aneurysms of anterior circulation by intracranial procedures. In: Youmans J. R. (ed): Neurological Surgery, Vol. 3; pp. 1663-1696; W. B. Saunders.
28. Yasargil, M. G., Carter, L. P.: J. Neurosurg., 40: 218-223 (1974).
29. Yokota, A., Tazaki, H., Murayama, K., Shimura, T., Higushi, H., Yagima, K., Nakazawa, S.: Neurol. Surg., 11: 521-528 (1983).
30. Yoshimoto, T., Ushida, K., Suzuki, J.: J. Neurosurg., 50: 40-44 (1979).

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POSSIBILITIES FOR THE IMMUNOCYTOCHEMICAL DIAGNOSIS IN PRIMARY CEREBRAL TUMORS

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The exact classification of cerebral tumors and the definition of the degree of anaplasia are a prerequisite for the optimal therapy (6). For that purpose the routine staining methods are complemental during the last 10 years with immunohistochemical methods (ICHM). As a result of the latter tumor antigenes, individual components of the cytoplasm and secretory products of the tumor cells are evaluated and certain features of the separate cell type may be defined.

In this study our aim was to examine the value of some ICHM: – GFAP, S-100 proteins for more detailed information on the differentiation of astroglial tumors, and the pluripotentials of certain blastoms (medulloblastoma). We want to share our experience with the use of ICHM for some diagnostic problems, concerning the histogenesis of tumors.

MATERIAL AND METHODS

The biopsy speciments of 25 cases of astrocytomas gr. I-III, oligoastrocytoma, multiform glioma, medulloblastoma, ependymoblastoma, astrocytoma with Rosenthal changes of the neurofibrils etc. were used in the study. The specimens were prepared on paraffin blocks of materials fixed on 10 % neutral formalin. The ICHM for GFAP, S- 100 protein and cytokeratin according to the classic reaction (PAP), described by Sternberger in 1979 (8) were applied.

RESULTS

The study on the group of astrocytomas gr. I-III and some mixed tumors (oligodendroastrocytoma) showed that tumors of low malignancy (gr. I-II) had more positive reaction for GFAP. Here are astrocytomas with Rosenthal changes of the gliofibrils with this note that tumor cells without Rosenthal change are strongly positive. Rosenthal

cells themselves were most frequently negative or slightly positive (Table 1, cases 1 – 7). Next to it is a group of 4 cases with astrocytomas gr. II and even gr. II-III, where the reaction is very positive. The 6 cases of astroglial tumors with marked anaplasia resembling glioblastoma have different results. The two cases of ganglioglioma and ependymoblastoma are with positive reaction for GFAP (this finding for ependymoblastoma is reported by others too (7)).

The medulloblastoma (2 cases) showed bipolar differentiation. Groups of cells with positive GFAP reaction wich sometimes are clearly seen on Hematoxilin-Eosin staining as astrocytes or astroblasts, are found, as well as S- 100 protein positive cells which showed potential for neuronal differentiation. The cells with positive GFAP reaction usually surround the vessels and that confirms their connection with the astrocytes. In case Nr. 20 the vessel walls in some parts of the tumor were positive too. The bipolar differentiation of the tumor in case Nr. 21 was helpful for the correction of the first diagnosis of malignant astrocytoma.

In the last 2 cases on the Table 1 the ICHM were very helpful for the correct diagnosis. In case Nr. 22 the operative finding was of extramedullary tumor (neurinoma) even on the HE staining there was some suspicion of schwanoma (Fig. 1). The positive GFAP reaction in many cells was decisive for astroglial tumor (Fig. 2). In the last case Nr. 23 the clinical suspicion for malignant meningeal tumor as well as the impression of the pathologist for metastasis were cleared by the negative reaction for cytokeratin and the very positive reaction for GFAP (Fig. 3).

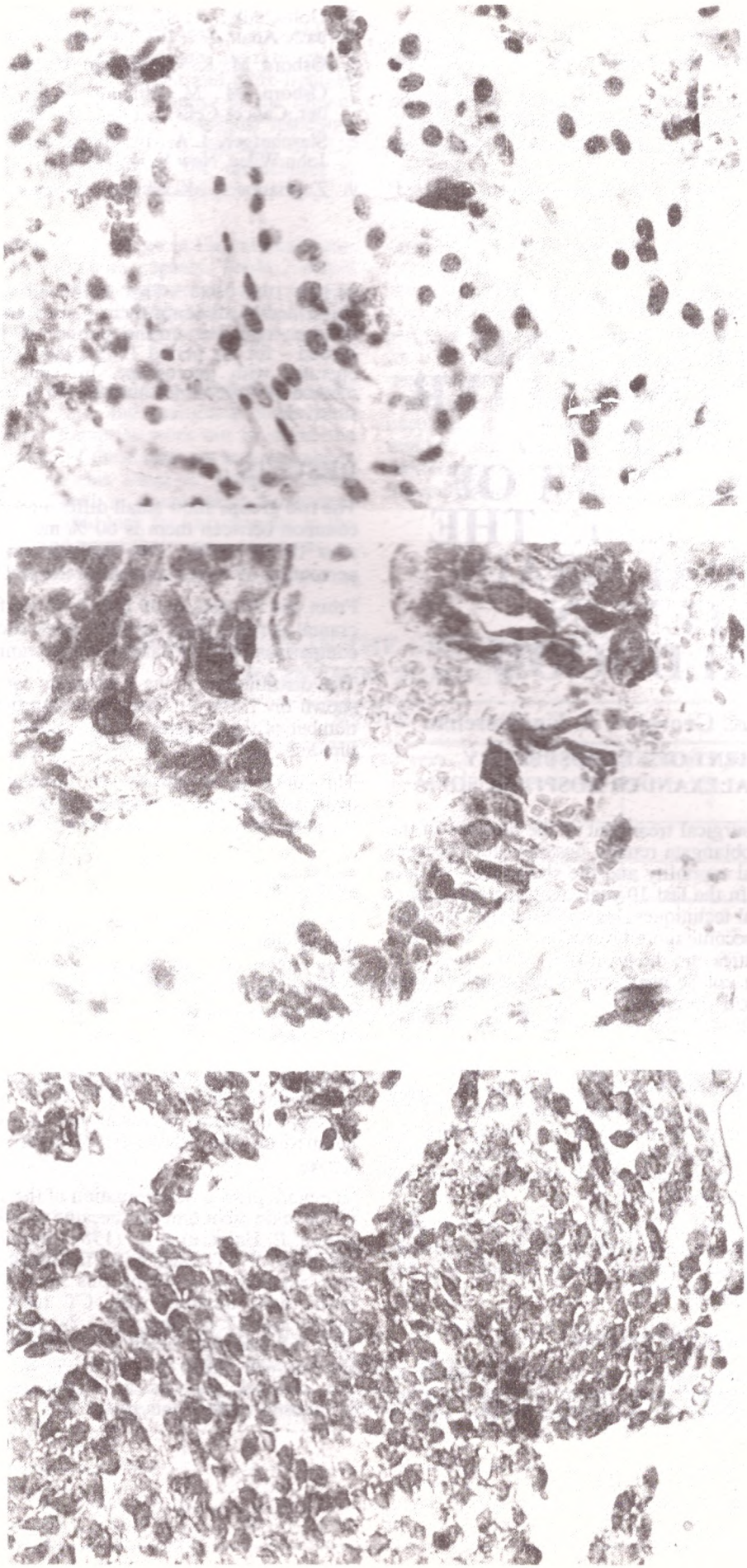
DISCUSSION

It is well known that the subjective assessment of the malignancy of a tumor is not always precise and is in direct dependence on the professional competence of the pathologist. We had the chance to be convinced by our participation in the study, organised in 1980 – 1982 by Zaprianov (2) for the definition of the maturity and type of a series of cerebral tumors (mostly astroglial). The results of the diagnosis of 7 well trained and with rich experince pathologists, made independently one of the other, were quite different. During the last decade most authors report that the astroglial tumors examined by them showed positive reaction for GFAP independent of the features of anaplasia (3, 6). Later it was stressed on the fact that with the increase of tumor anaplasia the number of positive cells is decreased (1). These statements are in agreement with the first 7 cases of primary cerebral tumors examined by us. In the next group of 4 cases (NN 8 – 11) the great number of marked cells may be the reason than they were diagnosed as more malignant that they were in reality. Many authors state that GFAP is expressed predominantly in the giant cells of the multiform glioma, but as far as the malignancy of these tumors is related to the small cell component, it may be expected that more malignant course will have cases with more negative reaction and not the opposite (5).

Our study confirms the opinion of other authors that nowadays it is very difficult to give prognosis on the biological behavior of astroglial tumors on the basis of the reaction for GFAP only (1). But as far as GFAP is relatively specific marker for glial cells it may be helpful in many cases for solving the histogenetic difficulties in the diagnosis of primary cerebral tumors.

Table 1

Case N	Pathomorphologic diagnosis	GRAF	S-100protein	Cytoker
1. 338/8	Oligodendroastrocytoma gr. II-III	+		
2. 365/88	Oligodendroastrocytoma gr. II			
3. 284/88	Astrocytoma with Rosenthal gr. I	+		
4. 381/88	Oligodendroastrocytoma gr. I	++		
5. 416/88	Astrocytoma with Rosenthal gr.I	++		
6. 385/88	Oligodendroastrocytoma gr. II	+++		
7. 282/88	Astrocytoma with Rosenthal gr. I	+++		
8. 379/88	Astrocytoma gr. II-III	++		
9. 280/88	Astrocytoma gr. II	++		
10. 306/88	Astrocytoma gr. II	+++		
11. 306/88	Astrocytoma gr. I	+		
12. 322/88	Glioblastoma multiforme	+		
13. 368/88	Glioblastoma	+		
14. 399/88	Glioblastoma	++		
15. 278/88	Multiform glioblastoma	+		
16. 267/88	Multiform glioblastoma with meningial invasion	++		
17. 263/88	Glioblastoma	+++		
18. 229/88	Ganglioglioma	++		
19. 273/88	Ependymoblastoma	++		
20. 253/88	Medulloblastoma	++	+	
21. 593/88	Medulloblastoma (Astrocytoma?	+	++	
22. 530/88	Astrocytoma gr. I (Neurinoma?)+++	/-/		
23. 10/91	Glioblastoma (meningioma? or metastasis?/	+++		/-/



REFERENCES

1. Gottschalk,I., W. Enisch: Arch. Patologii 3,1987,3-11. (Russian)
2. Milenkov, Chr., R. Raytchev, I. Peeva et al.: Scientific Session NVMI Plovdiv, 1982,p. 6 (Bulgarian)
3. Bignami, A., D. Dahl, D.C. Rueger : Adv. Cell Neurobiol 1, 1980, 285-291.
4. Clark, H.B., J. Minesky, D. Agrawal et al.: Amer. J. Pathol., 121, 1985, 96-101.
5. Gottschalk,I., H. Martin, H. Barz et al.: Zbl. allg. Path. path. Anat., 131, 1986, 43-48.
6. Osborn, M., K. Weber: Lab. Invest., 48, 1983, 372-394
7. Osborn, M., M. Altmannsberger, E. Debus, K. Weber,:Cancer Cells, 1, 1984, 192-200
8. Sternberger, L.A.: In: Immunocytochemistry, 2nd ed., John Wiley, New York, 1979
9. Zaprianov, Z., K. Christov: Cytometry, 9, 1988, 380-386

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RESULTS AND PROGNOSIS AFTER SURGICAL TREATMENT OF TUMOURS IN THE CAUDAL BRAIN STEM IN CHILDHOOD

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The results of the surgical treatment of the tumours in the pons and medulla oblangata remain unsatisfactory because of the high surgical mortality and the short terms of survival. (2, 4, 6, 10) In the last 10 years with the application of the microsurgical techniques, laser and ultrasound aspiration, the results become more favourable (1, 3, 4, 8). Despite all the difficulties and the great risks of the surgery of the brain stem can not be overcome. The majority of the cases are with a bad prognosis and die at an average 2 years after the diagnostication of the tumour and only 20 – 35 % survived more years (4, 10). Comparatively favourable in the prognosis are the exofytic brain stem tumours, low grade astrocytomas as well as the cases without an early and fast progressive neurological deficit. On the contrary, the survival period is reduced with the intrabrain stem, histologically malignant tumours which are charactarized with early signs of neurological deficit and hypodense CT image (1, 6, 7, 9).

With this report our aim is to analyse our surgical experience and to emphasize the most significant prognostic factors influencing the evolution of the tumour and the possibility for a longer survival.

MATERIAL AND METHODS

The object of our investigation are 57 children (up to 14 years old) with caudal brain stem tumours, treated surgically in the Clinic of Neurosurgery, Sofia, for the period 1980-1989. The follow-up of our cases was performed by dividing them in two groups: the first – 22 children operated in the period of 1980-1989 and the second – for the period 1985-1989 – 35 cases, as 22 were operated with microscope. In some cases we used the method of "Life table" analysis for the follow-up of 5-year survival.

RESULTS

The two groups show small differences in the survival. The common between them is 60 % mortality in the first year after the operation. Our analysis shows a little greater percentage of 5-year servival in the second group (table 1).

From our 57 cases in 30 (50. 5 %) we found paresis of the cranial nerves. Their survival rates show worse results in comparison with the cases without cranial nerve paresis.

The distribution of the operations for the two groups is shown on Table 2. There is a significant increase of the number of the biopsies in the second group of brain stem tumours.

The follow-up of the 5-year survival in the cases with exofytic and cystic astrocytomas of the brain stem shows the more favourable prognosis of these cases (Table 3).

DISCUSSION

The caudal brain stem tumours have a more peculiar place among the tmours of the Central Nervous System in children. They are not homogeneous in reference to the clinical, histological and CT characteristics. This is the reason for the differences in the results of the treatment and prognosis. Some authors as A. Albright (1986) attribute a prognostic value to the early appearance of partial paralysis and paralysis of cranial nerves as an unfavourable factor. In 50. 5 % of our cases we found paralysis of cranial nerves as well as a decrease survival in comparison with the cases without paralysis of cranial nerves.

The more precise determination of the size and localization of the brain stem tumours became possible with CT investigation. E. Halperin et al. (1989) emphasize the hypodense character of CT image as an unfavourable prognostic sign. Our experience confirms this opinion. From 47 caudal brain stem tumours with CT investigations, 21 were hypodense. 12 of them (57.1 %) have died up to 1 year after the operation while in the other group (26 cases) in the first year only 6 (23 %) have died. These differences in the survival give us a certain reason to believe in prognostic value of this sign.

The most common operation in the caudal brain stem tumours is the biopsy. In the literature there are rare reports for a subtotal extirpation for the tumour and more frequent for an evacuation of a cyst with a biopsy of the tumour node. It is admitted that the volume of surgical resections is connected with the new operative methods as: CT controled stereotaxic manipulations, the application of laser, the ultrasound aspirator and evoked potentials (1, 3, 4, 5).

Whit the years a considerable change in the surgical treatment of the brain stem tumours appears. Table 2 shows that with the application of microsurgery in our practice the number of biopsies is considerably increased. It is all too obvious the difference in our surgical activity in the two groups brain stem tumours.

Our experience shows that there are differences in the survival between the benign and malignant brain stem tumours. Many authors attempt to work out in detail the histological characteristics of these tumours and to evaluate their prognostic value. In this sense E. Halperin et al. /1989/ find a worse survival in anaplastic astrocytomas while A. Albright et al. /1986/ look for karyokinesic in the histology of the tumour and prove their worse prognosis.

Our observations are that the better survival is with the histologically proven astrocytomas with an exofytic growth penetrating in LV ventricle, as well as with cystic astrocytomas (Table 3). In the cases with cystic astrocytomas we have evacuated the cyst with a biopsy of the solid part of the tumour. With the accumulation of the experience, microsurgery gives us the opportunity to enlarge the volume of the excision.

In conclusion we consider that the surgical treatment of the caudal brain stem tumours in children, although proved in our practice, has to be defended especially for the biopsy. We consider that the biopsy is nesessery to verify in detail the histological nature of the tumour which is an important prognosticative significance. Besides sometimes looking for a brain stem tumour, we can find an ependymoma or medulloblastoma, parasitic cyst and arterio-venous malformation. We can restrain from the biopsy as an exeption when we are absolutely sure the species of the tumour and the child has to be send directly for radiotherapy. In case of a hydrocephalus we consider that the more longlasting effect is achieved by ventricle shunts.

Table 1

5-YEAR SURVIVAL IN THE GROUPS OF CAUDAL BRAINSTEM TUMOUR

	Survival					
	UP to 1 yer	**UP to 2 yers**	**UP to 3 yers**	**UP to 4 years**	**UP to 5 yers**	**TOTAL**
I-st group	15	4	1	–	2	22
II-nd group	21	9	1	1	3	35
TOTAL	36	13	2	1	5	57

Table 2

DISTRIBUTION OF DIFFERENT OPERATIONS IN CAUDAL BRAINSTEM TUMOURS

	Revision (exploration)	**Revision or biopsy + shunt**	**Biopsy**	**VA shunt**	**Drainage according to Torkildsen**	**TOTAL**
I-st group	10	3	5	4	–	22
II-nd group	14	4	10	6	1	35
TOTAL	24	7	15	10	1	57

Table 3

SURVIVAL FOR 5 YEAR PERIOD IN CYSTYC AND EXOFYTIC ASTROCYTOMAS OF THE BRAINSTEM SURVIVAL

	UP to 1 yer	**UP to 2 yers**	**UP to 3 yers**	**UP to 4 years**	**UP to 5 yers**	**TOTAL**
Cystic astrocytomas 4	4	2	1	-	2	9
Exofytic astrocytomas	9	3	1	1	3	17
TOTAL	13	5	2	1	5	26

REFERENCES

1. Albright, A.L., A.N. Guthkelch, R.J. Parker -et al. J. Neurosurg. 65, 1986, 751-755
2. Coffey R.J., L.D. Lunsford – Neurosurgery, 17, 1985, 12-18
3. Epstein F., – Cancer, 56, 1985, 1804-1806
4. Epstein F., E.L. Melleary – J. Neurosurg. 64, 1986, 11-15
5. Halperin E.C., S.M. Wehn, J.W. Scott et al.- Medical and Pediatric Oncology, 17, 1989, 116-125
6. Hoffman H.J., L. Becker, M.A. Graven -Neurosurgery 7, 1980, 243-248
7. Hoffman H.J., A.R. Stroink, G. Davidson, et al. -Concepts pediat. neurosurg., Basel s. Carger, 7, 1985, 105-116
8. Littman P., P. Jarrett, L.T. Bilaniuk et al. -Cancer, 45, 1980, 2787-2792
9. Packer R.J., R.A. Zimmerman, T., T.G. Luerssen et al. – Neurology, 35, 1985, 397-401
10. Walker M.L., B. Storrs – Concepts in Pediatric Neurosurgery, Basel & Carger, 1985, v. 5, 178-186

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TRAUMATIC SUBDURAL AND EPIDURAL HAEMATOMAS. RESULTS FROM THE SURGICAL TREATMENT

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The problem of the traumatic intracranial-extracerebral haematomes is frequently discussed in the neurosurgical literature (2,4,7,8,11,12,14,24,25).

The aim of this article is to analyze the results of the surgical treatment of these haematomas, making emphasis on the chronic subdural ones (SDH). Furthermore some atypical cases of interest in the clinical practice are presented.

For the period between September 1988 to September 1991 in the Neurosurgical Department(*) were admitted and treated surgically 76 patients with traumatic intracranial-extracerebral haematomas. As shown at Table 1, the greatest number of patients is the group are these ones with chronic SDH. The average age is 60.1 years. The male/female ratio is 60:1 (42 males and 7 females). More that a half of the patients are alcohol-drinkers, which is postulated to be a factor promoting chronic SDH (9,16,19,20,21).

Table 1

LOCALIZATION OF THE HAEMATOMA	TYPE OF HAEMATOMA	NUMBER
EPIDURAL	ACUTE CHRONIC	5 2
SUBDURAL	ACUTE SUBACUTE CHRONIC	6 12 49
COMBINED(**)		2
	TOTAL	76

The standard operative procedure used for the surgical treatment was evacuation trough one or two (9,13,17,18) burr hole craniotomy (1,4,13,17,18). On some of the patients a larger craniotomy was performed for the extirpation of the haematoma capsule. In patients with marked remaining subdural cavity (e.g. insuffitent brain reexpansion), a closed-system drainage was used.

We have tried the technique described by F. Grissoli (6). It consists of intrarachial infusion of Ringer-lactate in an attempt to augmentate the intraoperative cerebral reexpansion. Unfortunately, we could not achieve the excellent results described by the author. Since 1989, we accepted the proposed by T.M. Markwalder (14) 5-graded scale for the evaluation of a patient with chronic SDH. This scale is needed for a quick evaluation of the neurological status of each case at the admittance and following up the course of the disease. Those of the patients who had been operated before we introduced the scale were carefully investigated on the basis of the review of the medical records.

We consider as a main advantage of this scale the possibility of standardizing the data from different cases. This is put in use making comparison between the results from different studies, gathering and proceeding the information.

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(**) *Subdural and Epidural (usually acute)*

Table 2

GRADE	NUMBER OF PATIENTS AT ADMITTANCE		OUTCOME	
			DISCHARGED	DEATHS
0	3	Grade0		
		0	3	–
		I	–	–
		II	–	–
		III	–	–
		IV	–	–
I	3	0	3	–
		I	–	–
		II	–	–
		III	–	–
		IV	–	–
II	34	0	30	
		I	1	
		II	1	2
		III	–	
		IV	–	
III	7	0	6	
		I	–	
		II	–	1
		III	–	
		IV	–	
IV	2	0	2	–
		I	–	–
		II	–	–
		III	–	–
		IV	–	–

It may be concluded from our experience that the grading has no value for the prediction of the outcome (Table 2). This is true because of it's inapplicability in evaluation the somatic and age-related changes in every particular patient.

In one case, after operation a serious infection (meningitis) of the nervous system was observed. The absence of local changes in the operating wound makes up to think that the contamination is due to the chroniosepsis after bronchopneumonia which the patient was suffering preoperatively. A wound infection without nervous system affection is observed in 3 patients. Due to clinical and CT scan data for reoccurrence of the SDH nine of the patients were reoperated, two of them suffered a third one. The mortality rate by surgical intervention was 6.1 % (3 patients). At the postmortem examination, no connection between the lethal exitus and the operation was established. Only in one patient is found to have a residual haematoma without substantial dislocation of the brain structures.

In making CT diagnosis of intracranial haematoma, mistakes or wrong interpretations are possible.

We have a case of a comatose patient admitted at the hospital immediately after the head injury. The early CT examination was negative. Few hours later, the second CT examination demonstrated an large acute SDH.

In another case bilateral subacute isodense SDH were discovered regardless of the two consecutive CT scans with negative results.

Another patient twice underwent an operation for SDH. Due to lack of improvement in the neurological status and available CT data for recurrence, of the haematoma with considerable dislocation, we decided to proceed with large osteoplastic craniotomy. We were surprised by the absence of liquid collection. In fact, a very thick capsule was located, which formation simulated on the CT – scan the image of SDH and caused brain dislocation. After it's total removal an significant improvement of the neurological

status was achieved. The CT examination at the discharge of the patient was negative. In two cases operated for traumatic intracranial-extracerebral haematomas, as an additional intraoperative finding, neoplastic formations were discovered. Similar cases are described by some authors (10,15). The discussion and the review of the literature were presented by us in 1989 elsewhere (5).

The chronification of a haematoma located in the epidural space is quite rare. Moreover the lack of a proper conception about the period, in which an acute epidural haematoma became a chronic one, leads to differences between various authors (3,22,23). We have operated two patients in which the time elapsed after the head trauma exceeded the longest period mentioned in the literature. Intraoperatively in both of them, marks of blood clot organization was found. The cases with a brief review of the literature will be discussed in a separate paper.

Most of the authors claim that patients with congenital arachnoid cyst are at high risk for developing a SDH after even minor head trauma.

A 65 years old male was admitted with comparatively good general condition (II grade according to the accepted scale) with data from the CT scan for SDH. A control CT scan was performed after successful removal of the haematoma and normalization of the neurological status. It shows that the haematoma was totally evacuated, but the pole of the left temporal lobe was still collapsed. According to the typical CT finding, without hystological verification, we accepted that it is the case of traumatic break of the capsule of a congenital arachnoid cyst with the blood vassels in it. As a result a chronic SDH was formed. Catamnestic follow up of the patient, who continues being in good health by persisting of the CT finding withheld us of additional diagnostic manipulations and procedures.

As a conclusion it could be said that our results of the operative treatement do not differ considerably from those, described in the literature. The shown clinical cases were selected because we consider them as being clinically interesting and should be considered in the everyday clinical practice.

REFERENCES

1. Auer, L.M. et al. – J.Neurosurg.,1981, 54 , pp.366-369
2. Bullock,R. et al. – J.Neurosurg.,1990, 72,pp.9-14
3. Clavel, M. et al. – Acta Neurochir., 1984,86, pp. 115-118
4. Filipov F. et al. III Scientific session Stara Zagora, October 5-6, 1989, p.345
5. Gabrovsky,St. et al. – IV National Meeting of Neurology, Pleven, October, 14-15, 1989
6. Grisoli,F. – Neurosurgery, 1988, 23, 616-621
7. Haselberger,K. et al. – Acta Neurochir., 1988, 90, 111-116
8. Iwadate,J. et al. – Neurol.Med.Chir.,1989,29,117-121
9. Iwakima, Tand C.V. Brungraber, – J. Neurosurg., 1973, 38, 488-493
10. Jamjoom,A.B., Cast,I.P. – Brit.J.Neurosurg., 1987, 1(3), pp. 385-388
11. Loew,F. and Kivelitz,R. – in Handbook of Clin. Neurol. 24, part II, 1976, Vinken & Bruin eds.
12. Loew,F. – in Advances and Technical standards in Neurosurgery, 19, 1982, Springer Verlag eds., pp. 113-131
13. Markwalder, T.M. – J. Neurosurg., 1981, 54, pp. 390 – 396
14. Markwalder, T.M. – J. Neurosurg., 1981, 55, pp. 637 – 645
15. McKenzie,C.R. et al. – Neurosurgery, 1990, 27(4),pp. 619-625
16. Munk,P. et al – J. Comp. Ass. Tomography, 1988, 12 (6), pp 1073-1075
17. Niikawa,Sh. et al. – Neurol.Med.Chir., 1990, 30(3), pp. 169-172
18. Page,A. et al. – J. Neurol Neurosurg. Psych. 1987, 50, 1001-1007
19. Richards, T., Hoff, J. Surgery, 1974, 75, pp. 253-258
20. Ryback, R., Desforges, J.J. Arch.Int.Med. 1970, 125, pp. 475-477
21. Sonne,M.N., H. Tonnesen – Brit.J.Neurosurg., 1992, pp. 125-130
22. Sparatio R.R. et al. – Trauma, 1972, 12(5), pp. 435-439
23. Tatagiba,M. et al. – Surg. Neurol., 1989, 32, pp. 453-458
24. Vanev,M. – Scientific papers of "Pirogov" Institute 1988, pp. 3-14
25. Zotov U.V. – Surgery of traumatic intracranial haematomas and brain contusions. L. Medizina, 1984, p. 56

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ABOUT TWO CASES OF CHRONIC EPIDURAL HAEMATOMAS

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The epidural location of a haematoma of traumatic origin is well known and widely discussed elsewhere. As usual these lesions are acute. The so-called "lucid interval" is present in less that one third of the cases (6).

The chronic course of a epidural haematoma (EHD) is quite uncommon. No more than a hundred cases are described in the literature.

According to Chadly (4), initially, Jacobson in 1886, had informed for such a lesion, but the first detailed description was made by Rochet and Logre in 1916 as an autopsy finding. Nearly a hundred years after Tatagiba et al. (18) made a large review of the literature available, summarizing the already described 63 cases adding to them 8 of his own.

We present two cases of chronic EDH, operated in the Department of Neurosurgery(*) .

Case 1 B.A.S.,Nr.6526/1988. A 42 year old male suffered a head injury without loss of consciousness. Immediately after the incident he complained from a light headache, dizziness and vomiting. After two weeks in excellent condition without any complain, a gradually increasing clumsiness of the right hand makes him ask for a medical consultation. He was referred to the Neurosurgical Department with a suspicion for a chronic subdural haematoma (SDH). At admission the patient was alert. The neurological examination revealed a right-sided hemiparesis. No speech disturbances are observed. The CT-scan with contrast enhansment demonstrated a low-density extracerebral haematoma in the temporoparietal region. A high density rim surrounded the collection. A midline shift was present. The patient was operated (at the 20th day after the trauma), with a left osteoclastic craniotomy over the haematoma. A dark brown liquid collection surrounded by a well-developed capsule was found epiduraly. The liquid was aspirated and the capsule adhering the dura was partially removed. After a smooth postoperative period the patient was discharged in good health. He was followed-up five years. He still remains in excellent condition.

Case 2 R.A.K.,Nr.407/1989. A 38 year old male underwent a head injury with skull base fracture. He was admited in a hospital in the country in a soporous state. Lumbar puncture demonstrated traumatic subarachnoid hemorrhage. Gradually the condition of the patient improved, but after a period of two weeks a deterioration of the consciousness was observed. He was transported to the Neurosurgical Department. At admission the patient was in comatose state. Fundoscopy revealed bilateral papilloedema, up to 3 – 4 D. The CT-scan demonstrated a leftsided isodense extracerebral haematoma in the frontotemporal region. A considerable brain compression with a midline shift was present. The investigation was performed without contrast medium. Because of the rapid deterioration, an emergency operation was decided (19 days after injury). A left frontotemporal craniotomy was performed and an epidural haematoma was evacuated. The blood clot showed signs of organization. Granulation tissue forming the haematoma capsule harbored brown liquid. The bleeding source was not located. Postoperatively a marked improvement of the patient's condition was observed and he was discharged after three weeks, completely alert with normal neurological status. In a three year's follow-up he remains in good health without any late sequels of the head injury.

DISCUSSION

Although the temporal criteria for the chronic subdural haematoma are well defined (For an extensive review see Markwalder 1981 (11) it is not the case with the chronic epidural ones. This is probably because this condition is rare. Various authors discuss different criteria about the interval of time between the head injury and the operation, to define an EDH as a chronic one. Some consider as interval a period of 48 – 72 hours prior the operation (13,16); others 6 days (5) and a third group – two weeks (8). Both cases presented above are admitted and operated after an interval of time exceeding the terms given by the literature. Concerning this, we maintain the opinion of that the period after which an EDH could be identified as a chronic one is nearly three weeks. This is probably the time required for clot organization and formation of a capsule.

The incidence rate of the chronic EDH remains unknown due to these differing viewpoints of the authors. The highest rate was reported by Iwakuma (8) – 21 patients with chronic EDH operated 13 days or more after head trauma in a series of 69 cases. In the series of Pozzati et al. (12) the incidence rate was 18 % (from 166 cases) for the patients operated 3 days after the injury. The nine cases reported by Chadly (4), represent 6.75 % of the patients operated 2-3 weeks after the trauma. In our series of 76 patients with intracranial extracerebral haematomas of traumatic origin the EDHs are only seven, including the described ones. We withhold make conclusions about the incidence of the chronic EDH only on two cases.

According to Tatagiba the site of the haematoma seems to be more often in the frontal region (35 %), followed by the temporal (21 %), and the parietal (17 %). Only in one case the clot was situated infratentorially. The predominance of the frontal localization was stressed by other authors (4,7). This is not the opinion of Pozzati (12) who finds the most frequent localization in the temporal region. Some authors consider a close relationship between the haematoma site and the outcome. According to them (9,10), when the chronic EDH is located in the frontal area, the clinical symptoms occur slowly and the disease prognosis is better. On the contrary Tatagiba did not find such a correlation.

The bleeding source is not always easy to determine. Although there are reports for late rebleeding in the haematoma cavity (13), Arutjunov (1) did not accept the opinion of some Russian authors that the chronic EDH is a result of a delayed bleeding in the following days after the trauma. Becker (2), believe that 24 hours after the injury it is almost impossible to find the exact location of the bleeding point. The common view, is that usually the bleeding source is the majority of cases is a low-tension vessel-venous or diploic (3,7,12), but in the series reviewed by

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Tatagiba from 63 patients, bleeding of venous origin was found in 14 cases, of arterial – 12, and in 37, the site was not detected. According to his own observations only in one from eight he was able to find the source. We were not able to find the bleeding point in both of the presented cases.

At the operation, brown liquid and clot organisation are usually found. A developed haematoma capsule is not an obligatory finding. Iwacuma and Brunngraber, believe that a 5 weeks interval is required for it's formation (8). The opinion of Arutjunov (1) is that this period can not be well defined and is related to individual tissue reactions in every particular patient. In our cases, the operative findings and histological studies demonstrated a developped capsule 20 days after the head injury.

The male preponderance, usually in age under 40 is emphasized in all reports (4,18). Two or three weeks ago the patient suffered a various in severity trauma of the head. A skull fracture may be present in some cases (4), but a minor injury, without loss of consciousness may also be the cause of for a chronic EDH (14). The commonly accepted opinion is that in chronic EDH, the surgical removal of the mass lesion leads to an excellent outcome in the majority of cases. An osteoplastic craniotomy is often required for the extirpation of the capsule. Some authors advocate the nonsurgical management, because a spontaneous resolution of the haematoma may occur (15,17).

There is still no clear definition of the chronic EDH. Probably experimental studies are required to demonstrate the clot organization and the formation of the haematoma capsule in the epidural space.

REFERENCES

1. Arutjunov, A.I. – in Guide of Neurotraumatology, Ch.13 "Traumatic intracranial hemorrages and trunciate haematomas": 377-391, M. Medicina 1978
2. Becker,D.P. – Diagnosis and treatment of Head injury in adults – in Neurological Surgery 2nd.ed. Vol.4, J.R. Youmans ed.,W.B.Saunders co: 2030
3. Benoit B.G., et al. – Epidural Haematoma: – Can.J.Neurosci.;1982,9, 321-324 (cited by Cooper)
4. Chadly,M. – Les hematomes extra-duraux chroniques – These pour Doctorat en Medecine – Acad.D'Orleans-Tours ; Juin 1973
5. Clavel,M. et al. – Chronic Epidural Haematomas. Acta Neurochir.,1984. 86,(2) ; 115-118
6. Cooper,P. – In Head Injury – Williams & Wilkins Co. 1987 – Ch.13 – Posttraumatic intracranial Mass Lesions : 285-312
7. Hirch,L.F. – Neurosurgery, 1980,6,518-522
8. Iwacuma Tand, C.V. Brunngraber – J.Neurosurgery, 1973, 38: 488-493
9. Jamiesson,K.J. – Epidural Haematoma – In Vinken and Bruyn eds. Handbook of Clin.Neurol.Injuries of the brain and skull. Part II. Vol.24, Amsterdam-Oxford. 1976, 261-274
10. Jamiesson,K.J. and Yeland,J.D.N.- J.Neurosurg. 1868, 29, 13-23
11. Markwalder,T.M. – J.Neurosurg.(54) May 1981, 637-645
12. Pozzati,E. et al. – J.Trauma, 1980, 20, (9), 795-799
13. Pozzati,E. et al. – J. Trauma, 1987; 27(5), 579-780
14. Servadei,F. et al. – Acta Neurochir. (Wien) ; 96, 39-45
15. Shields,C.B. – Neurosurgery, 1980,6: 512
16. Sparatio,R.R. et al. – J. Trauma, 1972,12, (5) : 435-439
17. Tochio,H. et al. – Neurosurgery, 1984,; 15(1) : 96-100
18. Tatagiba,M. et al. – Surg.Neurol. 1989; 32, 453-458

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MICROSURGERY OF THE OCULOMOTOR NERVE IN CRANIOBASAL LESIONS

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Lesions of the oculomotor nerve are among the most frequent before and following surgery of craniobasal tumors, aneurysms, malformations or traumatic injuries (1, 5, 6, 7). H. Egger and J. Gilsbach (1985) point out that oculomotor nerve lesions were observed in 15, 7% of cases with tumors of the tentorial edge and that postoperative paresis of the nerve was the most common complication of cranial nerve surgery.

Despite their frequency lesions of the oculomotor nerve especially following various surgical procedures are scarcely studied and many details concerning their nature, prevention, treatment and prognosis remain obcure. The aim of the present study was to evaluate the type, degree and evolution of oculomotor lesions following extensive surgery of the craniocerebral base.

Surgical anatomy: The oculomotor nuclear complex is a collection of cell columns lying ventrally to the central gray in the midline at the level of the red nucleus and innervating all the extraocular muscles (exept the lateral rectus and superior oblique), the levator palpebrae muscle and providing preganglionic parasympathetic fibers to the ciliary ganglion (4). The root fibers arising from the oculomotor nucleus pass ventrally some medial to, some traversing the red nucleus and emerge in the interpeduncular fossa on the anterior aspect of the midbrain. Here the nerve fibers pass below the posterior cerebral artery and above the superior cerebellar artery, run in the crural and carotid cisterns and penetrate the transversal plate of the cavernous sinus. The entry point in the cavernous sinus is lateral to the posterior clinoid process (from 3 to 13 mm, 7, 4 mm on average), medially to the anterior petroclinoid plica and dorsocaudally to the anterior clinoid process (3-11 mm, on average 7, 5 mm). The nerve courses in the lateral wall of the cavernous sinus above the trochlear and ophthalmic nerves and through the superior orbital fissure it branches to the extraocular muscles and cilliary ganglion.

Lesions of the oculomotor nerve may occur in the nuclear complex, in the intracerebral root fibers, in the cisternal part of the nerve trunk, in the cavernous segment and in the orbit. Here only lesions of the cisternal part of the oculomotor nerve are discussed although in some cases it is very difficult to differentiate with lesions of the cavernous segment (3,4).

Operative material: Cases treated with microsurgical technique (tumors – extracerebral or intracerebral-extraaxial, aneurysms) where microdissection and some manipulation of the cisternal part of the oculomotor nerve were analysed – Table 1:

Table 1

TYPE OF PATHOLOGY AND FREQUENCY OF OCULOMOTOR LESIONS BEFORE AND AFTER OPERATIVE TREATMENT

Type of pathology	Number of cases	Oculomotor Lesion	
		preoperative	postoperative
I. Tumors of middle fossa	143	29	36
meningioma	74	11	27
metastasis	12	8	5
putuitary adenoma	28	3	1
craniopharyngioma	7	1	1
chordoma	3	2	1
cholesteatoma	3	1	0
temporal glioma	14	2	0
Gasserian neuroma	2	1	1
II. Vascular diseases	17	8	5
basilar aneurysms	3	0	2
ACommPost aneurysms	11	7	2
Temporal cavernoma	3	1	1

Oculomotor lesions (ranging from slight diplopia to complete paralysis – ophthalmoplegia oculomotoria) were found most frequently in metastatic tumors and aneurysms of the posterior communicating artery. The operative intervention with excision of the pathologic process and decompression of the nerve was with non-predictable effect on the oculomotor function – with the exception of cases where complete anatomic transection of the nerve was verified. In almost every case where some kind of dissection or manipulation of the nerve were necessary there was postoperative deterioration of oculomotor function ranging from slight mydriasis to complete ptosis and paralysis of the extraocular muscles. in cases with posterior communicating aneurisms the pupilloconstrictor fibers running in the superior part of the nerve trunk were mostly affected while in tumor cases pupillary dilatation as the only sign of oculomotor lesion was uncommon. In cases with meningiomas where extensive and protracted dissection aimed at the complete excision of these benign lesions was the rule the percentage of postoperative impairment of the oculomotor nerve function was the greatest (36,5%). These findings are in agreement with the statements of other surgeons for the third nerve "being the most sensitive" and showing "most often postoperative worsening" (2) in prolonged surgery for benign lesions.

In sellar tumors oculomotor lesions were rarer and invariably were due to compression of the cavernous segment of the nerve as its cisternal part was quite away from the direction of extrasellar growth. The operative decompression was not associated with third nerve dissection and postoperative worsening was exceptional. The soft consistence of the sellar tumors had its role as it had in cases with temporal gliomas and cavernomas (the last being harder in the presence of calcification).

The degree of third nerve impairment is not dependent on the size of the lesion for ther were cases with large tumors or aneurysms with evident compression of the nerve but preserved function as well as lesions with small size but complete functional paralisis. Factors of greater importance for the degree of impairment were the site and direction of growth, the occurence of acute episodes (hemorrhage, infarction, inflammation).

We have made an analysis of the site and direction of growth as the cause for oculomotor dysfunction in 25 cases operated during the last 3 years (Fig. 1).

In cases with origin of tumor growth medially or dorsocaudally to the entry zone of the third nerve in the cavernous sinus (7 cases and 5 cases correspondingly) where the nerve was dislocated in upward or lateral direction the proportion of oculomotor dysfunction was greater then in cases with laterally or anterosuperior origin of the tumor growth (7 cases and 6 cases correspondingly). The postoperative impairment of the nerve is more frequent in the former cases because the nerve trunk is in the operative field and it has to be constantly protected from injury. Doubtless this is the cause for the more frequent postoperative deterioration of the oculomotor function in cases with medial or dorsocaudal origin of growth of the lesion.

The possibillities for microsurgical suture of the nerve are very limited as the nerve is supplying many musles and even in cases with incomplete injury the spontaneous regeneration is aberant. So the only chance for preservation of the oculomotor function is the early diagnosis and timely operative treatment of craniobasal pathologic processes.

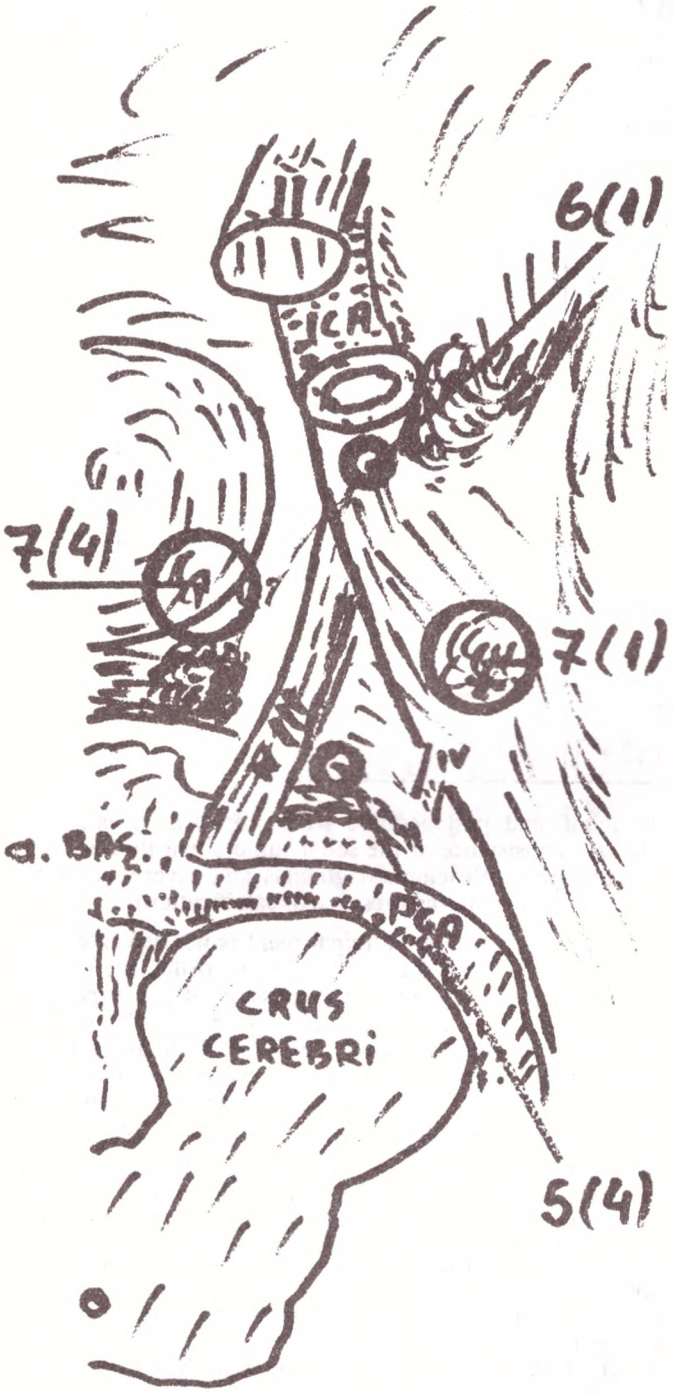

Fig. 1. Origin and direction of growth of lessions reffering to the third nerve trunk and entry zone in the cavernous sinus.

REFERENCES

1. Arnold, H., H. D. Hermann: In: Advances in Neurosurgery (eds. H.Dietz, M. Brock, M.Klinger), Springer, Berlin, 1985, 183-185.
2. Eggert, H. R., J. M. Gilsbach: Ibid, 195-202.
3. Inoue T., H.R. Rhoton, D. Teele at al.: Neurosurgery, 26, 1990, 6, 903-932.
4. Lang, J.: In: Advances in Neurosurgery (eds. H. Dietz, M. Brock, M. Klinder) Springer, Berlin, 1985, 173-182.
5. Perneczky, A., E. Knosp, Ch. Matula: Acta Neurochir. Wien, 92, 1988, 76-82
6. Sekhar, L., P. Janetta, J., Maroon: Neurosurgery, 14, 1982, 3, 268-275.
7. Symon, L.: In: Advances and Technical Standart in Neurosurgery, (eds. L. Symon et al.), Springer, Berlin, v. 9, 1982, 69-112.

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INTRAOPERATIVE USE OF SOMATOSENSORY EVOKED RESPONCES IN ANEURYSM SURGERY: OBSERVATION ON 38 CASES

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INTRODUCTION:

One of the important tasks in treatment of cerebral aneurysms is to protect the patient from critical hypoperfusion and ischemia during surgery. The increased ischemic risk after subarachnoid haemorrhage have been demonstrated by some authors, showing a well delineated relationships between impaired cerebral blood flow (vasospasm), somatosensory evoked potentials (SEP) and clinical grading of patients according to Hunt and Hess scale (10, 18, 22).

The theoretical basis for intraoperativ SEP monitoring is provided by extensive experimental work on cerebral blood flow, bioelectrical activity and neuronal viability (1, 8, 11, 12, 16, 19, 25). Initial results in applying SEP monitoring to detect early neurological damage during critical surgical maneuvers in aneurysm surgery have been reported (3, 6, 7, 15, 20, 21, 26-29) and further experience is beeng recently reported (2, 4, 5, 13, 14, 17, 23, 24, 27). Nevertheless, the present literature lacks precise identifica-

tion of the tolerans limits for intraoperative SEP changes and additional confusion results from different anesthesiological protocols, use or no use of deliberate arterial hypotension and deliberate parent vessel clipping during aneurysm dissection.

The initial experience with intraoperative monitoring in cerebral aneurysm surgery was evaluated by the author during his research stay at the University Neurosurgical Department in Giessen, Germany with the support of A. von Humbold foundation, Bonn, Germany. In this study we report on the observed electrophysiological alterations and compare them with the patients postoperative outcome. By doing so we hope to contribute additional information on the prognostic value of these methods.

MATERIALS AND METHODS:

This analysis comprises 38 patients, (25 females and 13 males, mean age 52 y) oprated on in the period 1988-89 for cerebral anerysms in the Neurosurgical clinic of the University of Giessen, Germany with the additional use of intraoperative SEP monitoring. These included 31 patients with solitary aneurisms / arteria communicans anterior (ACoA) - arteria cerebri anterior (ACA) - complex - 10, arteria carotis interna (ACI) - 12, arteria cerebri media (ACM) - 8 and tip of the basilar arteri (BA) -1/ as well 7 patients with multiple aneurisms -6 had 2 and 1 patient had four lesions. Fifteen patients were in grade I/I-II/, 12 in grade II, 6 in grade III and 2 in grade III-IV according to Hunt and Hess, 1969(10). The remaining 3 patients were excluded from this preoperative clinical grading because their second operations were monitored. Within 72 hours following subarchnoid hemorrhage were operated on 21 patients, and the remaining 14 underwent surgery between days 4 and 52.

For premedication Flunitrazepam was used, anesthesia maintenence was achieved by N_2O/O_2, rarely halogenated anesthetics, Fentanyl. Body temperature was kept between 35-36° C. In only 2 patients deliberate intraoperative arterial hypotension was used during certain periods of dissection (sodium nitroprusside). In aneurysms of ACI and ACM contralaterral median nerve stimulation at the wrist was performed and in cases with ACoA-ACA- complex aneurysms we took an effort to monitor the leg area following posterior tibial nerve stimulation behind the inner malleolus. Short latency SEP were recorded continuously throughout the operation, starting after induction in anesthesia.

The stimulus intensity was of such degree as to elicit small twitch of the thumb, resp. of the toe (or 2 mA above motor threshold), stimulus mode - restangular pulses with duration 0,3 msec, frequency 5/sec. In medianus -SEP recordings needle electrodes were placed over Cz(+)-P_3, P_4(-) and in tibialis SEP monitoring Fp_2/+/-Cz setting was used. Using a Toennis DA IIR system 256 sweeps were averaged and the analysis time in medianus SEP was 50 msec (corresponding values for tibialis SEP-100-500 repetitions and 100 msec). The low frequency cutoff was 1000 Hz, the low one - 50 Hz. CCT in median nerve SEP was calculated as a difference of N20 and N14 peak latencies (9).

RESULTS:

Thirty eight operative procedures for intracranial aneurysms were continuosly monitored by means of SEP. Because of technical difficultes we failed to obtain well delineated responces in 1 patient with tibialis post. SEP.

Anesthesia related changes in CCT were observed in all cases (from 5,98 ± 0,78 in anesthesia induction period up to 6,35 ± 0,75 msec during aneurysm dissection and 6,10 ± 0,76 msec at the end of surgery).

During operation 17 different "surgical events" have been registered - 13 of them in 9 patients with medianus SEP (Table 1) (out of total 30 cases) and 4 - in 4 patients with tibialis SEP monitoring. In the majority of occasions these were premature aneurysm ruptures, requiring temporary clipping of the parent vessel (vessels) to put the bleeding under control. In 4 occasions brain retraction by itself caused the observed SEP- alterations and therefore it was considered as a "surgical event". This remarkable sensitivity of SEP monitoring is well demonstrated during different steps in surgery in Figure 1. (Patient N 8 from Table 1), an uneventful postoperative course was operated.

CASE REPORTS:

Case 1.

A 68 year old female (patient N 2 in Table 1) underwent early operation for clipping of left ACI -ACoP aneurysm; the patient was grade I-II. During dissection of the aneurysm neck premature rupture of the fundus occured, followed by a pronounced temporal lobe swelling. Temporary clipping of the proximal ACI and partial resection of the temporal lobe pole were accomplished. The definitive clipping of the aneurysm was additionally hampered by an atheromatous plaque in the carotid wall. The surgeon succeeded in clipping using 3 Aesculap cips, but the proximal occlusion of ACI lasted for alltogether 15 minutes. During these maneuvers arterial blood pressure was kept between 105-115/50 mmHg. As seen in figure 2, dramatic changes in SEP were observed. Amplitude of N20 dropped promptly after ACI occlusion and 2 minutes later the cortical responce was not visible without any sings of recovery thereafter. In the postoperative period the patient remained comatous with right sided hemiplegia, left oculomotor paresis and transitory midbrain syndrome. The CT scan revealed infarction of the ACP territory and the corresponding brain stem ganglia.

Case 2.

In patient N3 from Table 1 two aneurysms (ACM and ACoA) were treated during the same procedure. Source of bleeding was obviously the aneurysm, located in the angle between left ACM trunk and anterior temporal branch. During dissection a rupture of the aneurysm dome occured. Temporary clips were placed proximal and distal on the main trunk and on the temporal branch for 5 minutes, which allowed the surgeon to clip securely the aneurism neck. This event was accompanied by CCT prolongation, reaching 8, 9 msec and N20 amplitude reduction /Figure 3/. Within 7 minutes following blood flow release SEP returned to baseline values. It is important to note, that during this operation deliberate arterial hypotension /MABP around 60 mmHg/ was used. The patient awoke with moderate right hemiparesis and sensory dysphasia. On the next day she regained to some extent motor function, but rightsided dyskinesia /hemibalism/ developed, lasting for 10 days. This picture was thereafter slightly retrograde and the patient was discharged for rehabilitation. Interestingly, no visible CT changes could be demonstrated. One may speculate, that in this case the effect of temporary blood flow interruption had been significantly enchanced by the systemic arterial hypotension. Another explanation could be the possibility for inadvertent clip occlusion of a thalamostriate perforating branches of ACM, which supplies a territory, lying beyond the somatosensory pathways monitored.

As regarding patients with posterior tibialis SEP, 4 surgical events in 4 patients out of alltogether 11 investigations were noted. In 3 patients temporary clipping of A1 for 21, respectively 5 and 3 minutes was not accompanied by any SEP alterations and clinical deterioration. Umfortunately, in the last patient with intraoperativ rupture of ACoA - aneurysm and temporary clipping at both A1 segments for 5 minutes a technically unsatisfactory recording was ob-

tained; in the same case a new postoperative deficit was encountered, consisting in mild left leg paresis, which disappeared within the next 24 hours.

Several groups can be differentiated regarding the presence or absence of "surgical events" and corresponding SEP recordings and if we try to compare this groups to the preoperative clinical grading /Table 2/, one may not directly conclude, that a clear relationship exists between them. Nevertheless, refering to both tables 1 and 2 it becomes evident, that grade II and III patients are more prone to display SEP changes during trivial surgical manipulations like brain retraction and following intentional parent vessel clipping.

In 2 out of 9 patients with "surgical events" and medianus SEP alterations a postoperative deficit occured /one of them operated on under deliberate arterial hypotension/. The CCT prolongation in the remaining 7 patients ranged from 6,3 up to 9,3 msec; in 5 of them N20 was completely abolished within 1-8 minutes and returned to baseline values 2 to 4 minutes thereafter. Thus, temporary occlusion of ICA lasting up to 2 minutes and of the ACM up to 5 minutesseemes to be tolerated well in our cases. The definitive brain demage in case N 2 was not unexpected since SEP were lost prompt und inreversible following ICA occlusion in spite of blood flow release 15 minutes thereafter. It seems, that safety limits for ACI occlusion lie somewhere in the wide range between 2 and 15 minutes, provided the SEP have returned to baseline within 5 minutes after blood flow restoration.

Concerning late postoperative deterioration, suggestive of postponed cerebral vasospasm and observed in 6 patients in this series it is important to stress, that an uneventful SEP monitoring does not exclude the possibility for such complications.

DISCUSSION:

Although the method awaits further use and accumulation of experience, our results are indicative of its usefulness and reliability in more complicated types of aneurysm surgery. In spite of this we are reserved in outlining more precisely the prognostic value of the observed SEP changes since this is relative unextensive experience. In the conditions of intraoperative arterial normotonia CCT prolongations up to 9, 3 msec and N20 loss within 1-8 minutes following the event with subsequent recovery time lasting from 2 minutes for ACI occlusion to 5 minutes for ACM clipping were tolerated well from the patients. As a grave prognostic sign may be regarded the prompt and irreversible loss of SEP.

According to experience of the group from National Hospital, London (21, 22, 26, 27, 29) ACI and ACM could be occluded up to 10 minutes with impunity, provided CCT prolongation did not exceed 9-10 msec. On the other hand, none of their cases with postoperative neurological deterioration have had normal CCT after temporary vessel clipping and CCT has been prolonged over 10 msec. Ischemic injury could be expected in case of N20 abolishion, but if this happenes immidiately following temporary clip placement. If these changes developed 3-4 minutes thereafter, the risks for postoperative were estimated as unsignificant. The reappearence of N20 up to 20 minutes after blood flow restoration is considered to be a positive prognostic sign. Our findings are in agreement except for the only patient with deliberate arterial hypotension. Focusing on the last observation one must recall the conclusions of Ducati et al, 1988 (4), that for all patients with normal preoperative SEP recordings (i. e. without vasospasm) mean intraoperative arterial pressure values over 60 Torr are safe as regards critical CCT values of approximately 9msec. In the context of clearly established relationship between CCT, cerebral blood flow and preoperative clinical grading (22) one may interpret the prevalence of SEP alterations among our grade II and III patients.

Undoubtedly, the most important question is how SEP alterations influence upon surgeon's decision making and strategy. Friedman et al, 1987 (5) proposed in definitive SEP abolishion clip readjustment and performance of an extra-intracranial arterial bypass at the end of operation. Reversal of temporary clippind seems to be obviously a reasonable step only if intentional vessel occlusion has been done as a preventive measure. However, when a temporary clip has been placed to stop bleeding from premature aneurysm rupture the situation is more critical (24). In these circumstances Schramm et al. (24) advise after achieving incomplete hemostasis by means to release the temporary clip for several minutes until adequate clipping is accomplished. The authors emphasized, that unaltered SEP monitoring gives the surgeon a feeling of safety especially in risky manevers during aneurysm surgery like long lasting temporary parent vessel occlusion, intentional permanent vessel occlusion and application of a ring clip onto a giant aneurysms of ACI.

Table 1

DETAILED ANALYSIS OF THE SEP-CHANGES DURING 13 "EVENTS" IN 9 PATIENTS WITH N. MEDIANUS STIMULATION

No, PATIENT, GRADE	EVENT	CCT-CHANGE (msec)	TIME TO CCT-CHANGE (min)	TIME TO CCT-RECOVERY (min)	TIME TO N20 DISAPPEARENCE (min)	TIME TO N20 RECOVERY (min)	POSTOPERATIVE MORBIDITY
1. M.M., I-II	Rupture, TC ACI for 1min 20sec	6,3-8,0	2	2	-	-	-
2. B.E., I-II	Rupture, TC ACI for 15min	-	1	no recovery	2	no recovery	+
3. M.W., II	Rupture, TC ACM for 5min	6,5-8,9	2	7	amplitude↓	7	+
4. S.D., II	Rupture, TC ACI for 2min	7,1-7,5	2	10	-	-	-
	Brain retraction	7,1-8,0	10	4	-	-	-
5. E.J., III	TC ACM for 5min	-	-	-	2	5	-
6. N.K., II	Compression ACI	-	1	5	1	5	-
	Clipping aneurysma neck	7,5-9-3	1	5	aplitude↓	5	-
7. B.L., III	Brain retraction	6,0-6,8	10	3	ampltude↓	4	-
8. B.E., III	Brain retraction	6,5-7,8	4	5	3	4	-
	Brain retraction	6,5-8,0	2	4	8	2	-
	Hemorrhage, RR 40/0	-	-	-	2	4	-
9. O.H., II-III	Rupture, RR 100/60	6,8-8,8	5	6	-	-	-

TC - temporary clipping, CCT - central conduction time, ACI - a. carotis interna, ACM - a. cerebri media, A1 and A2 - segments of a. cerebri anterior

Table 2

RELATIONSHIP BETWEEN PREOPERATIVE CLINICAL GRADING AND INTRAOPERATIVE SEP-CHANGES PATIENTS WITH CEREBRAL ANEURYSMS

SURGICAL "EVENTS" & SEP-CHANGES / PREOPERATIVE CLINICAL GRADING	WITH "EVENTS" AND SEP-CHANGES	WITH "EVENTS" BUT WITHOUT SEP-CHANGES	WITHOUT "EVENTS" AND SEP-CHANGES	TOTAL
I (I-II)	3	1	11	15
II	3	-	9	12
III	4	1	1	6
III-IV	-	1	1	2
Total	10	3	22	35

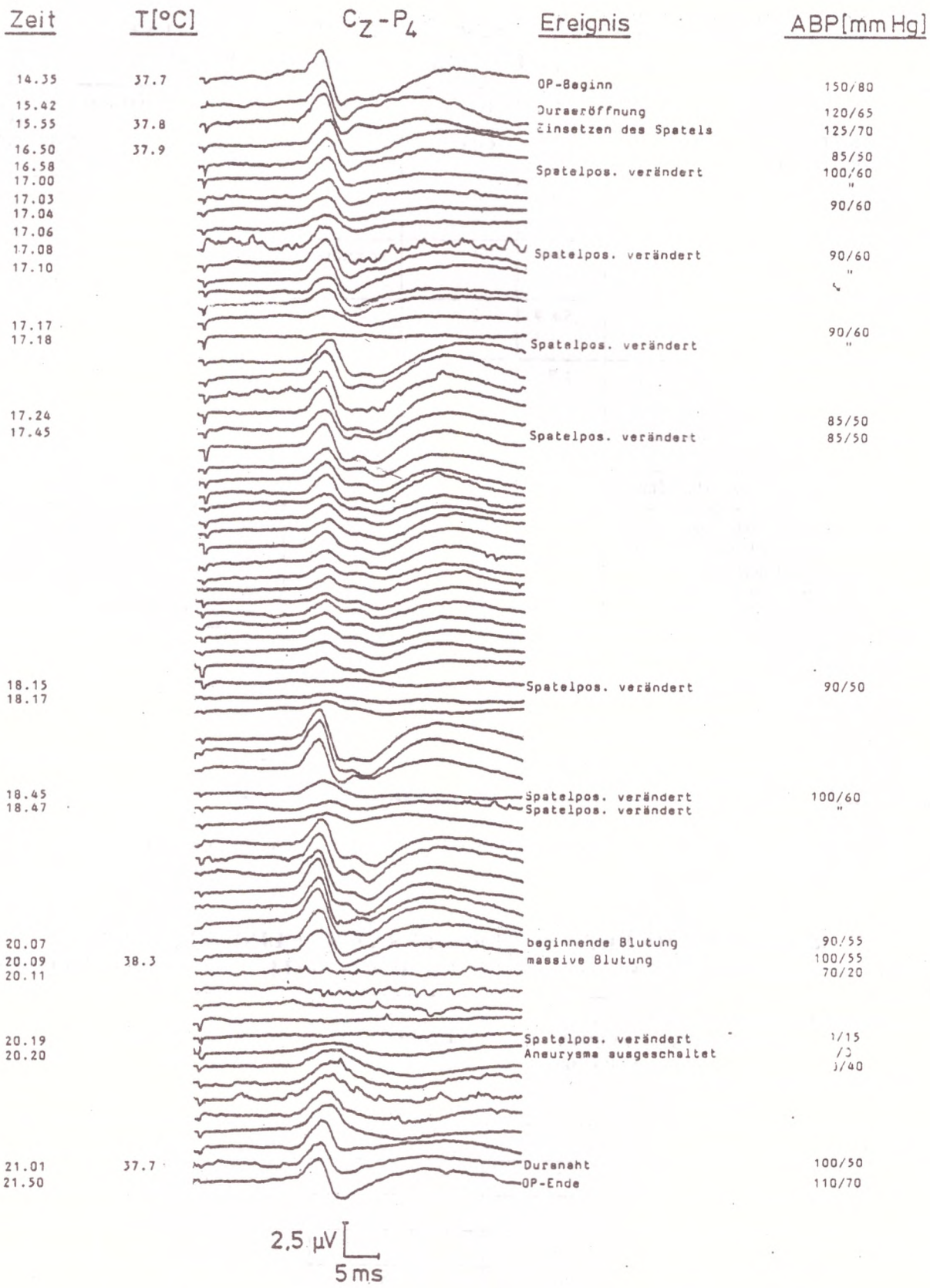

Fig. 1. BASILARISANEURYSMA SEP-MONITORING

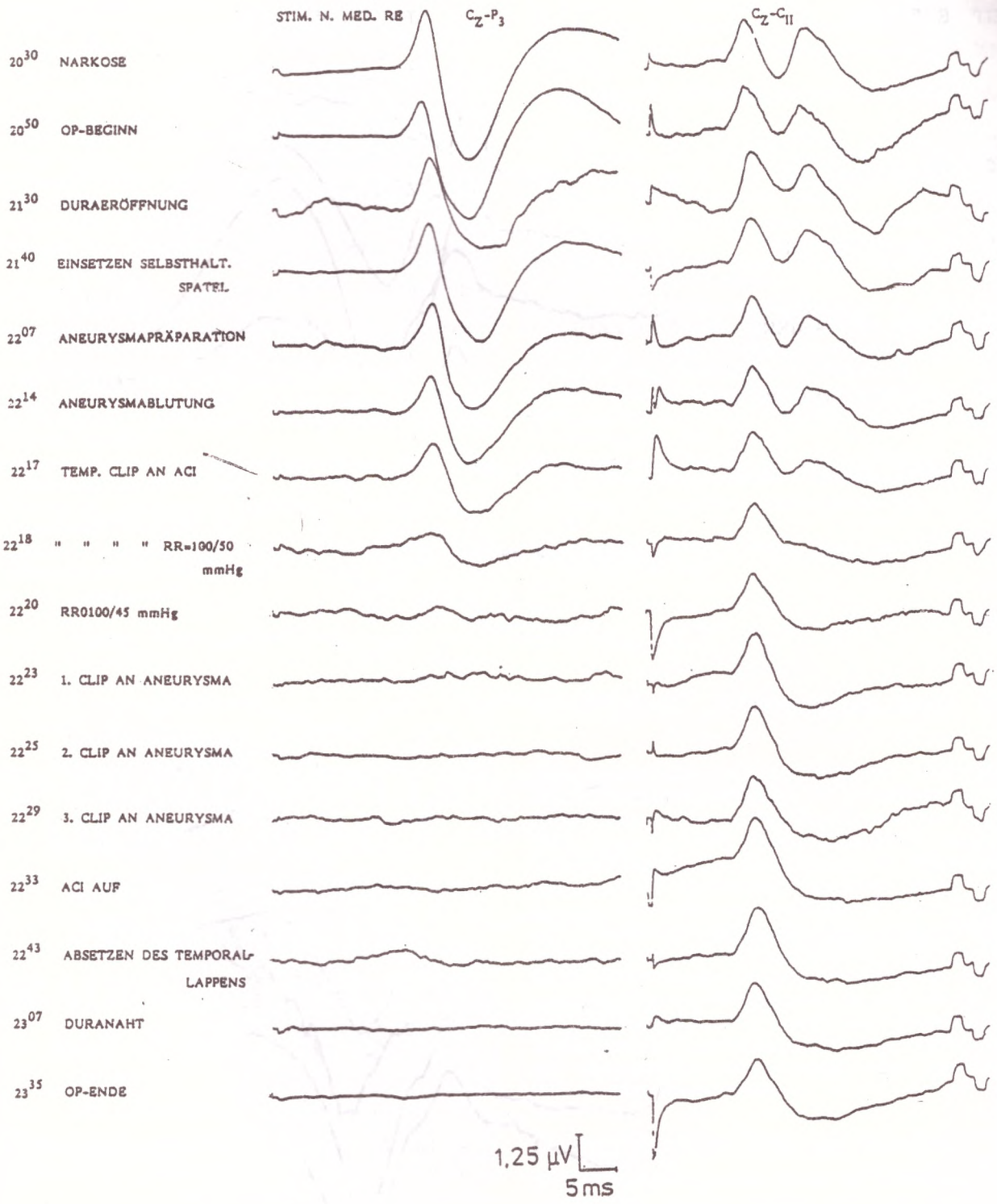

Fig. 2. SEP-MONITORING BEI ANEURYSMA DER A. COMM. POST. LI

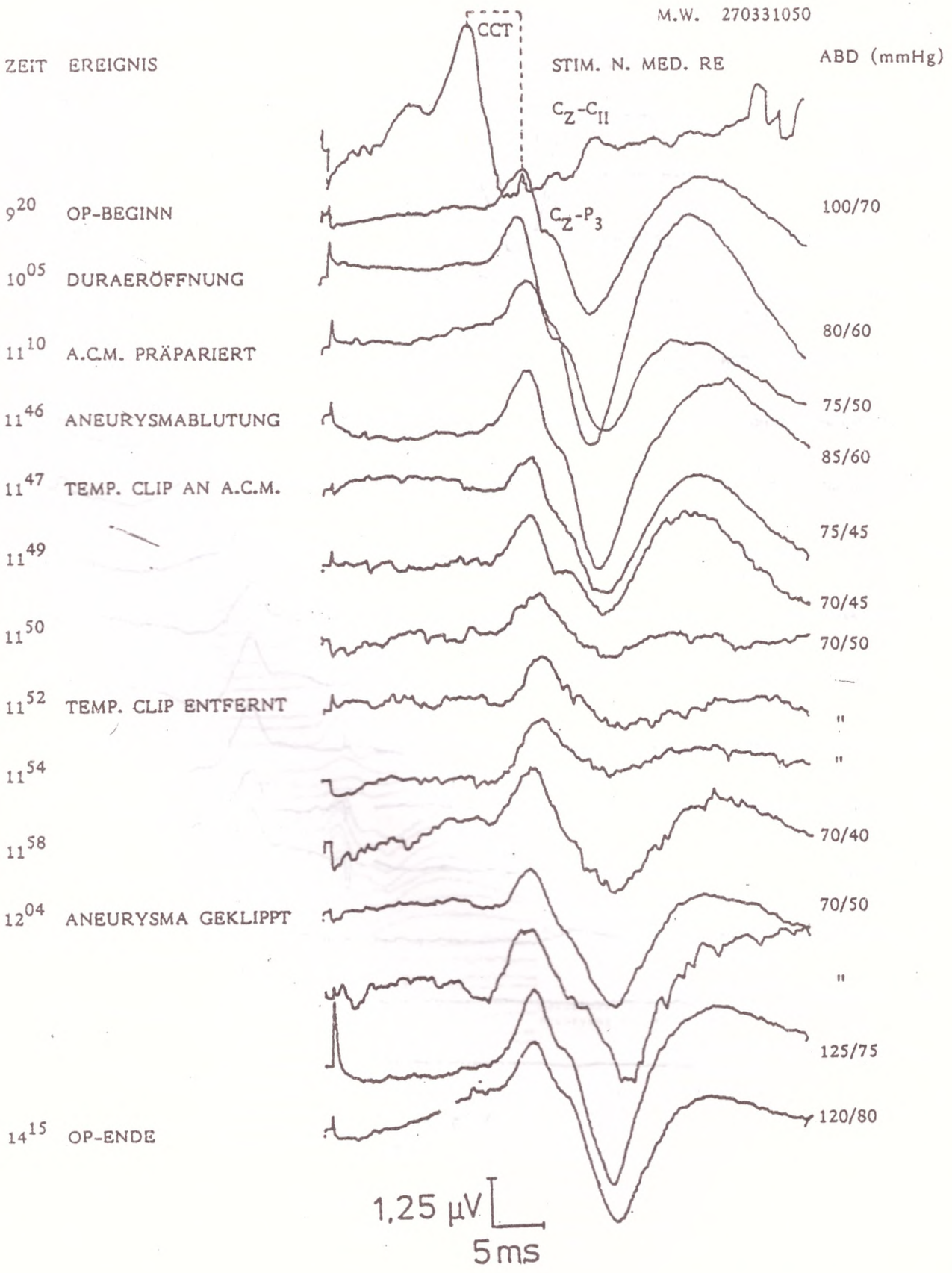

Fig. 3. SEP-MONITORING BEI ANEURYSMA DER A. C. MEDIA LI

REFERNCES

1. Branston N.M, L.Symon, H.A.Crockard, E.Pazstor. Exp Neurol 45,1975, 195-208.
2. Buchtal A., M.Belopavlivic, J.J.A.Mooij. Acta Neurochir (Wien),1988,93, 28-36
3. Carter L.P., P.A.Raudzens, C.Gaines, R.M.Crowell. Neurosurgery 15, 1984, 22-28.
4. Ducati A., M.Landi, M.Cenzato, E.Fava, P.Rampini, M.Giovanelli, R.Vilani. Acta Neurochir (Wien), Suppl 42, 1988, 8-13
5. Friedman W.A., B.L.Kaplan, A.L.Day, G.W.Sypert, M.T.Curran. Neurosurgery , 20, 1987, 678-687
6. Grundy B.L. Neurosurgery 11, 1982, 556-575.
7. Grundy B.L., P.B.Nelson, A.Lina, R.C.Heros. Neurosurgery 11,1982,64-67.
8. Hargadine J.R., N.M. Branston, L.Symon. Stroke 11,1980,637-643.
9. Hume A.L., Cant B.R. EEG Clin Neurophysiol 45,1978,361-375.
10. Hunt W.E., M.R.Hess. J.Neusurg 28,1968, 14-20.
11. Jones T.H., R.B.Morawetz, R.M.Crowell, F.W.Marcoux, S.J.Fitzgibbon, U.De Girolami, R.G.Ojemann. J Neurosurg, 54,1981,773-782.
12. Kataoka K., R.Graf, G.Rosner, B.Radermacher, W.D. Heiss. J Neurol Sci 79,1987, 117-127.
13. Kidooka M., Y.Nakasu, K.Watanabe, M.Matsuda, J.Handa. Surg Neurol 27,1987, 69-76.
14. Koht A., J.Schramm, E.Watanabe, G.Schmidt. Clin Neurol Neurosurg 89-2/1, 1987,71(abstr.)
15. Lauwthes Y., M.Da Motta, Ch.Saint-prix, W.Ferreira. 8th European Congress of Neurosurgery, 1987, Barcelona - Abstracts, 13.
16. Lesnicks J.E., Michele J.J., F.A.Simeone, S.De Feo, F.A.Welsh. J Neurosurg 60, 1984, 490-494
17. Little J.R., R.P Lesser, H.Luederes. Neurosurgery, 20, 1987, 421-427.
18. Ljunggren B., H.Saveland, L.Brandt. Neurosurgery 13, 1983, 629-633.
19. Loftus C.M., D.D.Bernstein, J.Starr, T.Yamada, S.Wegrzynowicz, T.Koster. Neurosurgery 4, 1987,503-508.
20. McPherson R.R.W., E.F.Niedermeyer, R.J.Otenasek, D.F. Hanley. Neurosurg 59, 1983, 146-149
21. Momma F., A.D.Wang, L.Symon. Surg Neurol 27, 1987, 343-352.
22. Rosenstein J., A.D.J.Wang, L.Symon, M.Suzuki. J Neurosurg 1985,25-30.
23. Schramm J. In: Neuromonitoring in Surgery (ed. J.E.Desmedt), Amsterdam, Elsevier S.P., 1989, 263-274.
24. Schramm J., A.Koht, G.Schmidt, U.Pechstein, M.Taniguchi, R.Fahlbusch. Neurosurgery, 26, 1990, 61-70.
25. Symon L. Acta Neurochir (Wien) 77, 1985, 1-7.
26. Symon L., A.D.Wang, E.Costa, I.E.Sylva, F.Gentili. J. Neurosurg 60, 1984, 269-275.
27. Symon L., T.Murota. In: Neuromonitoring in Surgery (ed.J.E.Desmedt), ElsevierS.P., Amsterdam, 1989, 263-274.
28. Thurner F., Schramm J. Anaesth Intensiv Med 27, 1986, 42-46.
29. Wang A.D., J.Cone, L.Symon. Costa e Sylva I.E. J Neurosurg 60, 1984, 264-268.

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INTRAKRANIELLE MENINGEOME : ANALYSE DER OPERATIVEN RADIKALITÄT BEI 668 PATIENTEN

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EINLEITUNG:

Die Meherzahl der intrakraniellen Meningeome (IM) sind gutartige Neubildungen,weshalb die chirurgische radikale Entfernung von entscheidender Bedeutung ist (3,8,10). Die Lokalisation und Ggröβe des Tumors, die Zugänglichkeit, die Beziehung zu funktionell wichtigen Hirnstrukturen,die histopathologischen Besonderheiten und Vaskularisation sind als bestimmende Faktoren für die radikale Enrfernung der IM bekannt. Der heutige Einzatz moderner operativer Technologien (LASER, Ultraschallaspirator), neuer mikrochirurgischer Zugänge und hiriprotektiver Maβnahmen bei schonendender anatomischer Dissektion führte zur Senkung der Morbidität und Mortalität (1,8,11-13,16,). Dennoch gelingt trotz des unbestrittenen Fortschritts in der Neurochirurgie und Neuroanastesiologie auch heute die radikale Entferning der IM nicht in jedem Fall. Im Unterschied zu fruheren Publikationen, die sich auf ein kleines Patientengut beziehen (7), wurde eine zusammenfassende Studie über die Faktoren erstellt, die die Operabilität der IM eingeschränkt haben, wobei eine repräsentative Fallzahl ausgewertet wurde.

PATIENTENGUT UND METHODIK:

In der Neurochirurgischen Klinik der Medizinischen Universität Sofia wurden zwischen 1974 und 1989 668 Patienten mit IM, davon 274 (41%) Männer und 394 (59%) Frauen operiert. Folgende Ausgangsdaten wurden für die Analyse benutzt:

1. Die Topik des Prozesses (Tabelle 2). In 16 Fällen (2.4%) wurden histologisch maligne IM festgestellt.
2. Die klinische Symptomatik wurde in 3 Gruppen unterteilt, die den klinischen Zustand zum Zeitpunkt der Intervention zusammenfassend darstellen: im latenten

Stadium der Erkrankung operierten wie 107 Patienten (16%), im manifesten Stadium 484 (72%) und im Stadium einer klinischen Dekompensation wurden 77 Patienten (12%) bei uns vorgestellt und operiert.

3. Anzahl der operativen Eingriffe und Ausmaβ der Exstirpation. Es wurden 499 Patienten einmal operiert, 129 Patienten zweimal, 28 Patienten dreimal, 4 und mehr Interventionen führten wir bei 12 Patienten durch. Bei der ersten Operation betrug die Zahl der Patienten 668, bei der zweiten 169, bei der dritten 40 und bei der vierten und folgenden 12.

4. Die Radikalität der Entfernung (Radiksalismus) des IM wurde folgendermasen eingestuft: Totale Exstirpation, subtotale Exstirpation, partielle Exstirpatoin Biopsie und externe Dekompression (darunter wird die zusätzliche Kalottenfragmentenfernung nach Tumorexstirpation nicht in Betracht gezogen). Unter totaler Exstirpation verstehen wir Radikalitat Grad I (komplette Entfernung mit Resektion der betroffener Dura und Knochen), Grad II (komplette Entfernung mit anschliesender Koagulation der duralen Haftstelle) und Grad III nach Simpson, 1957 (14) (komplette Tumorentfernung ohne Resektion oder Koagulation der duralen Haftstele (z.b. bei infiltriertem Sinus). Im letzten Fall handelte es sich um eine sogenannte "radikale" Intervention bei schwierig zugänglischem IM (parasellärem,petroclivalem u.a.). Je nach Größe des Tumorrestes (Grad IV nach Simpson 1957) versteilen wir die Patienten in die obengegannten Gruppen. In die Gruppe mit subtotaler Entfernung sind die Fälle mit geringerem Tumorrest,der fest und oder in funktionell wichtigen Gehirnstrukturen und Hirngefasen eingeaschen ist, was die komplette Entfernung zu risikoreich machte, eingeordnet. Hierbei war im Vergleich zu früheren Fällen das Ausmas des Tumorestes unter mikrochirurgischer Technik kleiner und betrug durchschnittlich 5-15 mm(7).

ANALYSE UND ERGEBNISSE:

Eine totale Extirpation nahmen wir bei 418 Patienten (62,6%) unserer Serie vor,eine subtotale Entfernung wurde bei 88 Patienten (13,2%) durhgefuhrt,eine partielle bei 131 (19,6%), eine Biopsie wurde bei 18 Patienten entnommen (2,7%) und eine externe Dekompressoin erhielten 13 Patienten (1,9%). Symptomfrei oder neurologisch deutlich verbessert wurden bis zum Zeitpunkt der Beendigung der Katamnese (Ende 1990) 503 Patienten (75,3%) 40 blieben postoperativ unverändert (6%), 41 zeigen eine Verschlechterung (6,1%) und 84 verstarben operative Mortalität von 12,6% . Darüberhinaus betrug die operative Mortalität bei der ersten Intervention 8,&% , bei der zweiten stieg sie auf 9,5% ,bei der dritten erreichte sie 20% und bei der vierten und nachfolgenden 16,7% . Nach Einführung der Computertomographie in unserer klinik (1977) und der routinemasigen Mikrochirurgie (1976), sowie auch der Ultraschallaspiration, ist die operative Mortalitat auf unter 6% gesunken.

Die totale Entfernung der IM war beim ersten Eingriff nicht immer möglich (Tabelle 1). Das Verhälthis der radikalen (totalen und subtotalen) Interventionen gegenüber der Zahl der inkompletten bleibt bei der ersten und zweiten Intervention unverändert -2:1. Inkomplette Eingriffe waren gewöhnlich Folge entweder einer manifesten klinischen Dekompensation zum Zeitpunkt der Operation oder einer unkontrollierbaren Blutung und Gehirschwellung,wobei in den nachsten Schritten eine radikale Entfernung angestrebt wurde. Aus Tabelle 2 wird ersichtlich, daβ die Chancen fur eine komplette Exstirpation stiegen, wenn der Tumor oberflächlich und entfernt von groβen arteriellen und venosen Gefäβen und Hirnnerven lag. Tabelle 3 demonstriert die Abhängigkeit der radikalen Entfernung vom klinischen Stadium - die Möglichkeiten zu einer totalen und subtotalen Exstipation betragen in den latenten und manifesten Stadien etwa 77%, jedoch nur etwa 66% im Stadium einer klinischen Dekompensation , und zwar vor allem bei oberflächlich gelegenen Tumoren. Der kritische praoperative Zustand war erwartundsgemäss mit einer sehr hohen operativen Mortalität in etwa 1/3 der Patienten verbunden. Als Ursache für eine inkompletten Tumorentfernung lag am häufigsten ein Umwachsen des IM von grosen arteriellen Gefäβsen, vom venösen Sinus und vom hirnstamm sowie von Hirnnerven (44%) vor. Als zweithäufigste Ursache ist eine wird eine akute Hirnschwellung und eine ausgepragte intraoperative Blutung erwähnt (42%), ferner kommt als Ursache die Malignität der IM (9%) sowie ihr ausgedehntes Wachstum und das damit verbundene Dislokationsrisiko (5%) vor.

DISKUSSION:

Die Analyse zeigt, das die Topik des Tumor eine wichtige beschrenkende Rolle hinsichtlich der operativen radikalitat spielt,insbesondere gillt das fur die basalen und tiefgelegenen IM. Wenn wir uns auf eine fruhere Publikation beziehen (7), sind z.B.die Ursachen fur unzureichend radikale Operationen bei paraselläre IM in detaillierter Form folgendermaβen darzustellen: Ummauerung der Gefäβstrukturen des Circulus art. Willisi (insbesondere der kleinen perforierenden Gefäβe) im Tumorparenchym somie der medialen Wand der A. carotis interna in ihrem supraclinoidalen Abschnitt und des Sinus cavernosus, Beeintractigung von Chiasma und Sehnerven bei einer vorwiegend subchiasmalen Entwicklung des Tumors mit Verwachsungen der Tumorkapsel am Hypophysentiel,Hypothalamus und zerebralen Pedunkel sowie an den III, IV und VI Hirnnerven, extradurales Wachstum mit Knocheninvasion des Keilbeins und der Orbita, extreme Vaskularisation des Tumors usw. Unsere Erwartung,das der Anteil der komplett exstirpierten IM des Tub. sellae und Planum sphenoidale größer ist als des kleinem Keilbeinflügels hat sich nicht bestätigt (Tabelle 2). Die Ursache dafür liegt im Ausmaβ der Meningiome des Tuberculum sellae und Pl. sphenoidale zum Zeitpunkt der Operation. Die von uns erzielte Radikalität bei der Entfernung der parasellaren Meningeome beträgt nur etwa 67%, wobei sich die Angaben in der Literatur zwischen 77 und 100% bewegen (1,2,8,10,12,15,16), und zwar bei einer niedrigeren Mortalitat. Die späte Diagnosestellung der basalen Meningeome ist vorwiegend auf Nichterkennung der Frühsymptome (Hirnnervenausfälle) zuruckzufuhren, woraus ein groβes Tumorwachstum mit inkompletter Tumorexstipation resultiert. O. AL-Mefti (1) legt groβen Wert darauf, daβ bei entsprechender Früherkennung und bei kleineren Tumoren ein adäquater Zugang mittels mikrochirurgischer Technik die Aufrechterhaltung eines arachnoidalen Dissektionsplans zwischen Tumor und Hirnstrukturen durchaus möglich macht. Sowohl durch fortgeschrittenes Wachstum als auch durch Späterkennung läβt sich die nicht zufriedenstellende Radikalitat auch bei parasagittalen und konvexitalen Lokalisationen der IM erklären. Eine zusätzliche Rolle spielt hier auch die vorzeitige Unterbrechnung des Eingriffs aufgrund massiver Blutung mit Kreislaufzusammenbruch oder aufgrund Infiltration von sagittalen Venen und venosem Sinus. Obwohl die Radikalität der zweiten Intervention die der er-

sten nicht übertrifft, zeigte die postoperative Mortalität bei den Reoperationen deutliche Tendenz zu einer Erhöhnung bis hin zu unakzeptierbaren Werten. Letzteres war offensichtlich auf die angestrebte maximal komplette Entfernung zurückzufuhren (7). Hier stoßen wir auf eine prinzipiell wichtige, jedoch umstrittenen Frage, der einmaligen Radikalität auf Kosten erhohter Morbidität and Mortalität oder der Zurückhaltung vor eine maximalen, risikoreichen Radikalität mittels mehrerer Eingriffe, wobei sich der Patient in den Intervallen zwischen den Operationen in einem zufriedenstellenden Zustand befindet (17). Erfahrungsgmäß wurden wir letzteres befürworten. Einige Autoren weisen darauf hin, das bei basal gelegenen Mittellinienmeningeom mit teilweise infiltrativem Wachstum in Richtung Sinus cavernosus und nach sphenoorbital ein gutes funktionelles Resultat bei radikaler exstirpation wegen der Gefahr schwerwiegender neurologischer Defizite, Dienzephalonschädigung, Sehverlust und Hirnnerveninsuffizienz kaum erreichen läßt, ja sogar unmoglich ist (5,12). Die aggressivere Dissektion des Tumorgewebes von der Gefäßwand birgt das Risiko nicht nur einer Gefäßläsion, sondern auch die Gefahr der Entwicklung eines spaten postoperativen Vasospasmus oder einer Thrombose (5). Daher sollte der haftende Tumorrest getrent von seiner Durainserzion belassen werden,da die Wahrscheinlichkeit eines weiteren wesentlichen Wachstums als gering eingeschätzt wird. Pertuiset et al. 1985 (11) raten vor einer radikalen Operation bei Patienten mit erhohtem internistischen Risiko (was ubrigens bei basalen IM oft der Fall ist), als auch bei Patienten uber 70 Jahre ab. Hier erweist sich sogar bei oberflachlich gelegenen IM die partielle Exstirpation mit Unterbrechnung und Koagulation der duralen Haftsstelle als besonders ratsam.

Besonders Interesse erwecken die Ergebnisse einiger langfristigen retrospektiven Studien (6,9,14). Einerseits zeigt sich die Wachstumsrate der Rezidive bei radikal entfernten IM nach langjahrigen katamnestischen Verfolgung (15-20 Jahre) als ziemlich hoch - 19 -23%, andererseits bleibt jedoch unbestritten, das die erzielte Radikalität doch noch der wesentlichste Faktor ist, der die Prognose beeinflust. Dies bestimmt auch die neuen Tendenzen der Entwicklung in der Neurochirurgie bei IM: gröstmöglische Radikalität mit Hilfe neurophysiologischer Uberwachung und Hirnprotektion,schonende mikrochirurgische Preparation mittels neuer operativer Zugänge unter Erhaltung oder direkter Rekonstruktion des intracavernösen Abschnittes der a. carotis interna,prophylaktischer Angelen einer intraextrakraniellen arteriellen Anastomose u.a. (1,2,4,13).

Tabelle 1

ERZIELTE RADIKALITÄT UND REIHENFOLGE DER OPERATION

Operation / Radikalität	Erste	Zweite	Dritte	Vierte u. folgende
Totale Exstirpation	333	71	12	2
Subtotale Exstirpation	96	38	10	
Partielle Exstirpation	199	48	16	10
Biopsie	34	7	-	-
Extreme Dekompression	6	5	2	-
Summe	668	169	40	12

Tabelle 2

ERZIELTE RADIKALITÄT UND KLINISCHES STADIUM

Localisation	Totale Entfernung	Subtotale Enternung	Partielle Externe Entfernung	Biopsy Dekompression		Summe
Konvexität	130	14	19	6	4	173
Parasagittal	107	18	12	7	4	148
Tub.Sellae u. Pl.Sphenoid	43	14	33	-	4	94
Klienes Keilbeinflugel	40	16	20	-	2	78
Temporal	32	6	8	-	2	48
Subtentoriell	18	9	13	-	1	41
Tentorium	17	3	13	-	-	33
Kranioorbital	1	6	4	-	-	11
Orbita	5	1	2	-	-	8
Multiple	7	1	1	-	-	9
Intraventrikulär	7	-	2	-	-	9
Olfaktorius	3	-	2	-	1	6
Kraniospinal	5	-	1	-	-	6
Optische Kanal	3	-	-	-	-	3
Sinus Cavernosus	-	-	1	-	-	1
Summe	418	88	131	13	18	668

Tabelle 3

ERZIELTE RADIKALITÄT UND KLINISCHES STADIUM

Stadium / Radikalität	Latentes	Maniofestes	Dekomp.	Summe
Totale Exstirpation	65	314	39	418
Subtotale Exstirpation	17	60	11	88
Partielle Exstirpation	17	96	18	131
Biopsie	3	9	6	18
Externe Dekompression	5	5	3	13
Summe	107	484	77	668

LITERATUR

1. Al-Mefti O. Surgery of the cranial base,Kluwer,Boston,1988.
2. Al-Mefti O. J Neurosurg 75,1990,840-849.
3. Cushing H., L.Eisenhardt. Meningiomas. Their classification, regional behavior,life history and surgical end results. Spiringfield Ill., Ch.Thomas 1928,p 785
4. Dolenc V.V. Anatomy and surgery of the cavernous sinus.Sprinder Wien,1989.
5. Fohanno D., A.Bitar. Sphenoidal ridge meningeoma.Adv Techn Stand Neurosurg, Vol 14, Springfield, 1986, 137-174.
6. Jaaskeleinen J. Surg Neurol 1986, 25, 142-149.
7. Karkesselijan A. Neurol.Phychiat i Neurochir.(Bulg.) 24, 1985, No1, 37-41.
8. Maxwell R.E., S.N.Chou. Preoperative evaluation and management of meningeomas. In:Schmidek H.H., W.H.,Sweet (eds.) Operative neurosurgical techniques - indications,methods and results. Vol. 1, Orlando, Grune&Stratton 1988,pp. 557-554.
9. Mirimanoff R.O., D.E.Dosoretz, L.M.Lingood, R.G.Ojeman. J Neurosurg 1983,62,18-24.
10. Ojemann R.G. Meningiomas: clinical features and surgical management.In: Wilkins RH. Rengachary SS (eds.) Neurosurgery vol.1, New York McGraw-Hill 1985,pp 635-654.
11. Pertuiset B., S.Farah, L.Clayes, J.Gontorbe, J.Mertzger, M.Kujas. Acta Neurochir (Wien), 1985,76, 2-1.
12. Probst Ch. Acta Neurochir(Wien) 1987,84,99-102
13. Sekhar L.N., C.N.Seu., H.D.Iho, O.P.Janeska. Neurosurgery 24, 1989, 18-30.
14. Simpson D. J. Neurol Neurosurg Psychiat 20,1957,22-39.
15. Solero C.L., S.Giombini, G.Morello. Acta Neurochir (Wien)67,1987, 181-184.
16. Symon L., J.Rosenstein. J. Neurosurg 61,1984,No4, 633-641.
17. Watts C. Sphenoid wing meningeoma.In:Long D.M.(ed.) Current therapy in neurological surgery 1985, Decker Toronto,pp 14-16.

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POSTOPERATIVE RECOVERY OF THE ANTERIOR ROOT LESSIONS DUE TO LUMBAR DISK DISEASE

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The motor loss recovery with lumbar disk disease after surgical treatment is a matter of controversy. It is generally agreed however that when a significant motor loss is present, the recovery is not always possible, even after surgery. Many autors emphasize that surgical decompression must be done before the appearance of motor deficit and when it is present or deteriorates – an urgent surgical treatment is mandatory 2, 3, 4, 7. According to the Bulgarian neurosurgeon P. Petrov (1980) (1) good results could be expected if surgery is performed before the apperiance of clinical and BMG signs of root interstitial damage.

The aim of the present study is to asses the recovery of the anterior root lessions after surgery, by means of pre- and postoperative clinical examination and EMG investigation of the anterior root conduction.

MATERIAL AND METHOD

Sixti-five patients operated on for lumbar disc disease with unilateral radiculopathy were prospectivly studied. The operative findings were: lateral disc prolaps- in L5-S1 level – 29 patients, in L4-L5 level – 24 and double level herniation – in one case; root canal stenosis – L5-S1 – 6 cases and L4-L5 – 4 cases. The postoperative follow-up was done 10-15 monts after operation.

The operative results were classified according to the clinical assessment as:

– g o o d – without pain and obvious motor deficit, fully recovered labour fitness.

– u s t i s f a c t o r y – not fully recovered neurological deficit and labour fitness.

The anterior root condition was investigated by P. Merton and H. Morton's technique for transcutaneus spinal electrostimulation (5, 6, 8). Brief High voltage electrical impulses were delivered on the appropriate spinal level and anterior root motor evoked potentials (ARMEP) were recorded from proper muscles: for L3 myotom m. quadriceps femoris; for L4 – m. tibialis anterior; for L5 – m. peroneus longus and m. extensor digitorum brevis; for S1 – m. gastrocnemius (lateral head) and for S2 – m. abductor hallucis.

The assymetry of the ARMEP's latencies between the affected and the infact side over 1,5 ms was accepted as significant.

RESULTS

1. Clinical findnigs

Motor deficit was found preoperatively in 31 from all of the studied patients, as follow: 10 patients – with weakness of ankle dorsiflection; 4 – weakness of extension of a great toe; 12 – weakness of plantar flexion and 5 – with mixed foot paresis. Twenty-three of these cases had a muscle wasting as well.

Postoperative clinical examenation revealed full recovery in 12 patients (39%). In the remaining 19 patients we found improvement in 15 while 4 had no changes.

The postoperative results assessed clinically were:

– g o o d – in 41 patients (63,1%).

– u n s a t i s f a c t o r y – in 24 patients (36,9%).

2. EMG findings

Preoperative investigation revealed significant assymetry of ARMEP's latencies between symptomatiNc and asymptomatic sides in 48 cases (73,7%) for one or more recordings.

Postoperative EMG follow-up showed the following results: All of the patients without MEG abnormalies before operation had normal EMG findings after surgery (17 cases). The results in patients with positive EMG findings before surgery were: in 12 cases – normal postoperative EMG; in 30 – the EMG findings were improved; in three the findings wer obvious change and one patient had a deterioration.

There was a significant decrease of the assymetry of ARMEP's latencies between symptomatic and asymptomatic sides after surgical treatment (Fig. 1)

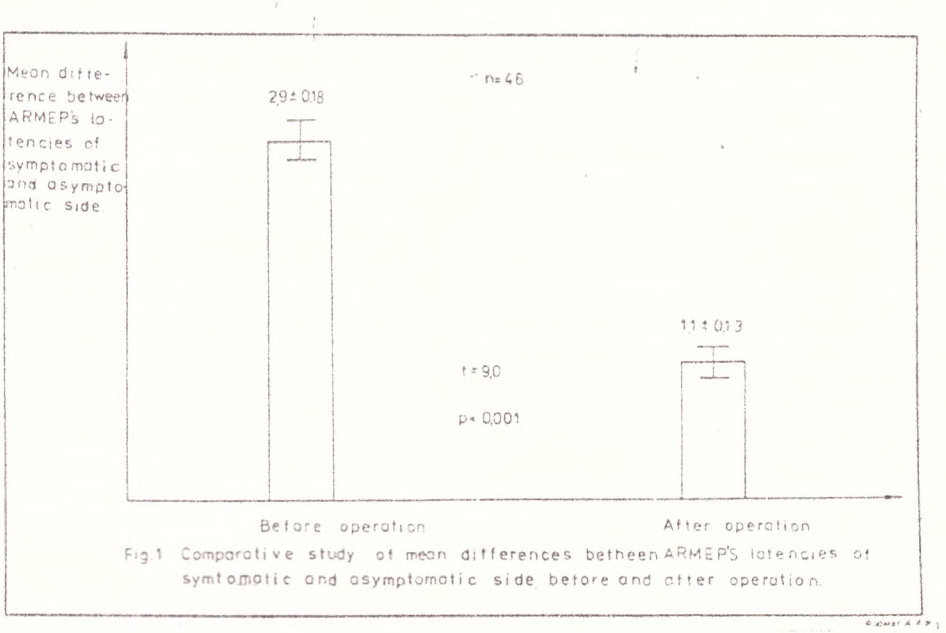

Fig.1 Comparative study of mean differences betheen ARMEP'S latencies of symtomatic and asymptomatic side before and after operation.

The recovery of anterior root conduction after surgery was always paralleled by clinical improvement (Fig. 2), with slight (not significant) advance in clinical improvement.

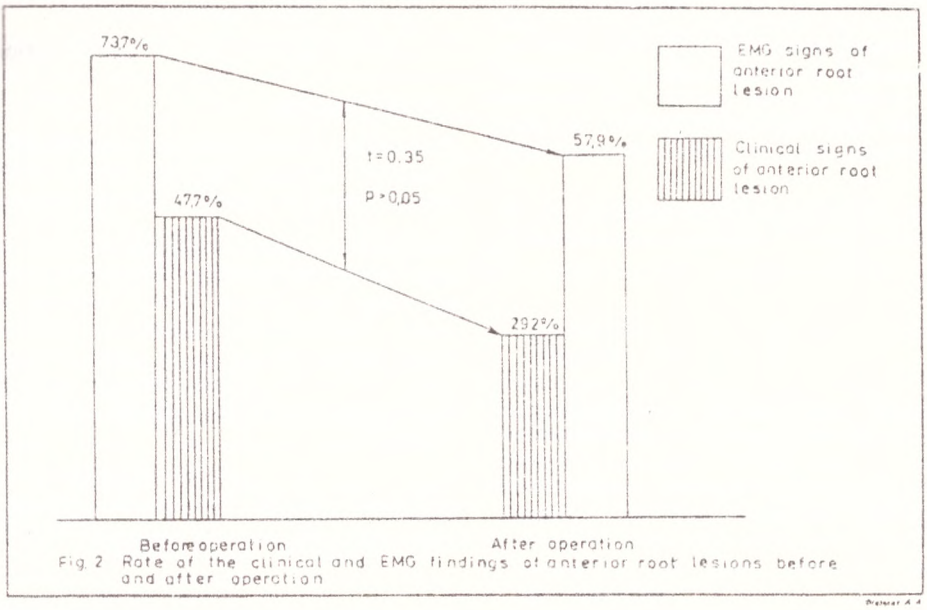

Fig. 2 Rate of the clinical and EMG findings of anterior root lesions before and after operation

We found that the operative results were highly innfluenced (r = 0,57) by the preoperative clinical and electrophysiological status of the patients (Fig. 3).

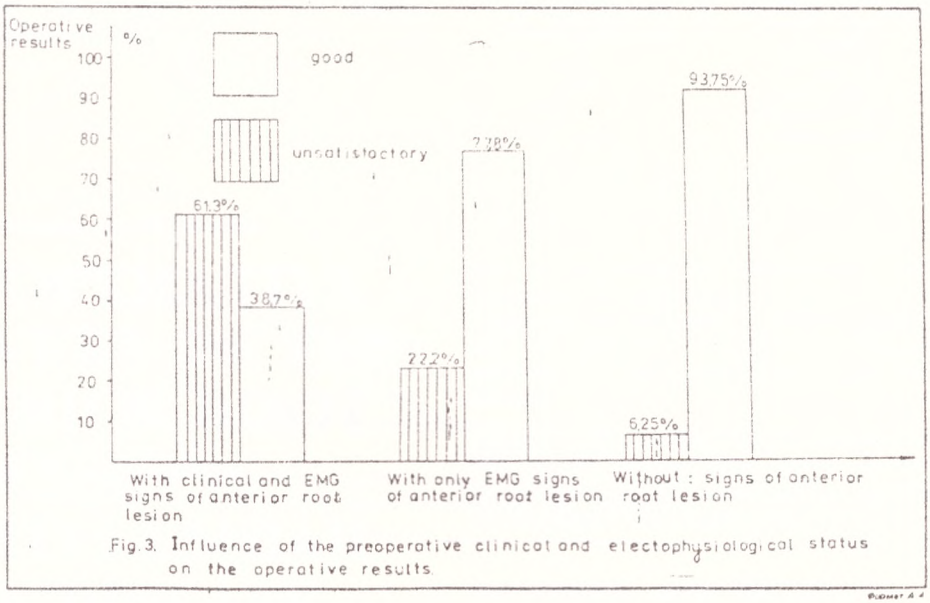

Fig. 3. Influence of the preoperative clinical and electophysiological status on the operative results.

The unsatisfactory results were prevailing in the presence of clinical and EMG signs of anterior root damage, while there were much better results in their absence. The good results prevailed in the stage of only conduction abnormalities of the anterior root.

DISCUSSION AND CONCLUSIONS

We found motor deficit in 47,7% and abnormal anterior root conduction in 73,7% of the patients in this series, thus revealing a high rate of subclinical anterior root pathology.

The results from the postoperative follow-up suggest that the motor loss due to lumbar disc disease not always disappears after surgery (there was full recovery in 39% of the cases in our series). The recovery of the anterior root conduction after surgery was always paralleled by clinical improvement.

A close corelation was found between the preoperative clinical and electrophysiological status and the results of the surgical treatment. Unsatisfactory results were prevailing in the patients with clinical and EMG evidencec of anterior root damage, suggesting that once anterior root

symptoms have appeared they are likely to become a chronic problem. The surgical decompression gives much better results in the stage of subclinical anterior root lession (with only EMG evidence), compared to the stage with clinical signs of motor deficit. Therefore the abnormalities of anterior root conduction may be a complementary indication for plainning surgery in the whole context of the clinical and radiological indications.

REFERENCES

1. Петров, П. Поясно-дискова болест. София, Мед. и физк., 1980.
2. Durning, V., M.L. Murphy: Post. Grad. Med. 79, 1986, 5, 54-74
3. Kerr, R, J. Codoux-Hudson, C. Adams: J. Neurol., Neurosurg., Psychiatry, 51, 1988, 2, 87-93.
4. Kramer, J. Intervertebral disc disease. Causis, diagnosis and treatment. Stuttgart, New York: Thieme, 1980.
5. Merton, P., H. Morton, D. Hill, C. Marsden: Lancet, 1982, 2, 597-600.
6. Noordhout, M. de, J. Rothwell, P. Thompson: J. Neurol. Neurosurg., Psychiatry, 1988, 51, 174-181.
7. Schuwer, U., K, Roosen: Neurochirurgia, 31, 1988, 1, 192-195
8. Tchalukova-Atanasova, N., St. Baykoushev, N. Jeljazkov: Folia medica, 1990, 4.

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DIAGNOSIS AND TREATMENT OF BRAIN CONCUSSION

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The methods for the objectifying of brain concussion (BC) in the routine practice are too limited and are based mainly on the history for a brain injury and loss of consciousness, a damage of the soft tissues of the cranium and the changes in EEG. The treatment is predominently symptomatic for the headache (the main symptom), the vertigo and the astheno-vegetative syndrome.

The investigations all over the world demonstrate that BC is an condition that includes changes in the brain blood flow and the metabolism of the nerve cell with complex pathological processes in the carbohydrate, protein and fatty metabolisms which gives the grounds to look for other criteria for an objectifying (1-10).

On the basis of 163 investigated patients with BC (biochemical, electrophysiological, psychological and other methods) we found that different deviations in the normal brain activity can be used as criteria for the Brain Concussion:

1. In 85 % of the patients with BC in CSF isoenzyme BB (with normal values of CPK and total protein) is found. In healthy persons, isoenzyme BB in CSF is absent.
2. In 72 % in blood serum isoenzyme BB (with normal values of CPK) is found. Normally isoenzyme BB in blood serum is absent.
3. Changes in the isoenzyme spectrum of LDH in CSF. Increasing of the isoenzyme LDH5 and decreasing of LDH1 in 52 % as the total value of LDH is normal.
4. A change of the equilibrium of the piruvate-lactate with an increase of the lactate in 52 % of the patients.
5. A change of the equilibrium of the piruvate-lactate with an increasing of the lactate in 52 % of the patients.
6. The spectrophotometry shows microhaemorrhages in a clear, colourless CSF in 22%.
7. SPECT – a change of the brain blood flow with areas of hypoperfusion.
8. EEG – in 12 % focal changes coinciding with the spot of the injury, in 8 % they do not coincide and in 32 % there are diffuse changes which in a dynamic following show a tendency towards a normalization.
9. Changes in the memory and attention with a tendency for a quick restoration.

These results and also of many other autors (1,3,8,10) show that the changes of the brain metabolism pass in three basic mechanisms:

a) As a result of changes mainly in the brain blood flow the permability of the cell membrane is increased and some enzymes pass into the intracellular fluid.

b) By a laceration of the cell membrane of nerve cells and a pouring out of the cell content.

c) A mixed type.

The increased values of lactate and isoenzyme LDH5 and the decreased value of LDH1 on the background of a hypoperfusion argue for a slight metabolic acidosis. The changes in EEG, memory and attention give the opportunity for a dynamic following and give an information for the degree and the rate of the health process.

In the course of the treatment, the patients were divided in two groups: A and B.

In group A we used a symptomatic treatment for the headache and the astheno-vegetative syndrome. When leaving the hospital on 7-10th day, 32 % of the patients were with a slight headache.

In group B the treatment was aimed to improve the brain metabolism: glucose and insulin, nootrop drugs, vasodilaters etc. In 92 % of the patients, the headache disappeard on the 3rd – 4th day.

In conclusion we can say that BC is a disease connected with changes of the brain metabolism and the brain functions. Our proposed criteria are not unique, their number can be enlarged in different directions depending on the degree of the information.

The treatment has to be of a mixed type – to improve the brain metabolism with a corresponding symptomatic treatment.

REFERENCES

1. Crockard,H.A., A.R. Taylor. Europ. Neurol., 8, 1972, 151-157
2. Elkind,A.H. Clin.J.Pain, 5, 1989, 1, 77-86
3. Enevoldsen,E.M., F.T. Jensen. Clin. Neurol. Neurosurg, 80, 1977, 4, 213,225
4. Gentlini,M. et al. J. Neurol. Neurosurg. Psychiatry, 48, 1985, 2, 137-140 5. Gentlini,M. et al. J. Clin. Exp. Neuropsychol., 9, 1987, 1, 55-57
5. Gentlini,M. et al. J. Clin. Exp. Neuropsychol., 9, 1987, 1, 55-57
6. Gibbs,F.A., E.L. Gibbs. Clin. Electroencephalogr., 18, 1987, 1, 10-11
7. Hans,P. et al. J. Neurosurg., 71, 1989, 1, 54-58
8. Karpman,R.R. et al. J. Trauma, 21, 1981, 2, 359-364
9. L'guori,G. et al. Child's Nerv. Syst., 5, 1989, 160-162
10. Rabow,L.,G.Tibbling. Acta Neurochir., 37, 1977, 3-4, 245-261
11. Rabow,L.,G.Hedman. Acta Neurochir., 28, 1979, 108-112

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ANEURYSMS ASSOCIATED WITH POLYCYSTIC KIDNEY DISEASE

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Borelius (1901) has been credited to be the first who described the association of brain aneurysms (BA) with polycystic kidney disease (PKD). (B. Weir,1987). According to the data reported by Sachs and Meyers (1951) 7 % of the patients with PKD have brain aneurysms (BA). N.H. Bigelow (1953) found much higher percentage (16). Conversely the results of Sachs and Meyers (1951), Walton (1956) and Imaizumi et al. (1983) have shown 3 %, 5% and 8% resp. of PKD among patients harboring aneurysms.

The higher incidence of brain aneurysms among the patients with PKD compared to the rest of the population rises the question of diagnostic studies to prove their existence, as well as the indications for surgery to prevent the aneurysm rupture (A.S. Levey et al. 1983; Oken, 1983). Moreover the perioperative care of patients with PKD requires carefull monitoring of the kidney function (B. Weir, 1987).

In some of the patients with SAH due to ruptured aneurysm and PKD there is no previous history indicating renal affection. However the pathological findings in urine analysis should lead one to suspect PKD. (B. Weir, 1987).

Surprisingly the association of BA and PKD has been only occasionaly recorded in more than 500 cases of aneurysm surgeries in the Dept. of Neurosurgery.

Case presentation

The following presentation of two cases of BA and PKD as well as a short literature survey should increase the awareness of this entity and related management problems.

Case 1. B.G.G. (admission Nr. 18557/1990 – Inst. Pirogov). This 20-years old male patient was admitted on august 22nd with sudden onset of confusion, restlessness, unsteady gait followed by severe headache and drowsiness. The neurological examination has shown neck stiffness and absence of local neurological signs. Lumbar puncture was done and the CSF examination revealed blood. DSA demonstrated aneurysm of the AcomA (Fig.1) This patient was a known case of PKD diagnosed since early childhood, and proven by CT examination (Fig.2). His mother had moderate form of PKD. He has been under medication for controle of high blood pressure. On 12th of September 1990 he was operated. Aneurysm of the AcomA was clipped. The patient's postoperative course was uneventful.

Case 2. K.I.P. (admission Nr. 4741/1986 – Dept. Neurosurg. Med. Acad.,Sofia).

This 22-years old normotensive female patient had suffered two previous attacks of SAH and was admitted conscious without focal neurological deficit. The angiographic study has shown aneurysm of the right supraclinoid ICA. Urine analysis demonstrated 25-30 RBC and several WBC per field. The rest of laboratory investigations were normal. When awaiting for scheduled surgery she suffered third attack of severe SAH. The neurological examination has shown a patient in grade IV-V according to Hunt and Hess grading scale. Despite the rescucitation measures she deteriorated and expired on 18 Apr. 1986. The postmortem investigation has shown policystic kidneys (Fig.3). The microanatomical study demonstrated aneurysm of the right supraclinoid ICA and blood in the adjacent subarachnoid spaces.

DISCUSSION

The literature review shows significant differences in the frequency of PKD, the frequency of BA in these patients as well as the rate of PKD in patients having BA. The early pathological analysis of Sachs (1950) has shown 4 cases of PKD in 60 postmortem examinations of patients suffering SAH and its complications. In a subsequent paper Sachs and Meyers (1951) reported incidence of 7% of BA in patients having PKD, where as the incidence of PKD in aneurysm patients was 3%. In one of the largest autopsy series of Brown (1951) based on 11245 cases the incidence of PKD was 0.32% and that of the BA 1.3%. Bigelow (1953) found 16% incidence of BA in cases PKD. Walton (1956) in another autopsy study reported 5% of PKD in 173 cases of BA. In the literature survey of Chester (1979) the incidence of BA in PKD cases is 16% and SAH proved to be cause of death in 15%. According to Wakabayashi et al. (1983) the incidence of PKD in BA cases is between 3 and 6%. Imaizumi et al. (1983) summarizing the autopsy files in Japan reported 8.4% incidence of BA in patients with PKD. Sohobe (1980) on the bases of 92854 autopsy reports has found 0.25% incidence of PKD and 1.6 of BA and the association of BA and PKD to be 0.01%. Since most of these results have been based on different referral patterns the differences are easily explainable.

Since the above reported cases are the only two among more that 500 aneurysm surgeries, obviousely the diagnosis of PKD in some patients with BA has been omitted.

The hypertention, palpable mass in the kidney area, protein in urine and pathological sediment should indicate the possibility of concomitant PKD. (Brackett and Morantz, 1982). The ultrasound, CT and pyelography can prove the existance and the severety of PKD. (B.Weir,1987). Since the incidence of BA in patients with PKD is higher it is reasonable to ask about the indications for angiography and surgery. Levey et al. (1983) concluded that angiography and surgery is indicated for patients below 25 years only. Their statement is based on the calculated risk of 0.001% of mortality rate for angiography, 0.01 mortality rate for aneurysm surgery if the aneurysm is single and located in anterior part of the Willis's circle, 0.02 if the aneurysms are bilateral, 0.03 if the aneurysm is located in VB system compared to the risk of 0.37 mortality and severe disability que to SAH. Their statements are critisized by Oken (1983) who considers the risk of morbidity and mortality due to SAH to be much higher than 0.37. Due to the recent advances in neuroimiging (DSA and MRI) the study of brain vessels is nowadays practically with very low risk. Wakabayashi et al. (1983) have published surgical results in patients with nonruptured aneurysms without morbidity and mortality. It should be emphasized that surgery for nonruptured aneurysms is much more easier from technical point of view. The perioperative care of the patients with PKD requires maintaining normal blood pressure, limited use of hyperosmotic solutions and monitoring of kidney function. (B. Weir, 1987). It might be conclused that in young patients suffering SAH PKD should be suspected. Moreover in patients with proven PKD attempt should be made to investigate the brain vessels for possible aneurysms, and surgery should be recommended for those without kidney insufficiency.

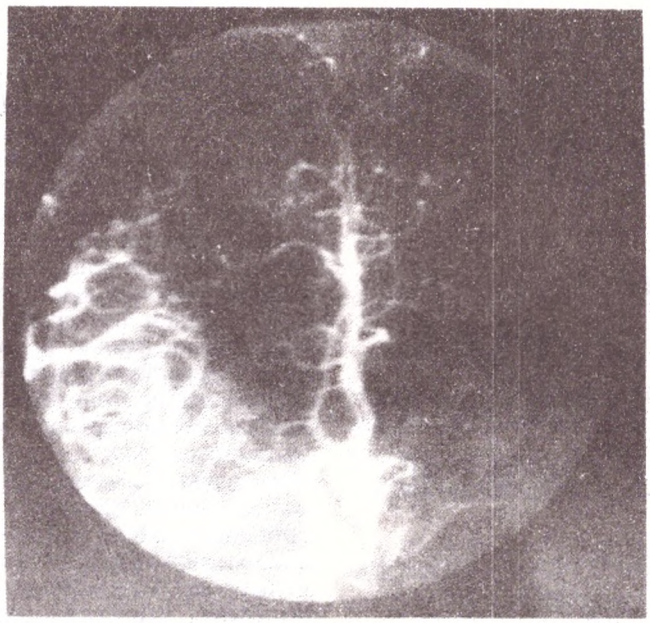

Fig. 1. Right carotid angiography showing aneurysm of the Acom A

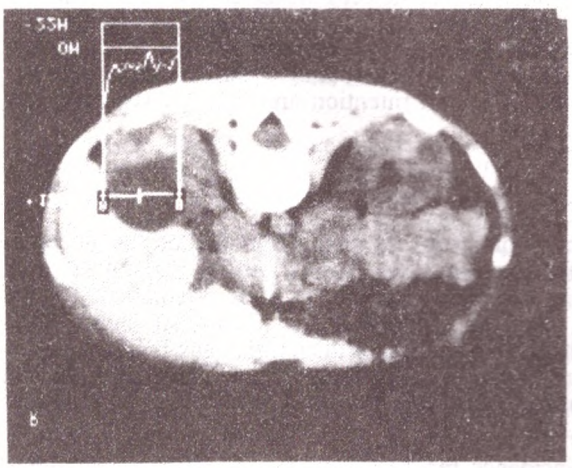

Fig. 2. CT demonstrating PKD.

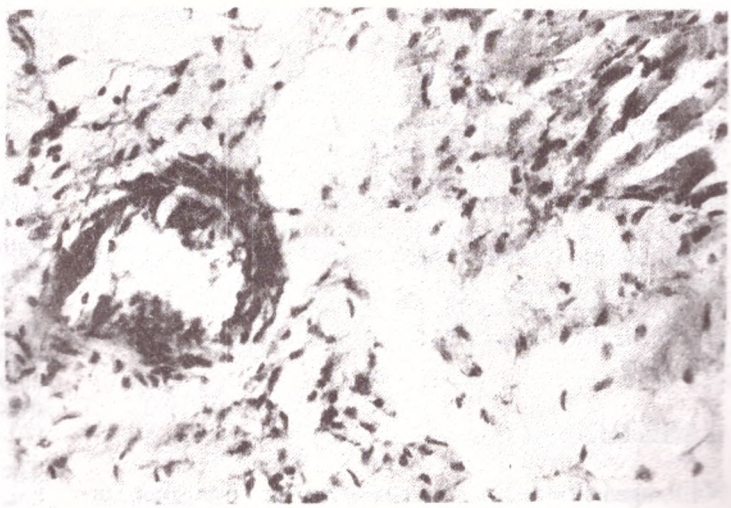

Fig. 3. Cystic formation in the kidney parenchimal showing chronic inflamatory changes in the interstitia.
Hemalaum Eosin
X 180

REFFERENCES

1. Bigelow, N.H.: Am.J. Med. Sci., 1953, 255:485-494
2. Borelius J.: Nord. Med. Ark., 1901,34: 1-27 Cit. B. Weir (1987)
3. Breckett C.E.Jr., R.A. Morantz: Special problems associated with subarachnoid hemorrhage. In Youmans J.R. (ed). Neurological Surgery, Philadelphia W.B. Saunders, 1982, Vol.3, pp 1808-1809
4. Brown,R.A.P.: Glasgow Med. J., 1951, 32: 333-348
5. Chester,A.S., Harris S.P., Schreiner G.E.: Am Family Physician, 1977, 16: 94-101
6. Fox,J.L.: Intracranial Aneurysms, New York, Springer-Verlag., 1983, Vol.I
7. Imaizumi,S.K. Sugita, K. Owada: Neurol.Surgery, II, 1983, 997-1001
8. Levey,A.S., S.G. Pauker, J.P. Kassirer: N. Engl. J.Med., 1983, 308: 986-944
9. Oken,B.S.: N.Engl. J. Med., 1983, 309: 927-928
10. Sachs,A.L.: Arch. Neurol. Psychiat., 1950, 63: 524 cit. B. Wier, 1987
11. Sachs, A.L., R. Meyers: Trans. Am. Neurol. Assoc., 1951, 76: 147-150
12. Sonobe,M., S. Takahachi, Y. Nagamine, H. Kawakati, H.S. Shibazaki, 1980, 32: 321-325
13. Snowdown,J., Br.J. Urol., 1974, 46: 583
14. Yakabayashi,T., Fugita,S., Ohbora Y. et al.: J. Neurosurgury, 1983, 58: 488-491
15. Walton, J.N.: Subarachnoid Haemorrhage e.d.s. Livingston Edinburg 1956
16. Weir,B.: Aneurysms affecting the nervous system. Williams & Wilkins, Baltimore, 1987, pp 61-63

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SUPLEMENT TO THE TECHNIQUE OF ONE – STAGE SURGICAL TREATMENT OF FRONTOETHMOIDAL ENCEPHALOCELE

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INTRODUCTION

A tendency has emerged during the last several years to one-stage surgical treatment of frontoethmoidal encephalocele. This new approach has numerous advantages over the traditional two-stage surgical treatment. This necessitates the improvement of the surgical techniques known so far with a view to shortening the duration of the surgical intervention. One-stage surgical treatment of that pathology is unthinkable whitout a surgical technique guaranteed the absolute air-tightness with the closing of the defect of the dura. We propose a surgical technique for closing the defect of the dura characterised by its physiology, safety and simplicity.

CASE REPORT

N.I.P. aged 12 months. The child was born from first normal pregnancy. The mother, aged 30, has been treated for sterility for 8 years. The existance of a cystic formation at the base of the nose has been established on birth. It was with a wide base, pulsating. No physical and mental retardation has been established. X-ray data: polycystic right lung; CT and craniography – frontoethmoidal encephalocele with a diameter of the internal orifice – about 40 mm, no involvement of the ventricular system. Cerebral angiography – no involvement of the major blood vessels. The operation was performed under general anestesia. Bifrontal craniotomy with the formation of bone-muscular flap with a base towards the right temporal muscle. The dura in the zone of the neck and bottom of the anterior cranial fossa is thinner and easily tearing. Its extradural separation proved to be impossible. The glial tissue, originating from the medio-basal-frontal segments of the brain and prolapsing through the hernal orifice was intradurally resected. That was followed by an oval skin incision around the base of the meningeal sac. The anterior wall of the meningeal sac is carefully separated to the level of the internal bone defect. A graft with a base in the area of the hernial neck is formed from the wall. The graft, with a shape and size corresponding to those of the internal bone defect, is invaginated intracranially through the bone canal. Its fixation, by single sutures (4/o thread, atraumatic) to the posterior and lateral edges of dura mater, surrounding the bone defect follows. Excellent airtightness has been achieved of the plastics. The strenght of the plastics was sufficient not necessitating an additional closing of the bone defect. The postoperative period was uneventful, with healing by first intention and with a very good cosmetic result.

DISCUSSION

Pathomorphologically the frontoethmoidal encephalocele is presented as a maningeal hernia with or without prolapse of brain substance, associated with a congenital skull defect, engaging the frontal and ethmoidal bones. Most often the internal orifice of the hernia neck is in the region of cecal foramen (2,4,7). Nasofrontal, nasoethmoidal and naso-orbital encephalocele are differentiated as sub-types (25,26,28).

Their incidence ranges from I/5 – 35 thousend children born alive (5,8,24). They are determined by many climatic, geographic and racial factors (19,22,27). A total of 78 children with that disease underwent operations within the period 1958-1989 at the Clinic of Neurosurgery, Alexandrov's University Hospital, Sofia.

Regardless of the unpleasant appearance, over 80 % of the children have been with normal mental and physical development (12,13). All viable children are to be subjected to surgical treatment (9,11). The majority of the present authors recommend an early surgical treatment (16,31).

The first operations are associated with the names of Fengar (1895), Dowman (1922), etc. (16,18). The majority of the authors use the two-stage surgical operation. The closing of the defect of dura mater is the key-moment of each intervention of that kind. Initially, via an extradural or subdural approach the base of the hernial neck is reached. The resection of the prolapsed, glially changed, cerebral tissue leads rarely to the deterioration of the neurologic symptomatics (19,20). After the separation of the dura from the area of the internal orific, it is fixed with single sutures. That method for elimination of the defect of dura mater is the most physiological one out of the so far known surgical techniques (1,2,3). Unfortunately, that proved impossible in case of bigger defects. On the other hand, the dura in that area is often in a poor condition and postoperative liquorrhea is often observed (11,16,17). In such cases, plastics is realized via grafts of various origin (6,9,14). Auto-tissues are preferred as fascia from the temporal muscle, part of fascia lata, periosteum from the frontal bone (23). Their application to such patients is also restricted. The problem is that a sufficiently large graft is not always possible to obtain, with a shape and size matching those of the defect. Furthemore, the separation of periosteum from the frontal bone could disturb the ossification processes in that area. The application of preserved tissues of biological origin makes the reputation as a method of choice (fascia, dura ot umbilical cord). Their advantage is that a graft well tolerated by organism could be obtained with the necessary size and strenght. Both that kind of graft and those with artificial origin always induce adhesions in the field of the plasics, hence disturbing the normal liquor circulation. Their fixation to the adjacent tissues is often technically difficult. Disadvantageous proved also to be their production, treatment, sterilization, preservation and storage (10). The use of synthetic materials is always connected with the solving of problems accompanying the implantation of foreign matter in organism (29,6). The closing of the bone defect is the next procedure at that stage of the surgical treatment. It is attained via small auto-bone-grafts, plastic or metal prostheses, synthetic matters (15,25). Some authors avoid the osseous plastics, relying on the strenght and air-tightness of the restored dura and the normal ossification processes of organism (21).

The external plastics, aiming mainly at cosmetic effects, is performed, most often, at a second stage. The interval between the two operations is different with the different authors (3,10). The removal of the meningeal sac is followed by a cosmetic reconstruction of the nose, hypertelorism respectively (15,19). The achievements of the modern surgical technique and reanimation directed the tendency to one-stage intra- and extra-cranial plastics (21,30). The majority of the authors, however, stick to the traditional surgical methods, making use only of the advantages of the one-stage introduction under general anesthesia (24,29).

We prefer the plastic potentialities of the extracranial part of the meningeal sac. In the majority of the cases its walls distally from the hernial neck, are distinguished for their thickness and strenght. It was removed as useless, during the other surgical methods, for the aesthetic shaping of the face. The plastics, per se, is realised via the mobilization of a flap from the anterior wall of the meningeal sac. The shape and size correspond to those of the intracranial defect. The so-formed graft, with the base in the area of the internal bone defect, is invaginated through the bone canal – intracranially. Via single sutures it is fixed firmly to the edges of the dura situated along the posterior and lateral walls of the bone defects. A reliable air-tightness and strenght of the plastics is attained. That technique is applicable even with primary sutured defect of the dura with a view to the improvement of the air-tightness and machanical safety of the plastics.

IN CONCLUSION WE COULD SAY:

1. It is one-stage plastics under the same general anesthesia.
2. Auto-tissues with their own blood supply are used.
3. Anatomic interrelations are created being close to the normal.
4. A tissue, being the closest to the physiological requirements is used as transplant. That reduces the risk of adhesions in the field of the plastics and impeded liquor circulation.
5. The size and shape of the graft could be formed depending on the type of the defect of the dura mater.
6. This technique is particularly effective in large defects, where the application of other autotransplants is technically difficult to realize.
7. The plastics, per se, is technically easy to realise, not prolonging the duration of the surgical operation.
8. Due to the strenght of the plastics, the plastic closing of the bone defect is not always necessary.
9. This technique allows the craniofacial reconstruction both at the same stage or at a later stage.

REFERENCES

1. Charoonsmith, T., Suwanwela,C. – Clin. plast. Surg., 1974; 1: 27-47.
2. Davis,C., Alexander,E. – J.Neurosurg., 1959; 16: 365-377.
3. Dhawan,I.,Tandon,P. – Child's Brain, 1982; 9: 126-132.
4. Drettner,B. – Acta Otolaryngol., 1963; 57: 181-187.
5. French,B. – In: J.Youmans, ed. Neurological Surgery. vol.3, second ed. Philadelphia: Saunders Co, 1982; 1236-1380.
6. Gerhard,H., Muller,G., Szdzuy,D., Biederman,F. – Zbl.Neurochirurgie, 1979; 40: 85-94.
7. Harverson,G., Bayley,I., Kiryabwire,J. – Clin.Radiol.,1974; 25: 317-322.
8. Hayashi,T., Ishibashi,A., Hashimito,T. – Chaild's Brain, 1982; 9: 139-140.
9. Henn,M., Voth,D.,Schwartz,M., Keßel,G., Mahlman,E. – In: Volth D., Glees P. eds. Spina bifida – Neural tube defects. Berlin: W. Gruiter, 1986: 255-231.
10. Karaguisov,L. – In: Operative Neurosurgery. Sofia: Medicina i Fiskultura. 1982: 151-153.
11. Matson,D. – In: Neurosurgery of infancy and childhood. second ed. Springfield: Ch. Thomas Publisher, 1869: 61-76.
12. Mealey,J., Dzenitis,A., Hockey,A. – J. Neurosurgery, 1970; 32: 209-218.
13. Merrem,G. – In: Lehrbuch der Neurochirurgie. Berlin: VEB Verlag Volk und Gesundheit, 1970; 330 – 331.
14. Muhling,J., Collman,H.,Renther,J., N. Neurosurg.Rev.,1989; 12: 21-23.

15. Mulliken,J. – Plast. & Reconstr. Surg., 1973; 51: 592-595.
16. Naim-ur-Rahman. J. of Neurological Sci., 1979; 42: 73-85.
17. Rahman,H., Agarval,V., Srivastava,A. – Indian J. Pediatr., 1979; 46: 210-214.
18. Raimondi,A. – In: Pediatric Neurosurgery, New York, Springer Verlag, 1987; 410-423.
19. Rapport,R., Durn, R., Alhady,F. – J. Neurosurg., 1981; 54: 213-219.
20. Riegel, D. – In: Pediatric Neurosurgery, ed. by Section of pediatric Neurosurgery of AANS. New York. Grund & Straton, 1982; 49-61.
21. Sergent,L., Seyfer,A., N. Gunby. – J. Neurosurgery, 1988; 68: 571-575.
22. Schirmer,M. – In: Einfuhrung In die neurochirurgie. Munchen: U&S Fachbuchen, 1975: 155-156.
23. Shillito,J. – In: Atlas of pediatric neurosurgical operations. Philadelphia: W.B. Saunders Co, 1982: 317-324.
24. Simpson,D., David,D., White,J. – Neurosurgery, 1984; 15: 14-21.
25. Suwanwela,C., Hongsaprabhas,C. – J. Neurosurg., 1966; 25: 172-182.
26. Suwanwela,C., Suwanwela,N. – J. Neurosurg., 1972; 36: 201-211.
27. Tandon,P. – Acta Neurol. Scand., 1970; 46: 369-383.
28. Tessier,P. – J. Maxillofac. Surg., 1976; 4: 69-92.
29. Whatmore,W. Brit. – J.Surg., 1973; 60: 261-271.
30. Whitaker,L., M.Broennle, Kerr,L., Herlich,A. – Plast. and Reconstr. Surg., 1980; 65: 561-570.
31. Yokota,A., Matsukado,Y., Fuwa,I. – Neurosurgery, 1986; 19: 468-478.

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SOMATOSENSORY EVOKED POTENTIALS MONITORING DURING CAROTID ENDARTEREC-TOMY(*)

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INTRODUCTION:

Cerebral monitoring and protection during carotid desobliteration (CD) in ischemic cerebrovascular desease remain a controversial issue. The question under discussion have enolved the rationale for monitoring and proper selection of monitoring methods (11, 15, 30). This is a purely prophylactic operation and for that reason a considerable effort is justified to prevent any operative mortality and morbidity that would be higher than those of natural history and medical therapy (9, 10, 15, 21).

Various methods for detection and prevention of critical intraoperative cerebral hypoperfusion were proposed assessment of the neurological status after awakening the patient during operation, local anestesia, use of arterial bypass shunts, carotid artery stump pressure estimation, intraoperative cerebral blood flow measurements, electrophysiologic monitoring by means of EEG and evoked potentials (EP) and use of nonanesthetic pharmacological interventions (3, 12, 19, 22, 25, 27, 34, 35). Undoubtefully, the most widely employed monitoring technique under general anesthesia is EEG (29-31), but nevertheless some authors questioned its value because of technical demands, too high sensitivity to several systemic factors and unsufficient reliability under general anesthesia. They found somatosensory evoked potentials (SEP) easier to record and interpret in carotid surgery, but conclude, that the use of the latter awaits further experience (8, 14, 16, 26).

The aim of the study was to make further contribution concerning the value of SEP monitoring in increasing the safety of CE. Special attention was paid on the identification of any reliable SEP-changes, which could be used as an appropriate guide during operation.

MATERIALS AND METHORDS

1. Patients characteristics:

In the period 1987-1989 there were 44 patients with hemodynamically significant carotid stenosis at the neck (35 males and 9 females, mean age 64,5), all treated surgically under SEP monitoring in the Department of Neurosurgery at the Justus-Liebig-University Giessen, Germany. The number of performed CE in 44 patients was 49. The most of the patients sought medical attention for transient ischemic attacks and mild insult, and 3 were asymptomatic. The medical risk factors are listed in table 1. The cerebral angiography revealed stenosis under 50% of the proximal internal carotid artery in 1 patient, between 50 and 75% – in 11 and over 75% – in 32 patients; the contralaternal carotid artery was stenotic in 26 patients and 16 had ulcerated plaques. The preoperative surgical risks were graded according the criteria of T. Sundt et al, 1987 (32) – Table II.

2. Intraoperative SEP monitoring:

Anesthesia was induced with Flunitrazepam, maintanence was achieved by N_2O/O_2, rarely halogenated anesthetic (Ethrane 0,4 vol%), Fentanyl (induction dose 0,2 mg bolus, maintenance 0,1 – 0,2 mg every 60 min). Body tempera

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ture was between 35 – 36° C.

Continuous short latency SEP monitoring with median nerve stimulation on the contralateral wrist was performed throughout the operation (rectangular pulse with 0,3 msec duration, frequency 5,3 Hz, intensity 20 mA). Recording needle electrodes were placed at $C_Z(+)$ – P3, P4 (-) and C_Z-C_2, sensitivity 1,25 mkV/DIV, analysis time 50 msec, 256 sweeps were averaged. The central conduction time (CCT) was calculated as a difference of N20 and N14 peak latencies (17). Special attention was paid on the SEP changes during the first 5 minutes after carotid clamping.

RESULTS:

This analysis comprises 41 monitorings (mean clamping time 38 minutes) with median SEP. The interpretation of the recordings was impossible in 3 cases because of unsatisfactory quality, but none of them had any postoperative ischemic complication.

Minor increase in cortical wave latency and CCT, related to anesthesia, were seen in all patients after induction. In 35 patients no essential SEP-changes were seen after carotid clamping (Table III).

The following SEP-alterations occured in the remaining 6 cases after carotid clamping (Table IV): CCT prolongation with up to 2,5 msec with recovery period ranging from 9 to 21 minutes; change of over 85% of the N20-amplitude for a period of 5 minutes, followed by full recovery after releasing the clamp. No postoperative ischemic deficit in the immediate postoperative period could be attributed to these SEP-changes. The origin of the observed ischemic postoperative complications in 2 cases on the second, respectively fifth day of following CE could not be ascribed to the carotid clamping and intraoperative ischemia, but were postoperative embolic complications.

The detailed analisis in Table IV revealed 2 subgroups.

In the first 3 patients the CTT-prolongation varied from 1,1 up to 2,1 msec without concomitant significant N20amplitude reduction; the latency of the N20 onset remained stable irrespectively of CCT prologion (e. g. increased N20 peak latency) (Figure 1). Besides stability of this first negative deflection of N20 a reversible deformation of N20-P35-complex was prominent. The latter is considered to be a sensitive indicator of insufficient cortical blood flow, but the observed changes were reversible, probably because of adequate collateral blood flow compensation. None of these patients awoke after operation with new neurologic symptoms. One may conclude, that as long as N20 remains identifiable, irrespectively of its flattening and deformation, the stable upward deflection of the wave guarantees the integrity of the somatosensory pathways and primary cortex. In certain circumctances such "dispersed" N20 does not allow peak identification and it remains questionable what is of greater importance – the traditionally measured from peak to peak CCT or N20 or the N20 onset latency.

In the last 3 patients of this group marked amplitude alterations were registered, in one of them with prompt disappearence of N20, Lasting for 4 minutes (Figure 2). No postoperative deficit, attributable to those SEP-changes was observed.

There was no reliable correlation between the preoperative risk grading and the SEP-changes (Table V).

DISCUSSION:

It has been shown experimentally, that in the primate cerebral cortex the cerebral blood flow-threshold for abolition of SEP is 12-18 ml/100g/min (5) Since the difference beetween flow thresholds for synaptic transmission failure and membrane alteration with major ion movement is small (only 5-10 ml/100mg/min), any SEP-abolition following parent vessel is an important warnings. Therefore intraoperative SEP monitoring provides useful information, since firm relationship exists between SEP and cortical blood flow (4, 7, 18, 33). This technique has been used to determine the need for temporary bypass shunting during carotid surgery (13, 20), but it is well known, that the use of shunts produces its own operative difficulties and risks. In our experience, none of the patients showed such pronounced and long-lasting SEP-changes, that could be used as an indicator for vascular shunting.

Our results confirmed the reliabilty and sensitivity of the method in evaluating the adequacy of collateral circulation during carotid crossclamping. The effects of anesthesia upon SEP did not significantly jeopardize their intraoperative usefulness. Like Markand et al, 1984 (20), we always use the signal, obtained immediately before vessel occlusion as a basal value to estimate the effects of carotid clamping. The SEP alteration in 14% of our cases witin the above mentioned ranges (CCT prolongation with up to 2,5 msec, amplitude reduction or transient N20 disappearence not exceeding 5 minutes with recovery period, lasting from 9 to 21 minutes) were not accompanied with postoperative neurological deterioration and reflected an incomplete ischemia with sufficient collateral blood flow compensation. This is in agreement with the conclusions of Ferguson et al., 1982 (11), that the situation in carotid endarterectomy is not one of arrest of circulation, but rather a relative reduction in flow.

As Amantini et al 1987 (1) pointed out, there has not yet been clearly demonstrated, that an increase of N20-peak latency during carotid ischemia reflects a conduction slowing along subcortical pathways. In fact, we similary questioned to some degree the significance of peak to peak measured CCT. Irrespectively of the N20-amplitude decrease, deformation and flattening of the wave the latence of the N20-onset remained stable in all these patients. This N20-P35-complex deformation has been ascribed to increased intracortical temporal signal dispersion, underlying bioelectrical anomalies during cortical ischemia (5).

Taking into account the fact, that the postoperative neurological complication, seen in our 2 patients were of embolic origin and not a result of clamping ischemia, we can conclude, that significant hemodynamic ischemia during temporary carotid occlusion under general anesthesia is a rare event. If SEP is attenuated, but still present and N20-onset latency remains stable, the surgical team should proceed working within the limits of 20-30 minutes (usually enough time), since it is theoretically known, that if left uncorrected for longer period the diminished to critically low levels blood flow would cause permanent neurologic damage (6). A completely unchanged SEP is a sign of adequate blood flow from collaterals, allowing the surgeon to complete without hast the operation (24).

Table I

MEDICAL RISK FACTORS AMONG 44 PATIENTS UNDERGOING CAROTID ENDARTERECTOMY

arterial hypertention	-	19	(43%)
tobacco use	-	12	(27%)
hyperlipidemia	-	8	(18%)
prior documented myocardial infarct	-	7	(16%)
angina pectoris	-	7	(16%)
coronary bypass	-	3	(7%)
peripheral arterial occl. disease	-	3	(7%)
obesity	-	2	(4%)
vitium cordis	-	1	(2%)
chronic obstr. pulmonary disease	-	1	(2%)
no identifiable risk factors	-	14	(32%)

Table II

ASSESMENT OF SURGICAL RISK IN 44 PATIENTS WITH CAROTID ENDARTERECTOMY

(Sundt et al, 1975, 1987)

GRADE	DEFINITION	CASES	
		(No)	Percent
1	Neurologicaly stable, no major medical and angiographical risk, uni- or billateral ulcerative-stenotic carotid disease	9	20,4
2	Neurologicaly stable, no major medical risk, but angiographically defined risks	5	11,4
3	Neurologicaly stable, medical risk factors, with or without angiographic risk factors	25	56,8
4	Neurologicaly unstable, with or without medical or angiographical risks	5	11,4
5	Known acute ICA occlusion	–	
	Total	44	100,0

Table III

CCT VALUES (MEAN ± SD) IN 35 PATIENTS WITH NORMAL INTRAOPERATIVE SEP MONITORINGS

(in % – coefficient of variabilty)

PRE-OP	OP-BEGIN	BEFORE CLAMP	CLAMP	AFTER RELEASE CLAMP	OP – END
6,14±0,73 (18,9%)	6,15±O,63 (10,2%)	6,42±0,86 (13,4%)	6,58±0,80 (12,1%)	6,47±0,86 (13,3%)	6,32±0,92 (14.5%)

Table IV

SURVEY AF THE CASES (NO=6) WITH SEP-CHANGES AFTER CAROTID CLAMPING

CASE		AFTER CLAMPING				RELEASE CLAMP
	L N20-onset	CCT			Change AN20(%)	
		change msec (%)	time to change	time to recover		
2. F.M.	stable	2,1(38%)	8 – 9'	10'	–	like before clamp
9. A.W.	stable	1,1(25%)	1 – 2'	15'	37,5% for 1'	like before clamp
14. G.W.	stable	2,0(41%)	1 – 2'	14'	–	like before clamp
19. B.M.		–	9 – 10'	–	> 90% for 4'	normalize promptly
41. R.A.	increase 1,5 msec	2,5(45%)	1 – 2'	21'	> 85% for 1'	like before clamp
45. B.M.	increase 2 msec	2,0(30%)	1 – 2'	9'	> 85% for 5'	normalize slowly

Somatosensory short latency EPs

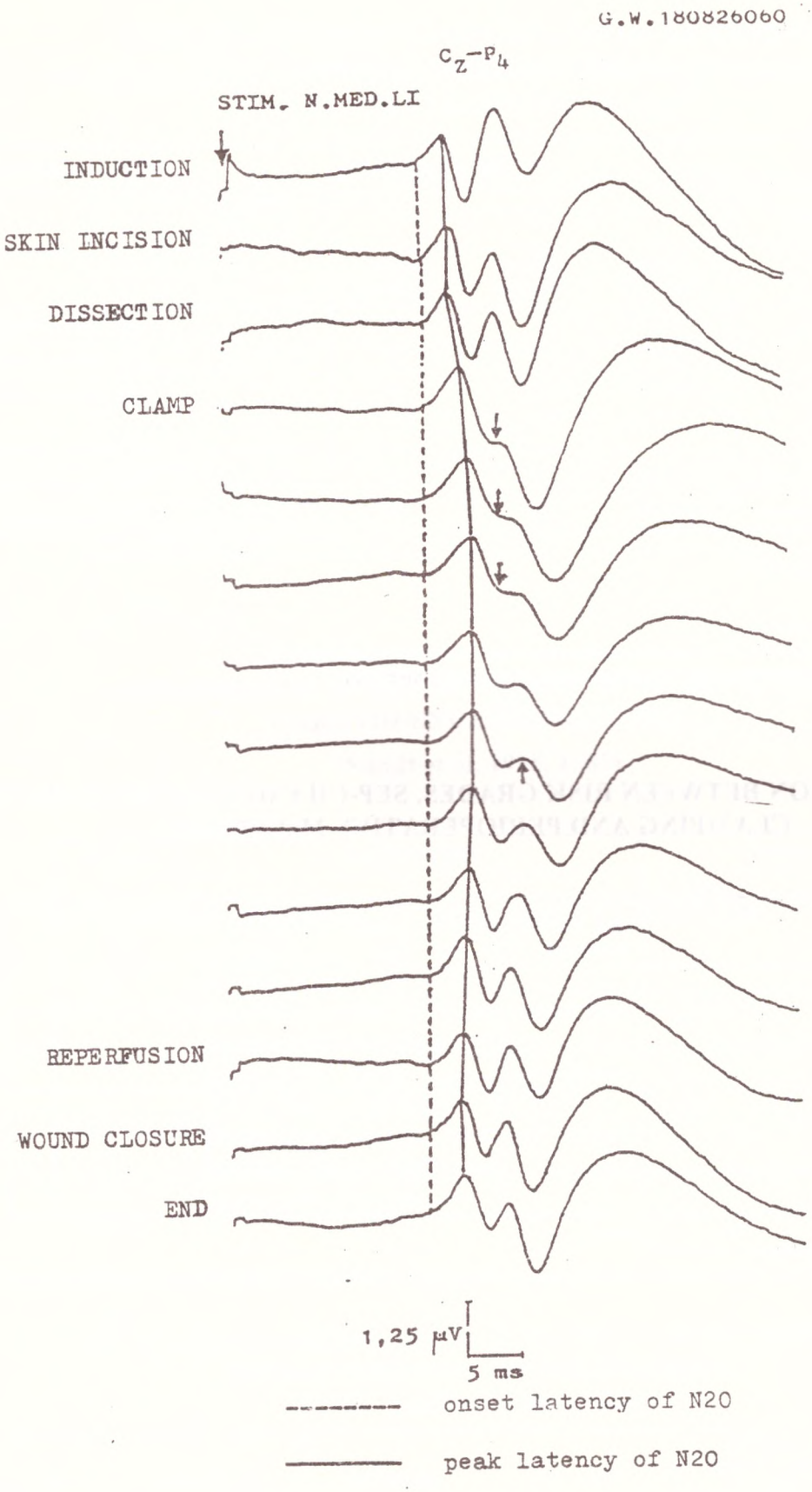
L – latency
latency to onset N20
latency to peak N20 CCT – central conduction time (measured as interpeak latency N14-N20) A – amplitude (peak amplitude measured from baseline)

Table V

CORRELATION BETWEEN RISK GRADES, SEP-CHANGES AFTER CAROTID CLAMPING AND PERIOPERATIVE MORBIDITY

RISK GRADE	No PATIENTS	SEP-CHANGES (No)	PERIOPERATIVE MORBIDITY (No)	
			STROKE	MI
1	9	1	–	-
2	5	–	–	-
3	25	4	2*	1
4	5	1	–	-
Total	44	6	2	1

MI – myocardial infarction
* – one of them without SEP-changes during monitoring time – embolic incident during anesthesia recovery period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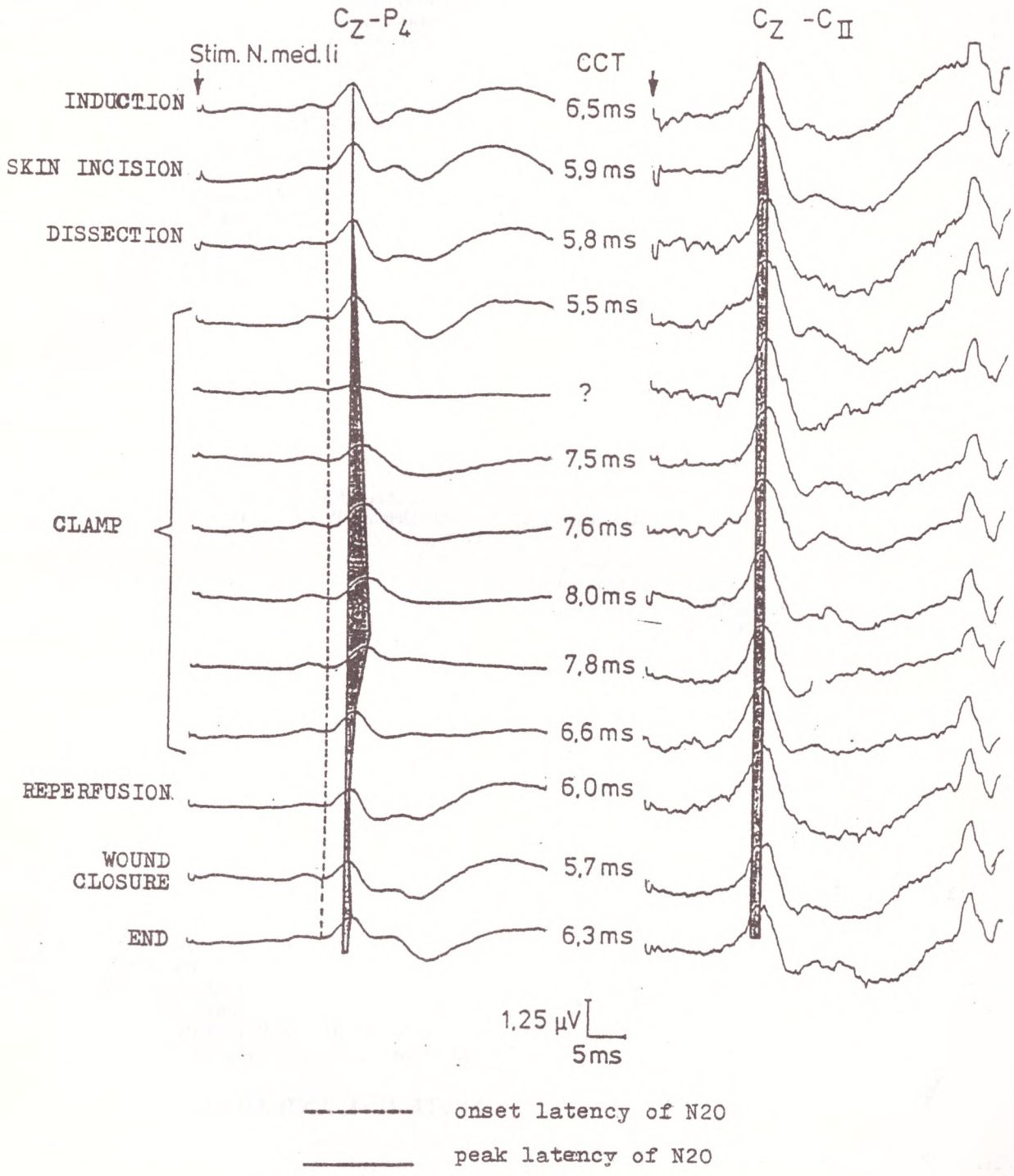
1,25 µV
5 ms
onset latency of N20
peak latency of N20

Fig. 1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6,6ms
6,0ms
5,7ms
6,3ms
1,25 µV
5ms
onset latency of N20
peak latency of N20

Fig. 2

REFERENCES

1. Amantini A., G. de Scisciolo, M. Bartelli, S. Lori, O. Ronchi, C, Pratesi and F. Pinto. Clin Neurol Neurosurg (abstr.), 89, 1987, 2/1 (suppl.) p. 30.
2. Astrup J., B. K. Siesjo, L. Symon. Stroke 12, 1981, 723-725.
3. Beebe H. G., J. M. Pearson, J. J. Coatsworth. Am Surg 44, 1978, 655-660.
4. Boysen G., H. C. Engell, G. R. Pistolese, P. Fiorani, A. Agnoli, N. A. Lassen. Circ., 49, 1974, 1023-1024.
5. Branston N. M., L. Symon, H. A. Crockard, E. Pasztor. Exp. Neurol., 45, 1974, 195-208.
6. Branston N. M., L. Symon. In: C. Barber (ed.) Evoked potentials. Baltimore, University Park Press, 1980, 527-530.
7. Bunegin L., M.-S. Albin, P. Hesel, R. Herrera. J. Cereb. Blood. Flow. Metabol. 1, suppl. 1, 1981, 226-227.
8. Carenini L., E. Bottacchi, M. Camerlingo, G. D. Alessandro and A. Matoli. Clin. Neurol. Neurosurg. (abstr.), 89, 1987, 2/1 suppl), p.122.
9. Committee on health care issues, American neurological assosiation. Ann. Neurol., 22, 1987, 72-76.
10. Cote R. and J. L. Caron. Stroke 20, 1989, 123-126.
11. Ferguson G. G. Stroke, 13, 1982, 287-289.
12. Ferguson G. G. and F. W. Gamache. Jr. In: Smith R. R. (ed.), Stroke and the extracranial vessels, New York, Raveb Press, 1984, 187-202.
13. Gentili F., W. M. Lougheed, K. Yamashiro, H. Ghate, C. Corrado. Can. J. Neurol, Sci. (abstr.), 13, 1986, p. 196.
14. Green R. M., W. J. Messick, J. J. Ricotta, M. H. Charlton, R. Satran, M. M. McBridge, J. A. De Weese. Ann Surg, 201, 1985, 785-792.
15. Grundy B. L. and R. Heros. In: J. Matjasko, J. Katz (eds.). Clinical controverades in neuroanesthesia and neurosurgery, Orlando, Grune & Stratton, 1986, 1-76.
16. Hargadine, J. R. In: Rand R. W. (ed.). Microneurosurgery, St. Louis, CVMosby Co, 1985, 92-110.
17. Hume A. L. and B. R. Cant. Clin. Neurophysiol., 45, 1978, 361-375.
18. Jacobs L. A., S. D. Brinkman, R. M. Morrell, J. G. Shirley, S. Ganji. Am. Surg. 49, 1983, 338-344.
19. Lee K. S., C. H. Davis, J. M. McWorther. J. Neurosurg., 69, 1987, 483-487.
20. Markand O. N., R. S. Dilley, S. S. Moorthy, C. Warren. Jr. Arch. Neurol., 41, 1984, 375-378.
21. Matchar D. B., S. G. Pancker. JAMA 258, 1987, 793-798.
22. Michenfelder J. D. In. Sundt T. M. Jr (ed.). Occlusive cerebrovascular disease. Diagnosis and surgical management, Philadelphia, W. B. Saunders, 1987, 163-169.
23. Morawetz R. B., H. E. Zeiger, H. A. McDowell. Surgery, 96, 1984, 184-189.
24. Nuwer M. R. Evoked potential monitoring in the operating room. New York, Raven Press, 1986.
25. Rowed D. W., M. L. Vilaghy. Can. J. Neurol. Sci., 8, 1981, 235-241.
26. Russ W., W. Krumholz. Anesthesist (abstr.) 33, 1984, p. 475.
27. Spetzler R. F., N. Martin, M. N. Hadley, R. a. Thompson, E. Wilkinson, P. A. Raudzens. J. Neurosurg., 65, 1986, 63-73.
28. Sundt T. M. Jr., D. G. Piepgras, M. J. Ebersold. Clin Neurosurg., 1981, 29: 391-403.
29. Sundt T. M. Jr., B. A. Sandock, J. P. Whisnant. Mayo Clin. Proc. 50, 1975, 301-306.
30. Sundt T. M. Jr. Stroke 14, 1983, 93-98.
31. Sundt T. M. Jr., F. W. Sharbrough, R. E. Anderson, J. M. Messck Jr. In. Sundt T. M. Jr. (ed.). Occlusive cerebrovascular disease. Diagnosis and surgical management. Philadelphia, W. B. Saunders, 1987, pp. 182-190.
32. Sundt T. M. Jr., D. G. Piepgras, M. J. Ebersold, W. R. Marsh, N. K. Fode. In. Sundt T. M. Jr. (ed.). Occlusive cerebrovascular disease. Diagnosis and surgical management. Philadelphia, W. B. Saunders, 1987, pp. 226-131.
33. Trojaborg W., G. Boysen. Electroenceph. Clin. Neurophysiol. 34, 1973, 61-69.
34. Wassmann H., R. Wuelleneber. In: Hartmann A. and Wassmann (eds.). Hirninfarkt. Aetiologie, Diagnose, Prophylaxe and Therapie. Muenchen etc, Urban & Schwarzenberg, 1987, pp. 359-383.
35. Zuccarello M., H. Yeh and J. M. Tew. Neurosurgery, 1988, 445-450.

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SURGICAL TREATMENT OF TUMORS IN THE LATERAL VENTRICLES

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The surgical treatment of tumors in the lateral ventricles sets a number of problems to the neurosurgeon. They are connected with the deep placement of these tumors in the hemisphere, which makes difficult the choice of adequate approach, considering the functional areas of the brain and the deep structures which can be affected while penetrating to the ventricles and removing the tumor.

The present paper aims at investigation the result of the surgical treatment of tumors in the lateral ventricles, their morphological features, disposition in different parts of the ventricle, some special features of the surgical approach and their removal as well as analyzing the causes of death and postoperative complications.

MATERIAL AND METHODS:

The material of the University Alexander Hospital Clinic of Neurosurgery compiled for a period of 10 years [July 1982- July 1992] has been investigated as well as the relevant material of the Neurosurgical department of the University Hospital "Queen Giovanna" [January 1989- July 1992]. Studying this material we found 53 cases with tumors in the lateral ventricles that had undergone operation and had been histologically verified. The number of male and female patients is nearly equal – 27 men and 26 women. The frequency of cases in different age groups is shown in Table 1.

Table 1

DISTRIBUTION OF 53 CASES WITH TUMORS IN THE LATERAL VENTRICLES ACCORDING TO AGE

	<	10 years	–	6 patients
11	-	20 years	–	9 patients
21	–	30 years	–	12 patients
31	-	40 years	-	10 patients
41	-	50 years	-	4 patients
51	-	60 years	-	9 patients
	>	60 years	-	3 patients
			TOTAL	53 patients

The tumor has been placed in the right lateral ventricle in 29 cases, in the left one in 21 cases and in 3 cases both ventricles have been affected. In patient with the tumor in the anterior parts of the ventricle, penetration of the neoplasm through the foramen Monroe in the 3rd ventricle, which can enter into the opposite lateral ventricle. The study does not include cases with tumors in the 3rd ventricle and with a secondary engagement of the lateral ventricle.

The clinical manifestations of the tumors of this location are non characteristic: headache in over 90 % of the cases and more rarely epileptic seizures were initially observed. The dynamics of clinical features is not subject but it is worth mentioning that in 2 cases the beginning was acute, accompanied by subarachnoid hemorrhage. The diagnosis is based on clinical examination, as well as on neuroradiological findings: CT-scan of all patients, angiography of 27 patients and in some cases brain scintigraphy was performed. The CT-scan provides the most valuable information allowing to define the exact location of the tumor in the ventricle, the direction of its growth, the involvement of surrounding structures, the penetration towards the 3rd and the opposite lateral ventricle. The degree of vascularisation (judging by the changes in the density after the introduction of the contrast media), the presence of hemorrhage or intratumoral cyst. Although the CT- image of the mass lesion in the lateral ventricle is not typical, in some cases its morphological nature can be predicted with a considerable degree of certainly. Some CT-findings – tumors of different histological features and location in the lateral ventricle are shown at fig. 1-8. Five of the cases were operated in the emergency, due to bad general condition and rapid focal symptom progression and disturbances of consciousness.

The operative findings shows that the tumors are most frequently located in the frontal horn originating mostly from the mediobasal brain structures, near the foramen Monroe. The tumor distribution in the different parts of the ventricular system is shown in Table 2.

Table 2

DISTRIBUTION OF THE TUMORS IN THE DIFFERENT PARTS OF THE LATERAL VENTRICLES

Frontal horn	21	Trigonum ventriculi(TV)	8
Cella media (CM)	3	Frontal horn and CM	8
TV and CM	6	TV and occipital horn	2
TV and temporal horn	2	Temporal horn	1
Whole ventricle	2		

Involvement of more than one topographical site was observed in 29 cases. In two, the tumor endaged the whole ventricle.

Depending on the location of the tumor and the place of the cortex incision, various operative approaches were used. The anterior transcortical (through the medial frontal gyrus) was adopted in cases with tumors located frontally (frontal horn and frontal parts of CM). The cortex incision was performed trough the middle of gyrus frontalis medius or for reaching in depth to the ventricle cavity a sulcus frontalis superior incision was used. [Fig.9-b] Tumors located in the region of trigonum ventriculi were reached by paramedian parieto-occipital approach.

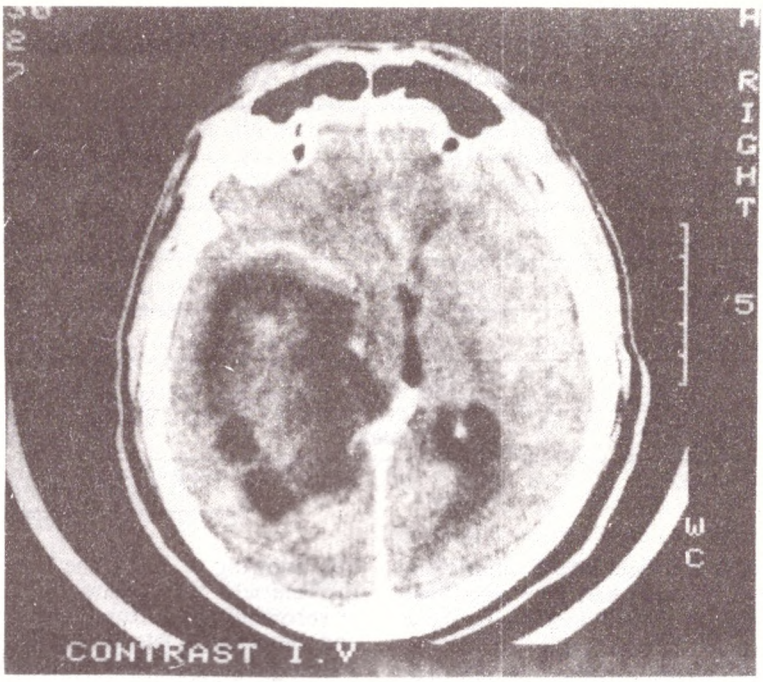


Fig.1: CT-scan tumor (astrocytoma) affecting the trigonum and the temporal horn of the left lateral ventricle

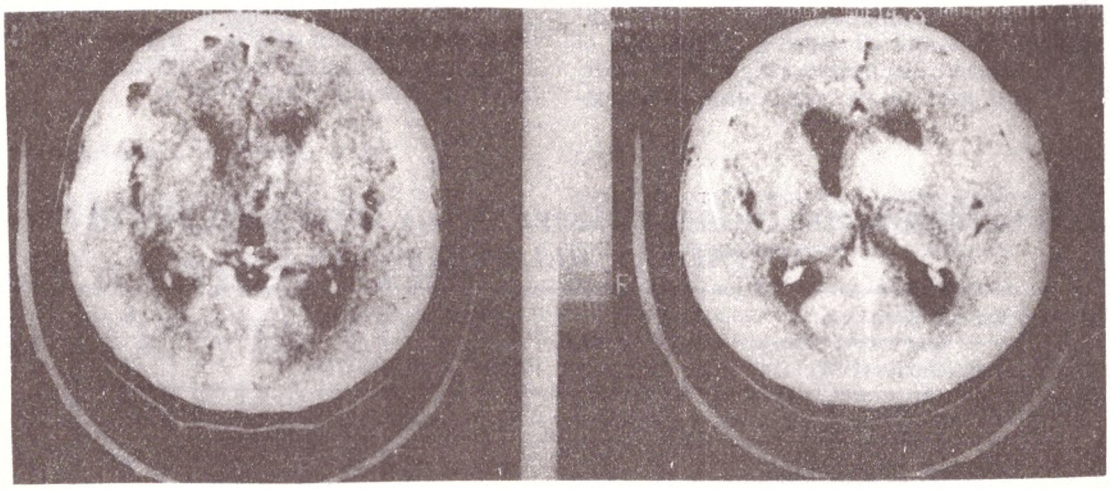


Fig.2: CT-scan shows tumor (subependymoma) involving the septum pellucidum and the frontal horn of the left lateral ventricle

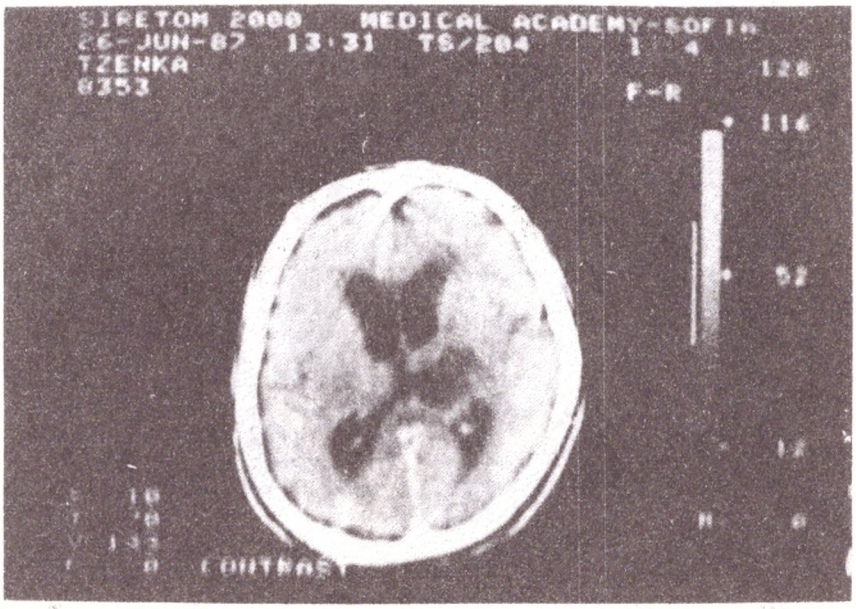


Fig.3: Cystic astrocytoma involving the trigonum of the right lateral ventricle

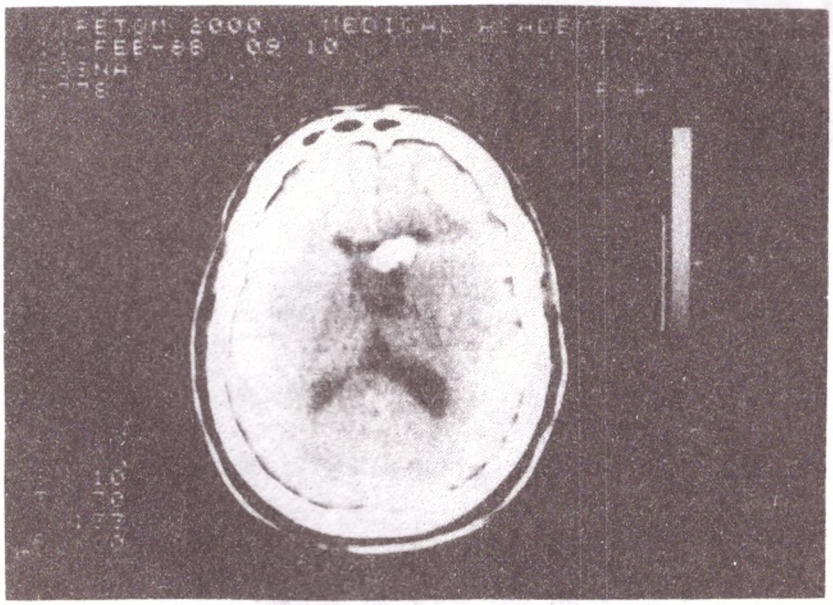


Fig.4: Oligodendroglioma affecting the right frontal horn. Calcification is observed

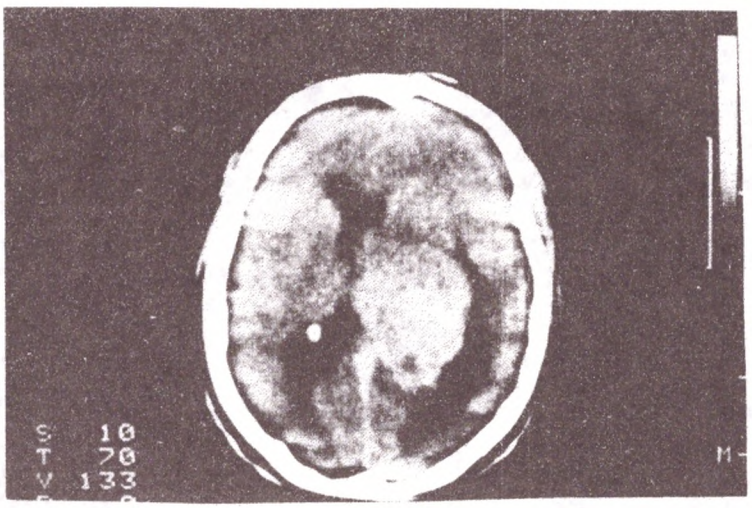


Fig.5: Meningioma in the right trigonum

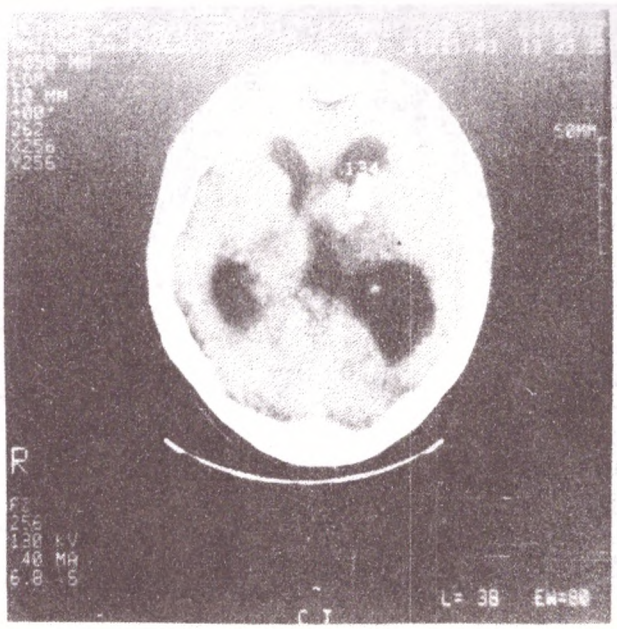


Fig.6-A: Giant cell astrocytoma and calcified nodule in the left frontal horn in a 27-year old woman with tuberous sclerosis

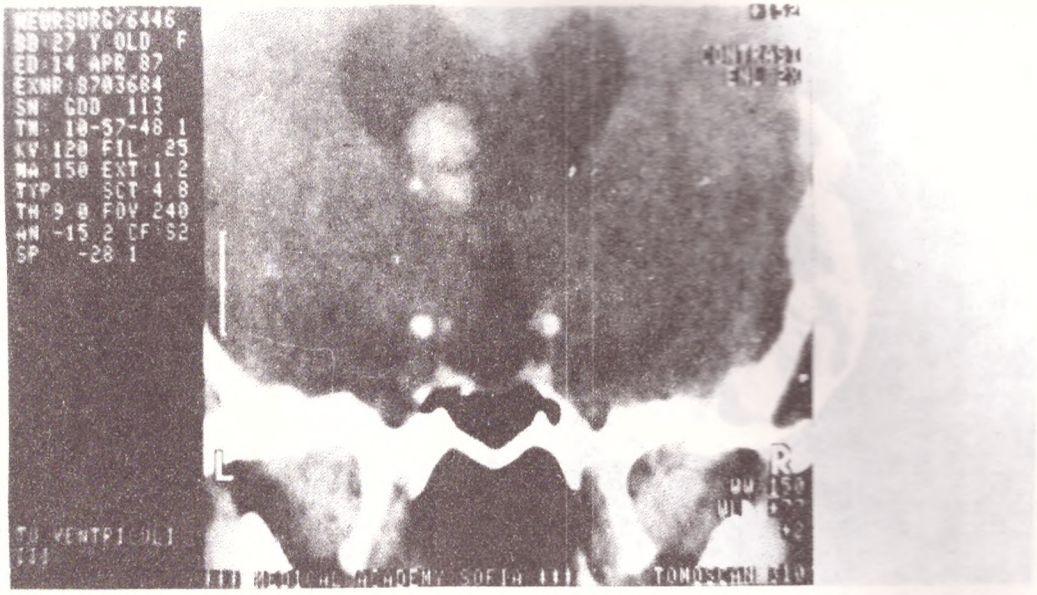


Fig. 6-B: *CT scan coronal reconstruction in the same case as Fig. 6-A*

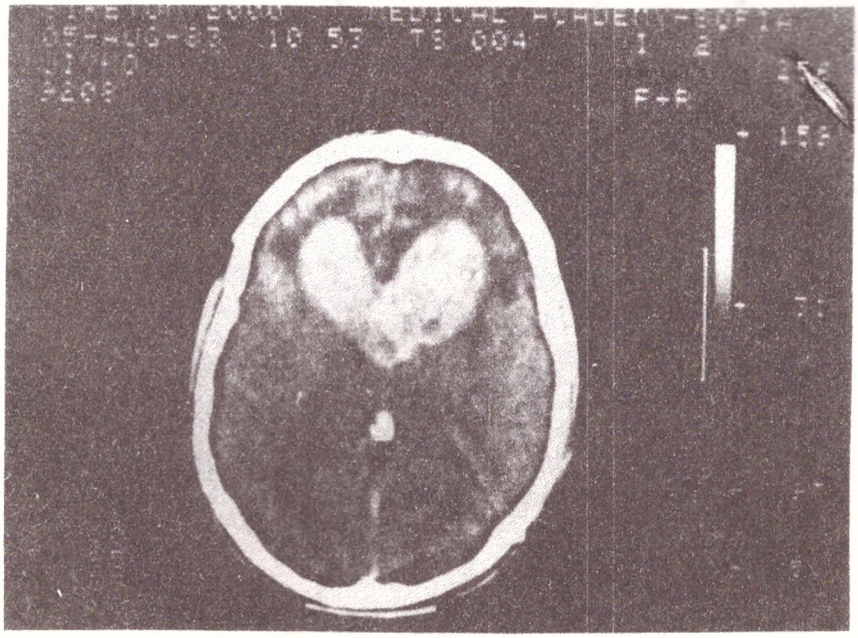


Fig. 7: *Metastases via CSF from pineal germinoma involving the anterior part of both lateral ventricle*

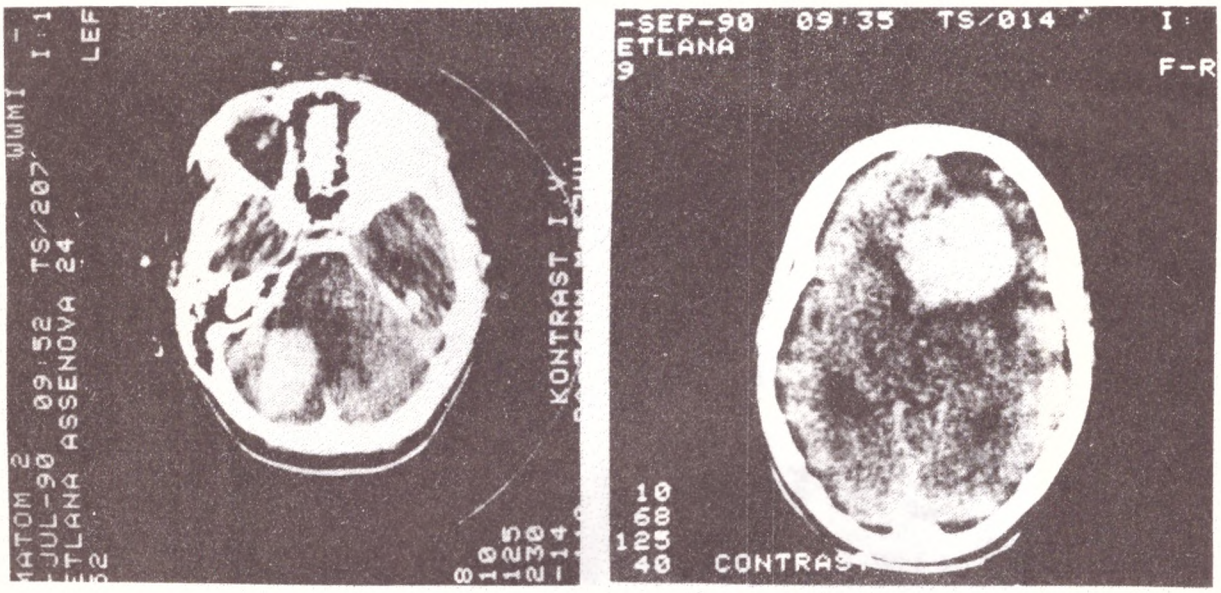
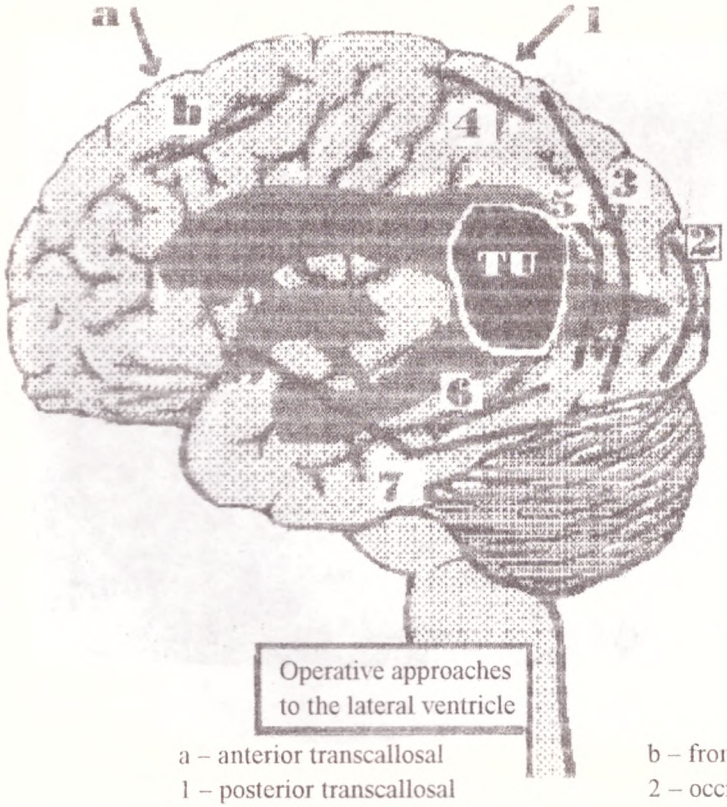


Fig. 8 A,B: Tumor (neuroblastoma) in the left cerebellar hemisphere [8-A] and in the right frontal horn [8-B]



Operative approaches to the lateral ventricle

a – anterior transcallosal
b – frontal transcortical
1 – posterior transcallosal
2 – occipital
3 – occipital lobectomy
4 – parieto-occipital
5 – parieto-temporal paramedian
6 – temporal
7 – temporo-occipital

Fig. 9. Different operative approaches to the lateral ventricle

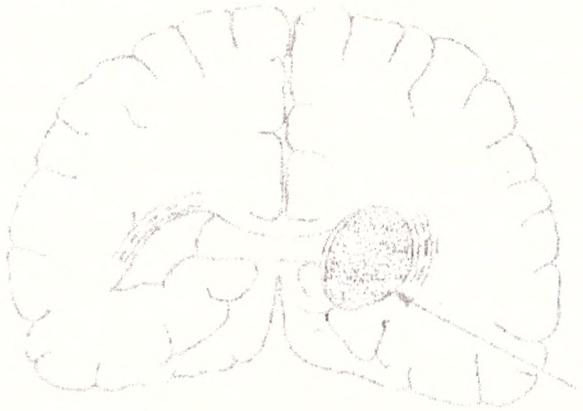


Fig.10. Tumor in the trigone of the lateral ventricle,,relationships, between the tumor, the ventricle wall and the optic radiation (1)

The transtemporal one was applied as well. In the latter care should be taken not to damage the fibers of the optic radiation which are situated closely near the lateral wall of the ventricle [Fif.10]. Approaching through the dominant hemisphere special attention should be paid to preserve the speech center of Wernicke. The transtemporal approach is suitable managing with well vascularised tumors (meningeomas, pl.papillomas) because it permits the access to the tumor base where the supplying vessels enter. All cases required minimal cortical and white matter incision enough wide to allow a sufficient approach. The brain retraction should be extremely careful. The parieto-temporal approach was applied in only one case.

Aiming at complete removal of the tumor at two-stage surgery was performed in three patients and a three-stage one in a single case. Total and subtotal tumor resection was carried out in 15 cases, removal of considerable part of the tumor was achieved in 24 cases, partial resection in 11, and biopsy in 3 cases.

Five patients died in the postoperative period. Three of them had been in grave condition, preoperatively; in two cases the control CT-examination showed intratumoral blood amassment and the ventricular damaging, which made operative revision necessary. Two other patients died more than a month after surgery – a woman with meningitis and marked CSF hyperproduction, cerebral oedema, and tonsilar hernation. Three months after the operation a patient harboring two neuroblastomas, died – one in the left cerebellar hemisphere and the other one expanding in the right frontal horn [Fig.6]. The diagnosis was made in the eight month of pregnancy. In the discussion of the case, in account had taken the presence of the tumors of multiple origin or tumor dissemination through the liquor circulation (the tumor in the frontal horn was identified as a metastatic neuroblastomas). After the successful removal of the cerebellar tumor, delivery by Cesarean section was performed, and with a second operation the other neoplasm was extirpated. After surgery the patient's condition was improving quickly but two months later, the neurological status gradually deteriorated. The control CTexamination was made which did not demonstrate any tumor recurrence. The finding was of considerable periventricular oedema with midline shifting. The patient died a month later. An autopsy a brain herniation without tumor recurrence was established.

The histological features of the tumors are shown in Table 3.

Table 3

Astrocytoma I-IV	13		
		Giant cell astrocytoma (Tuberous Sclerosis)	3
Oligodendroglioma	6	Meningioma	3
Ependymoma	4	Choroid pl.papilloma	4
Ependymoblastoma	5	Choroid pl.carcinoma	1
Gangliocytoma (Neuroblastoma)	1	Glioblastoma multiforme	3
Cavernous angioma	1	Undifferentiated glioma	1
Metastasis (*)	3	Mixed gliomas	3

(*) Metastasis from: Adenocarcinoma ventriculi – 1, Pineal germinoma – 1, Intracranial Neuroblastoma – 1

Among them are three cases of subependymal giant cell astrocytoma, the three of them next to foramen Monroe in patient having tuberous sclerosis.

In the postoperative period, apart from the cases with hemorrhage in the tumor and the ventricle, related to the surgery, delayed bleeding was observed in one patient. Among the complications observed are collapse of the ventricle, acute dilatation of the ventricle, oedema and in two cases meningitis. The possibility of development of an acute hydrocephalus should be borne in mind, as it may have fatal consequences. Such a danger exists in all cases of intraventricular tumors and even more so when no communication with the third ventricle has been achieved. To avoid this complication it is necessary to apply uni- or bilateral drainage.

DISCUSSION

Despite their comparatively low frequence (0.5-1.5 %) of all intracranial tumors and (1.3-9.1 %) in infant age (1,3,4,10,18), tumors in the lateral ventricles, show a number of peculiarities that should be taken into consideration in their surgical treatment: Careful preoperative analysis of the tumor location, degree of vascularisation and identifacation of the supplying vessels, choice of adequate approach and cortex incision, minimal retraction and work in depth.

The introduction of microsurgical technique, ultrasound aspiration and laser made possible the radical removal of these tumors, considerably reducing postoperative mortality.

The analysis of our material reviews a great variety of original and secondary tumors in the lateral ventricle. Other histologicaly different tumors, located intraventicularly, may also be encountered as well as non-neoplastic pathology: lymphomas (5,16), epidermoids (2), specific tuberculomas and nonspecific granulomas (6,14), AVM (11), parasitic cysts (8,13,15) and xantogranulomas of the choroidal plexus (7) etc.

With the application of microsurgical techniques other surgical approaches such as anterior transcallosal, temporo-occipital, anterior frontal, fronto-temporal (9, 12, 17, 19) [Fig.9].

Our study evidences to the fact that the surgery of mass lesions in the lateral ventricle is difficult and challenging. The present condition of the neurosurgical technique, anesthesioligy and intensive postoperative care allow the surgeon to operate successfully with minimal operative risk.

REFERENCES

1. Габровски,Ст. В. кн.: Тумори на централната нервна система в детската възраст.(ред.проф.Вецка) М.Ф.С., 78-83
2. Петков,А.и др. Неврол.Психиатр.Неврохир.,29, 1990,4 : 70-76
3. Черепанов,А.Н.,Е.М.Довгяло-Галагаян: Вопр.Нейрохир., 34,1970,4: 55-58
4. Шелия,Р.Н.:Опухоли желудочковой системи. Мед.М., 1973
5. Baumgardner,J.E. et al.: J. Neurosurg., 73, 1990, 2 : 206-211
6. Berthier,M. et al. : Neuroradiology, 29, 1987, 2 : 163-167
7. Brück,W. et al.: J. Neurosurg., 75, 1991, 2 : 324-327
8. Duplesis,E. et al. : Neurochirurgie, 34, 1988, 4:275-279
9. Heiskanen,O.: Child's Brain, 3, 1977, 2, 69-78
10. Koos,W.T., M.H. Miller: Intracranial Tumors of Infants and Children, Springer-Verlag, Stuttgard, 1981
11. Laine,E. et al. : Neurochirurgie, 27, 1981,3: 147-160
12. Lapras,C.: Adv.Tech.Stand.Neurosurg.,vol.II 1984, 3: 103-167
13. Mircevski,M. et al.: Neurochirurgie, 31, 1985, 5: 425-428
14. Penar,P.L. et al. J.Neurosurg.,68, 1988, 1,:145-148
15. Rhee,K.S. et al.: J.Comp.Ass.Tomogr., 11, 1987, 4: 598-601
16. Shibata,S.: Neurosurgery, 25, 1989, 1,: 14-19
17. Spencer,D.D., W.F. Collins: In Operative Neurosurgical Technics (Eds.: Shmidek, W.H.Sweet), vol.1, Grune & Stratton, N.Y., pp. 561-574
18. Tien,R.D. : Amer.J.Roentgenol., 157, 1991, 3-1283 -1290
19. Tirmurkaynak,E. et al. : Neurosurgery, 19, 1986, 5 : 685-723

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ALUMINUM CONCENTRATIONS IN SERUM OF PATIENTS WITH INTRACRANIAL TUMORS

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INTRODUCTION

Aluminum overload in brain has been established in cases of Alzheimer's disease, amyotrophic lateral sclerosis, epilepsy and Parkinsonian dementia (6, 8, 9, 10, 11, 14, 15, 16, 17). This trace element may cause encephalopathy, osteodystropy and anemia in persons with severe renal insufficiency on chronic hemodialysis treatment (1, 3, 4, 7, 12, 13). In this paper the results of the investigation of aluminium serum levels in patients with different intracranial tumors are reported. In the same patients the calcium serum concentration are measured, because this element is known as an antagonist of the aluminium in the human organism.

MATERIALS AND METHODS

The total number of the investigated patients with intracranial tumors is 71. Their distribution to the histological type of the tymor is shown on Table 1. The control group consists of 15 apparently heatly individuals

The blood samples were obtained by venopuncture with stainless needles and transferred onto 10-ml polypropylen sample tube, centrifuged at 100 rpm for 10 min. and the serum was transferred and diluted with matrix modifier in 3 ml polypropylene cuvete. The serum aluminium was determined by electrothermal atomic absorption spectrometry – a modified method of Bettinelli and Baroni (2). An atomic absorption spectrophotometer "Perkin-Elmer-Zeeman 5000 was used. The calcium in the serum was determined directly by a flame atomic absorption spectrometry.

All glassware and plastic tubes used for sampling and storage were soaked overnight in 20% nitric acid solution and rinsed with deionized water from "Millipore Milli Q Reagent Water System". All glassware and plastic tubes were checked for aluminum before use.

RESULTS

The results of aluminum serum levels in studied groups are shown on table II. The serum aluminum is significantly increased ($p < 0,01$) in malignant gliomas and meningiomas. In the small group of astrocytomas the difference is also significant ($p < 0,05$). The calcium serum concentrations in the patients with intracranial tumors do not show any change (Table III).

DISCUSSION

Nowadays the role of the aluminum in pathogenesis of Alzheimer's disease is widely accepted. G. J. Nailor et al. have disclosed in seven cases an elevated content of aluminum in whole blood, serum and hair (11). A. Van Rhijn et al. have found higher serum aluminum in senile dementia of Alzheimer's type compared with multiinfarkt (1 6). F.M. Corrigan et al., on contrary, depict slightly concentrations of aluminum in blood serum of Alzheimer's patients compared with healthy controls (5). Davidson and Ward have estimated 38 trace and bulk elements in the serum from 19 patients with recent onset of epilepsy. The concentrations of aluminum, strontium and zinc haave been significantly higher (8). J.M.H. Howard considers this finding of increased serum aluminum concentrations in epilepsy a nonspecific change, because slightly higher concentrations have been reported in depression, memory loss and in children with hyperactivity and learning disorders (9).

B. Winterberg et al. have studied ten patients with brain tumors, who have been treated with an aluminum rich antacid (Maalox 70) for ten days prior to the operations. The analysis of the brain tissue removed has revealed twofold higher aluminum levels compared with a control group of patients, who have received aluminum-poor drug. (18).

Our results show a significantly higher content of aluminum in the serum of patients with gliomas and meningiomas. It is not acceptable that aluminum may play a causal role in the appearence of these intracranial tumors. The aluminum is an element widely distributed in the human environment and some contamination with this metal is inevitable for the men. Obviously, this clinical phenomenon is connected with the impaired brain metabolism of the trace elements and the aluminum overload is characteristic for many diseases of the central nervous system.

Table I

DISTRIBUTION OF THE INVESTIGATED PATIENTS ACCORDING TO THE HISTORY OF THE TUMOR

Group	No
Glioblastomas	15
Malignant astrocytomas	17
Astrocytomas	5
Meningiomas	11
Hypophyseal adenomas	10
Acoustic neurinomas	9
Cancer metastases	4

Table II

ALUMINUM CANCENTRATION IN SERUM OF THE PATIENTS WITH INTRACRANIAL TUMORS

Group	Al µg/l (x + s)	t-test of Student
Glioblastomas	34,87 ± 25,96	$p < 0,01$
Malignant astrocytomas	25.21 ± 14,17	$p < 0,01$
Astrocytomas	36,93 ± 25,11	$p < 0,05$
Meningiomas	26,19 ± 16,06	$p < 0,01$
Hypophyseal adenomas	21,25 ± 16,17	N.S.
Acoustic neurinomas	14,49 ±10,28	N.S.
Cancer metastases	9,03 ±4,75	N.S.
Healthy controls	11,67 ± 6,52	

Table III

ALUMINUM CANCENTRATION IN SERUM OF THE PATIENTS WITH INTRACRANIAL TUMORS

Group	x mmol/l	Range
Glioblastomas	2,32	1,95 – 2,62
Malignant astrocytomas	2,38	2,11 – 2,87
Astrocytomas	2,28	2,03 – 2,66
Meningiomas	2,29	2,13 – 2,51
Hypophiseal adenomas	2,40	2,09 – 2,57
Acoustic neurinomas	2,33	2,15 – 2,40
Cancer metastases	2,34	1,85 – 2,80
Reference values		2,12 – 2,62

LITERATURE

1. Alfrey, A.C. Ann. Rev. Med.,1978, 29, 93-98
2. Bettinelli, M., N. Baroni. Analyst, 1985, 110, 19-22
3. Canavese, C., A. Pacitti, M. Salomone et al. Trace Elem. Med., 1989, 6, 104-107
4. Channon, S.M., S. Arfeen, M.K. Ward Ibid., 1988, 5, 154-157
5. Corrigan, F.M., J.D. Finlayson, G. Stevenson et al. Ibid., 1987, 4, 117-119
6. Crapper-McLachlan, D.R. Neurobiol. Aging, 1986, 7, 525-532
7. Crapper, D.R., S. Quittkat, S.S. Krishnan et al. Acta Neuropathol., 1980, 50, 19-24
8. Davidson, D.L.W., N.I. Ward. Epilepsy Res., 1988, 2, 323-330
9. Howard, J.M.H., Clin. Chem., 1984, 30, 1722-1723
10. Lewis,D.V., N.Mutsuga, W.H. Schnette, J. Van Buren. Epilepsia,1977, 18, 499-505
11. Naylor, G.J., A.H.W. Smith, A. McHarg et al. Trace Elem.Med., 1989, 6, 93-95
12. Parajon, J.P., E.B. Gonzalez, J.B. Cannata, A.S. Medel. Trace Elem. Med., 1989, 6, 43-46
13. Parkinson, I.S., M.K. Ward, T.G. Feest et al. Lancet, I, 1979, 9, 406-409
14. Perl, D.P., D.C. Gaidusek, R.M. Garruto. Science, 1982, 217, 1053-1055
15. Trapp, G.A., G.D. Miner, R.Z. Zimmermann et al. Biol. Psychiatry, 1978, 13, 709-712
16. Van Rhijn, A., F.M. Corrigan, N.I. Ward. Trace Elem.Med., 1989, 6, 24-26
17. Wetterling, T. Fortschr. Neorol. Psychiat., 1989, 57, 1-13
18. Winterberg, B., H. Bertram, N. Rolf et al. J. Trace Elem.Electrolytes Gealth Dis., 1987, 1, 69-72

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Anderson, D.C, Kozak, A.J: Brain Abscess. In: Clinical Neurology (R.J. Joynt, ed.), vol.2, Ch.25, pp. 7-43. J.B. Lippincot, Philadelphia, 1991.

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