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Congress of
NEUROLOGICAL
SCIENCES** **BRUSSELS**

Volume II

NEUROLOGICAL SURGERY

Edited by

**Dr. LUDO VAN BOGAERT
and Dr. J. RADERMECKER**



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Edited by

**L. van BOGAERT
J. RADERMECKER**

**FIRST INTERNATIONAL CONGRESS OF
NEUROLOGICAL SURGERY**

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PREFACE

It was possible to publish the complete texts of the reports, concerning the invited discussions, before the opening of the First International Congress of Neurological Sciences, by reason of a subsidy from the National Institute of Neurological Diseases and Blindness. They were distributed to registered members of the Congress. A certain number of copies were obtained after the Congress by members or experts who were not subscribers, on application to the publishers, the Acta Medica Belgica.

The résumés of all communications presented and received before the 15th May 1957 were published before the Congress and edited by Excerpta Medica. Each member received those relating to the subject for which he was registered. A certain number of copies concerning other subjects were obtained by those requiring them from our publishers, during and after the sessions.

During the administrative sessions which were held on the closing day of the First International Congress of Neurological Sciences, we were commissioned by the organisers of the various sections to publish the complete texts of communications concerning the topics and open communications, those discussions and communications received late, and those discussions not yet published.

We have added to this the complete reports and discussions of the special session organised on the opening day of the Congress by the International League against Epilepsy, the résumés of which were not published in those papers circulated in advance by Excerpta Medica. These documents have been included in the volume of the Fourth International Congress of Electroencephalography and Clinical Neurophysiology.

The original texts of the Round Table Conference, under the auspices of the C.I.O.M.S. and the Congress, dedicated to the problem of the Future of Neurology, and entitled 'Neurology at the Crossroads', the speeches delivered at the official opening session in the presence of His Majesty the King, those given at Louvain at the Centenary Celebration of Arthur Van Gehuchten and those at the Congress Banquet have been inserted in Volume V entitled 'Joint Meetings'.

An appeal was sent a few days after the Congress to all authors of reports or discussions. Many of them sent us modified or revised texts or supplementary figures. Some did not reply at all. We are publishing the texts which were received.

Many texts were written by their authors in a language other than their mother or habitual tongue. This is a obligation inherent in Congresses such as ours.

On the other hand, the medical editors complain more and more of a lowering standard of literary expression and a lack of grammatical care in the papers received. In order that the quality and intelligibility of our publication should be maintained we have often been obliged to modify the

texts submitted or to translate them into one of the three languages used at the Congress. We have only made those changes which were indispensable, being more concerned with retaining the exact thought of the writer rather than giving impeccable literary style. This was an additional source of work and caused an unforeseen delay to this supplementary publication. This is also the reason for not having been able to submit the proofs to the authors, nor send for those figures mentioned in the texts and not enclosed, or the missing bibliographies. This would have delayed publication for several years and, in view of the rapid development in the various subjects, would have affected their pertinence and significance.

We have done our best not to depart from the author's original ideas. If we have not always succeeded we ask you to excuse us.

We should like to thank Captain Maxwell and all those connected with Pergamon Press Ltd., for their work and time without which even an attempt at publication could not have been considered. We wish in particular to thank Miss M. Fleming for her valuable assistance in helping with the completion of this task.

Ludo van Bogaert
Joseph Radermecker

Editors

PREFACE

Les textes complets des Rapports, ceux des Discussions sur Invitation avaient pu être publiés avant le début du Premier Congrès International des Sciences Neurologiques grâce à un subside du National Institute of Neurological Diseases and Blindness. Ils furent distribués aux membres régulièrement inscrits au Congrès. Un certain nombre d'exemplaires ont été obtenus après le Congrès par les intéressés non inscrits, sur demande auprès de nos Editeurs, les Acta Medica Belgica.

Les résumés de toutes les communications présentées et reçues avant le 15 Mai 1957 avaient été publiés avant le Congrès dans les fascicules édités par Excerpta Medica. Chaque membre les a reçus pour la discipline où il était inscrit. Un certain nombre d'exemplaires concernant d'autres disciplines ont été obtenus par ceux qui le désiraient auprès de ces Editeurs, pendant et après les Sessions.

Lors des séances administratives qui ont eu lieu le jour de la clôture du 1er Congrès International des Sciences Neurologiques, les Bureaux des différentes disciplines nous ont confié la tâche de réaliser la publication des textes complets des communications concernant les thèmes et des communications libres, ceux des discussions et communications reçues trop tard, ceux des discussions non encore publiées.

Nous y avons ajouté les Rapports complets et les discussions de la Séance spéciale organisée le jour de l'ouverture du Congrès par la Ligue Internationale contre l'Epilepsie, rapports dont les résumés n'ont pas été publiés dans les fascicules précirculants des Excerpta. Ces documents sont insérés dans le volume du IVe Congrès International d'Electroencéphalographie et de Neurophysiologie clinique.

Les textes originaux de la Conférence de table ronde consacrée, sous les auspices du C.I.O.M.S. et du Congrès, au problème de l'Avenir de la Neurologie, les allocutions de la Séance Solennelle d'Ouverture en présence de Sa Majesté le Roi, celles prononcées à Louvain lors de la Célébration du Centenaire d'Arthur Van Gehuchten et au Banquet du Congrès sont insérés dans le volume V consacré aux 'Journées Communes'.

Un appel avait été envoyé au lendemain du Congrès à tous les auteurs de rapports et de discussions. Beaucoup d'entre eux ont envoyé des textes remaniés ou augmentés avec des figures supplémentaires. D'autres n'ont pas répondu. Nous publions ce que nous avons reçu.

Beaucoup de textes ont été rédigés par leurs auteurs dans une langue qui n'était pas leur langue maternelle ou leur langue véhiculaire habituelle. C'est là une servitude inhérente à des Congrès comme les nôtres.

D'autre part, les éditeurs médicaux se plaignent de plus en plus d'une baisse dans la pertinence de l'expression littéraire et dans le souci grammatical des travaux reçus. Pour conserver à la publication que nous avons entreprise une certaine qualité et surtout une intelligibilité

suffisante nous avons été obligés souvent de remanier les textes soumis ou de les faire traduire dans une des trois langues véhiculaires principales de nos Congrès. Nous n'y avons introduit que les changements indispensables, plus soucieux de ne pas altérer la pensée exacte des auteurs que d'apporter un texte linguistiquement impeccable. Ce fut une source de travail supplémentaire et l'origine d'un délai imprévu dans la réalisation de cette publication complémentaire. C'est aussi la raison pour laquelle nous n'avons pas pu soumettre aux auteurs les épreuves de leur contribution, ni réclamer les figures annoncées dans le texte et non envoyées ou les bibliographies manquantes. Ceci eut remis la parution à plusieurs années et aurait - en raison de l'évolution rapide de nos disciplines - enlevé à beaucoup de travaux leur actualité et, de ce chef, leur portée.

Nous avons fait de notre mieux pour ne pas trahir la pensée des auteurs. Qu'ils nous excusent si nous l'avons fait.

Nous tenons à remercier le Capitaine Maxwell et le personnel d'edition de Pergamon Press à tous les degrés de la hiérarchie du sacrifice inconditionnel qu'ils nous ont consenti de leur travail et de leur temps, sacrifice sans lequel la tentative même de cette publication ne pouvait être envisagée. Nous tenons à remercier tout spécialement à cette Miss M. Fleming de son aide si précieuse.

Ludo van Bogaert
Joseph Radermecker

Editeurs

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THROMBOSIS IN CEREBRAL ANGIOMA (AN UNUSUAL CASE)

MALFORMATION VASCULAIRE DU CERVEAU (UN CAS INHABITUEL)

F. COLUMELLA, G. B. DELZANNO, G. C. NICOLA
Milan, Italy

Spontaneous thrombosis of intracranial angioma seems to be little known. We could find in the literature only three cases similar to ours.

Noran (1) mentions that in only one case of cavernous angioma (the 10th of his series) a thrombosis existed of the venous lacunae. It is not clear whether an arteriography was carried out. The patient was operated on for intracranial hypertension, jacksonian epilepsy, hemiparesis and aphasia. The angiomatous mass was found in the left anterior parietal region.

Paterson and McKissock (2) quote more extensively two of their cases in which the thrombosis of the angioma was misleading in making the diagnosis. In the first case both arteriography and encephalography were negative and the malformation was accidentally found during lobotomy in a young epileptic, who, however, had a frontal left focus in EEG. Histologically the mass appeared to be an angioma, the bigger vessels were obstructed by connective tissue. The thrombosis was thus of old date. The other case suffered from serious temporal epilepsy with corresponding EEG focus, which in the X-ray pictures showed calcifications in the left temporal region. The arteriograms were negative and the ventriculography demonstrated slight dislocation of the ventricular system, typical for temporal expanding process.

Our case was a boy of 13 years when he came under observation. Family history was negative. He was the first child. Birth at the end was instrumentally induced. Weight at birth - 4.5 kg. He always seemed older than his age. Precocious mental development (at the age of 11 months he spoke and walked); at 8 years tonsils and adenoids were removed. At the age of 10½ years, slight headaches started, always traced to the left temporal region. He got progressively obese (at the age of 11 years his weight was 50 kg.). Thyroid treatment and injections of 500 I.U. of gonadotrophine was given on alternate days. After the 2nd injection a pulsating headache in the left anterior temporal region and marked somnolence started: the patient slept all day, was easily awakened, remained awake for meals, then vomited and slept again. The tests for motor deficiency were positive on the right side, papillary oedema was present, prevalent on the left side. X-rays of the skull showed that the digitate impressions were more marked than usual, and EEG (Fig. 1) showed good alpha rhythm, well organised for the age of the subject, mixed with theta inferior rhythm and with a real delta rhythm of high voltage. The stop-reaction was present and normal everywhere. The exploration with small distance between the electrodes, especially in advanced hyperventilation, presented a slow and nearly continuous high voltage activity, with alpha waves interfering, over the whole of the left

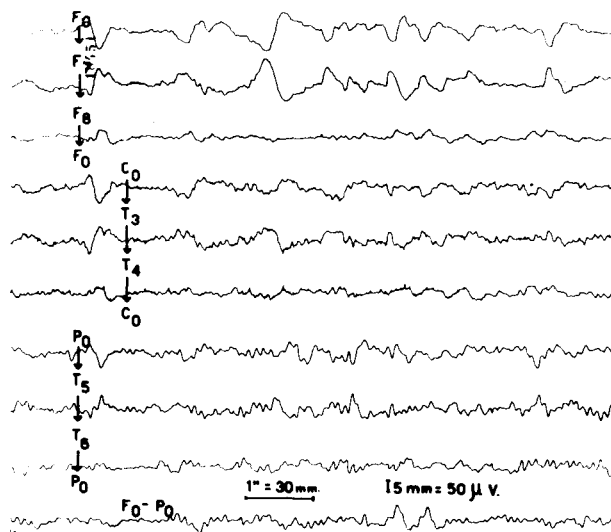


Fig. 1

EEG during the period of intracranial hypertension.

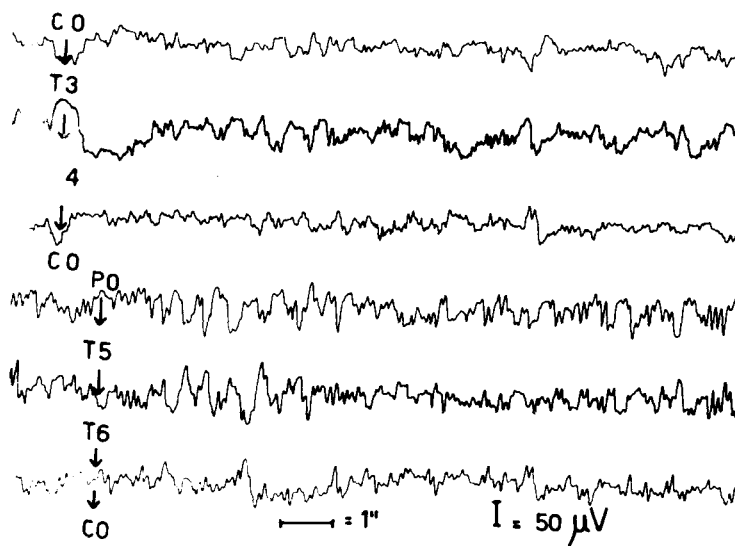


Fig. 2

EEG a few days before operation (epileptic period).

hemisphere, and particularly over the temporal region, where the rhythm was between 3 and 1.5 c/s.

Injections of hypertonic solutions made all clinical signs disappear within a week, except the papillary oedema. The latter after six months of marked oscillations definitely disappeared. The patient was then in good health.

One month before entering hospital, that is, 14 months later, the patient had six epileptic seizures, always of the same type, beginning with a sweetish-bitter taste, followed by rotation of the head and eyes towards the right side; at this moment the patient lost consciousness and had a short series of clonus of the right half of the face and of the right arm. There were no symptoms after the seizures. The EEG at this stage is shown in Fig. 2: diffuse disorganisation of alpha rhythm; many diffused slow rhythms (mainly delta) foremost at the left side; in the left temporal region these slow waves appeared as a mirror image (phase contrast) of each other, in addition to sharp waves, also in phase contrast.

On admission to hospital, the boy, notwithstanding his 13 years, had the aspect of an individual of 16, of athletic build, and showing no true morphologic symptoms of endocrine disturbances.

A.P. = 115/75, P' = 56, T = 36.5°C.

Physically normal, in accordance with his age; speech and movements normal; left-handed, except for writing.

Nothing abnormal was found in the head, neck, thorax, abdomen and genitals.

Cranial nerves: 1-3-4-5-6-8-9-11-12 normal.

Visus 10/10 in 00; fields normal; ocular fundus with well-defined rosy-pink papillae, arterial vessels normal, venous vessels slightly congested. Slight static and dynamic, prevalently mimic, facial weakness in the lower right part.

At the Romberg test, sensibilised: sufficiently compensated right pulsions.

Tretter-reaction: slight rotation to the right.

Babinski-Weill: star-shaped walk, because of deviation to the right.

Strength: symmetrical. Very slight pronation of the right arm at the tests for static weakness.

Tonus, trophism and sensibility: normal.

Cerebellar tests: some hesitation in the index-nose test and slight hypodiadocokinesis to the right.

Absence of abdominal, cremasteric, medio-plantar and right achilleous reflexes; Chaddock reflex present on the right side; marked prevalence of the left patellar reflex; plantar cutaneous reflex in flexion.

Sphincters under control.

X-rays of the cranium: small, well-defined sella; slight increase of digitate impressions.

Left carotid angiography. A.P. projection (Fig. 3): displacement to the right of the anterior cerebral artery, prevalently in the lower part of the vertical portion, which gradually returns to its normal position at the level of the free margin of the falx, that is, without formation of a step. M2-A2 space slightly enlarged. Above M1 and M2 a thin artery projects

in an abnormal position (arrow): it comes from the extremity of M_1 , forms an entire semi-circle open laterally, then disappears into the shadows of the Sylvian group. No displacements are observed in the venular and venous phase, neither are there any signs of pathological circulation. Lateral projection (Fig. 4): slight amplification of the course of the anterior cerebral artery around the corpus callosum; suspected lowering of the initial portion of the Sylvian group; two ascending frontal arteries (arrows) develop in a pincer-wise manner, projecting so as to embrace A_2 . The capillary phase is empty. In the venous phase the concavity of the middle cerebral vein is increased. Absence of pathological circulation in all phases.

Encephalography in the A.P. projection, (Fig. 5), the frontal horns, the medial cells and the third ventricle are displaced to the right. The left medial cell appears reduced in volume in the latero-lateral direction and assumes a falciform aspect because of the reduction of the superior external angle. The left temporal horn (arrow) is only slightly contrasted but shows a definite external displacement. The third ventricle is vertical. The peripeduncular cisterns are contrasted: the right one is laminar, the left is slightly lowered and verticalised and is deformed into an S shape.

Azotemia	=	g. 0.38 ‰
Glycemia	=	g. 1.16 ‰
Urine	=	normal
Haemoglobin	=	100%
Red blood corpuscles	=	5.190.000
Globular value	=	0.98
Leucocytes	=	4,800
Coagulation time and bleeding time	=	normal

In consideration of the anomalies found in the history of the illness and in the angiographic pictures, it was decided to carry out an exploratory operation. A wide left frontal flap was made, so as to uncover the initial part of the Sylvian scissure and the temporal pole as well. The brain appeared pale, with oedematose convolutions in the infero-posterior part of the frontal pole and in the temporal pole. The arachnoid of the Sylvian scissure, which showed a marked rusty colour, was incised, but it was not possible to see any other pathological manifestation, even in the depth of the scissure. An exploratory puncture was then made in correspondence to the foot of the third frontal convolution, where the cerebral oedema seemed to reach its maximum. 2-3 ml. of very dark, dense blood, were drawn, with fragments of cerebral substance of necrotic aspect. A longitudinal incision of 3 cms was then made in the same place. At two cms depth, towards the medial line, a cavity of an old hematoma was discovered, of the size of a small walnut, with hemosiderotic walls. Its postero-medial wall was constituted of formations which at first sight seemed to be blood clots, but which, upon closer examination, proved to be of a composite nature: they were similar to varicose veins, amalgamated with a very fine stratum of softened cerebral tissue, mixed with dark blood and small yellowish

crystals. This pathological mass was completely removed, bit by bit. The result was a cavity as large as a hen's egg, developed posteriorly and medially, that is, towards the basal ganglia. During the removal of this pathological tissue, a vertically developed artery was encountered, which crossed the pathological tissue and therefore seemed to be part of the lesion. It was coagulated and removed together with the lesion. The surgeon considered that this was the abnormally situated artery, which well appeared in the antero-posterior arteriogram. Around the pathological tissue, the cerebral tissue was found to be soft; in some points between it and the pathologic mass a thin necrotic layer was found, in other points a thin layer of dark and very thick blood, containing minute yellowish crystals.

Post-operative course.

An immediate, but slight aggravation of the inferior facial weakness; no speech disturbances. On the third day, short periods of agitation. On the seventh day the patient presented infrequent and momentary difficulties of speech. At 8.30 a.m. on the 8th day, a sudden, complete motor aphasia occurred, as well as small continuous masticatory movements, without loss of consciousness. After an hour and a half, slight facial clonus occurred with bradycardia ($P = 52$) and dislevelling of the right limbs in the tests for static weakness. At 3 p.m. there was a sudden disappearance of aphasia, of involuntary movements and of paresis: pulse rate 62. At 6 p.m., an EEG was made which showed (Fig. 6) a completely disorganised electrical activity, constituted by very slow and wide waves at 1-2 c/s, of irregular morphology, distributed non-synchronously over all the derivations, in non-rhythmic and non-symmetric succession, irregularly interrupted by frequencies of lesser amplitude at 3-5 c/s. During the recording of the large coronary derivations, there was a non-symmetric activity with a notable amplification of the potentials on the left temporal region, where the delta type elements with monorhythmic character often take on a paroxysmic and moderately rhythmic course.

The following day a slight bradycardia persisted ($P = 62$) while the lower right facial weakness diminished to a degree below what it had been before the operation. On the 18th day, the EEG (Fig. 7) showed, in a state of rest, a basic activity of medium voltage with rhythms of 8-9 c/s gathered into moderately well-modulated sequences, unstable over the left hemisphere. Numerous isolated, slow and rather wide elements were detected on it, often assuming a slow, sharp morphology in the temporal and central regions. In these regions the voltage was higher and the slow rhythms were more abundant, monophasic and irregular; infrequently, on the frontal region, they gathered into rhythmic groups with a parasinusoid aspect. During hyperventilation these phases spread to the right frontal region, but the basic activity was not modified. The stop-reaction was present, but less evident on the left hemisphere. In general there was a marked improvement.

The patient left hospital on the 19th day, quite normal from the clinical point of view.

Pathological anatomy.

The pathological tissue removed, not considering the necrotic cerebral tissue and the blood, constituted 22 fragments in all, with sizes varying

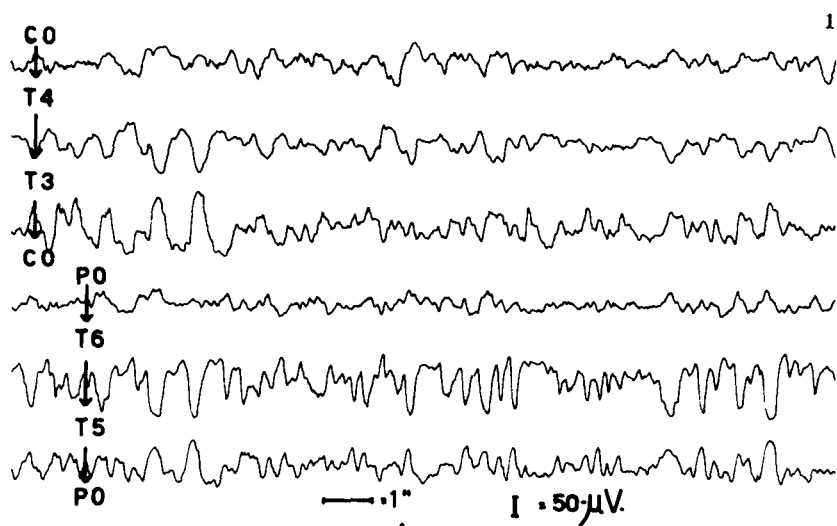


Fig. 6

EEG on the third day after operation,
three hours after epileptic state.

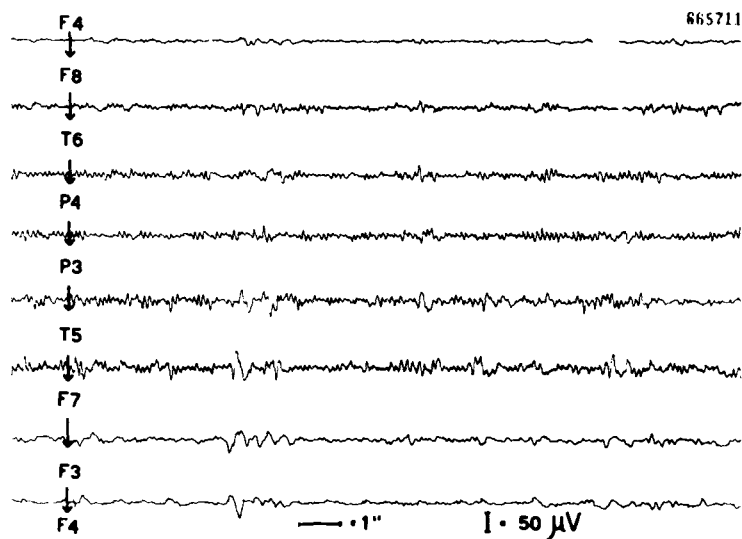


Fig. 7

EEG 18 days after operation

from a minimum of a lentil to a maximum of a hazel-nut. These fragments had the aspect of ectasic veins (Fig. 8). In sectioning them this was confirmed, because it was possible to distinguish the thin vasal wall and the lumen, which generally appeared to be obstructed by clots.

The histological examination (Fig. 9) showed the material to be veins, largely with thrombosis. The thrombi were relatively recent; in fact they presented initial connective proliferation (Fig. 10).

The patient has been examined periodically. The last control was made 13 months after the operation and he was found normal. The parents stated that the boy had had no more epileptic seizures and that, notwithstanding the operation, he had regularly continued with his studies.

The intelligence evaluation with the Wechsler-Bellevue method (V.I. = 2, 3) showed no signs of mental deterioration and gave a global intelligence quotient of 83% (V.Sc. = 75%, M.Sc. 91%). The EEG (Fig. 11) showed an abundant alpha rhythm, on the parietal and occipital regions, reacting well, wider to the left. On the whole tracings were recorded,

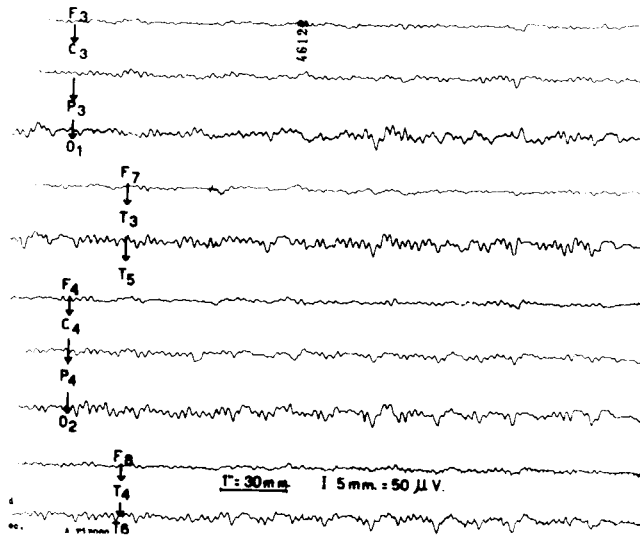


Fig. 11

EEG 13 months after operation.

mixed with alpha, 2-3 c/s oscillations, isolated or in short groups, of a maximum amplitude of 100 μ V, diffused and synchronous on the occipital and posterior temporal regions, prevalently on the left hemisphere. Hyperventilation did not substantially modify the tracing.

Insofar as the pathogenesis of the case is concerned, we present the hypothesis that the haemorrhage was precipitated by the vaso-dilatation due to the hormone treatment, and that the thrombosis was a consequence of circulation disturbances, caused by the haematoma. In fact, the ictus manifested itself during the hormone treatment, after which the symptoms became those of acute intracranial hypertension. While in the second evolution phase, a year and a half after the intracranial hypertension

crisis, the symptoms were exclusively of epileptic nature, which agrees with the clinical patterns of cerebral thrombosis. These assumptions are also based on the following facts ascertained during treatment:

- (a) the histological examination showed the thrombosis to be of recent date,
- (b) during the operation a haemorrhage focus was found in a rather advanced stage of evolution.

SUMMARY

A year and a half before operation a 13-year-old boy showed transient signs of intracranial hypertension, and 14 months later a right epileptic syndrome. Carotid angiography and encephalography showed signs of an expanding process deeply situated in the anterior half of the left cerebral hemisphere. At the operation the arachnoid of the Sylvian scissure was shown to be pigmented with hemosiderine. In an exploratory puncture of the posterior lateral part of the frontal lobe, (the needle being pointed at the head of the caudate nucleus) some cc of dark blood containing hemosiderine and colligated cerebral substance, were drained off whereas the incision of the cerebral cortex revealed a pathological mass, composed of venous ectasic vessels, mixed with extravasated blood and necrotic cerebral tissue. This pathologic mass was entirely removed. The resulting cavity, as big as an egg, was in the white substance of the frontal lobe, and in the region of the caudate nucleus. The histological examination showed a venous cavernous angioma with disseminated thrombosis. After the operation all symptoms and signs disappeared. Three similar cases are quoted in the literature.

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