THE 1982 PENFIELD LECTURE

Hemispherectomy for Seizures Revisited

Theodore Rasmussen

SUMMARY: The serious, late complication of superficial cerebral hemosiderosis, which appears after several years in 1/4-1/3 of patients who have undergone hemispherectomy, has resulted in recent years in a considerable reluctance to carry out this operation despite the fact it has proved to be highly effective in patients with medically refractory seizures associated with hemiplegia. Preservation of a small portion of the hemisphere, usually the frontal or occipital pole, has proved to be effective in preventing this late complication, but at the cost of a significant reduction in the effectiveness of the operation in reducing the patients' seizure tendency. Preserving the frontal and occipital poles but disconnecting them from the rest of the brain, resulting in a functional complete but anatomical subtotal hemispherectomy, retains the therapeutic effectiveness of a complete hemispherectomy while still protecting adequately against the serious late postoperative complication of superficial cerebral hemosiderosis and its associated neurologic deterioration, hydrocephalus and sometimes death.

RÉSUMÉ: L'hémosidérose cérébrale superficielle est une complication sérieuse qui se manifeste plusieurs années après l'hémisphérectomie et afflige entre 1/4 et 1/3 des patients qui ont subi cette opération. Il s'en est suivi une extrême réticence à y recourir, étant donné la gravité de cette complication tardive, en dépit de l'efficacité éprouvée de l'intervention à enrayer les crises d'épilepsie chez les patients affligés d'attaques réfractaires aux médicaments et associées d'hémiplégie. Une méthode a été instaurée pour parer à ces méfaits. Elle consiste à laisser en place une portion de l'hémisphère: le pôle frontal ou occipital le plus souvent. Le prix à payer est une diminution marquée du succès dans la réduction de la tendance aux crises d'épilepsie. Les pôles préservés sont coupés du reste du cerveau, produisant une hémisphérectomie qui est fonctionnellement complète, mais demeure anatomiquement quasi-complète. De cette façon, le patient bénéficie des avantages thérapeutiques de l'hémisphérectomie totale sans risquer la complication sérieuse et tardive que constituent l'hémosidérose cérébrale superficielle et ses séquelles que sont la détérioration neurologique, l'hydrocéphalie, et même la mort.

Can. J. Neurol. Sci. 1983; 10:71-78

The operation hemispherectomy, or hemicorticectomy as linguistic purists prefer to describe it, has waxed and waned in usage periodically since it was reported independently by Walter Dandy (1928) and by L'Hermitte (1928) as a dramatic effort to cure patients with malignant gliomas of the cerebral hemisphere. It soon became evident, however, that cures were not achieved, and that palliation and prolongation of life in these patients were no better than was achieved with more conservative treatment regimes. The operation, therefore, soon fell into disfavor and was rarely done during the late 1930's and the 1940's.

The pioneer Canadian neurosurgeon, Dr. K.G. McKenzie, in June 1938, carried out at the Toronto General Hospital what was probably the first hemispherectomy for infantile type hemiplegia and seizures, and reported the case in the Proceedings of the AMA the same year. However, it was Krynauw's 1950 report of hemispherectomy in 12 patients with infantile type hemiplegia and seizures that rejuvenated the procedure. His impressive results in reducing severe, medically refractory seizure tendencies in these severely handicapped individuals were soon verified in other neurosurgical centers in Britain, Europe, the United States and South America.

By 1953, Wylie McKissock in London had carried out hemispherectomies in a series of 18 patients with infantile type hemiplegia and seizures, with 12 patients rendered seizure free, 4 having only occasional seizures and only 1 patient not significantly benefitted. In Ransohoff's 1954 report to the A.R.N.M.D. on his first 3 hemispherectomy cases, he estimated that at least 100 such operations had been carried out by that time in neurosurgical centers around the world. The early MNI experience consisted of 15 hemispherectomies carried out by Dr. Penfield and myself between 1952 and 1958. Two monographs on hemispherectomy were published in the French literature by Gros and Vlahovitch (1955) and by Laine and Gros (1956). In 1961, H.H. White reviewed the literature and found 269 published cases of hemispherectomy in the treatment of infantile type hemiplegia and seizures. The average follow-up duration reported was 16 months, with only a few patients followed for up to 9 years.

The first hint of late trouble in hemispherectomized patients appeared in 1964 with a report of Laine, Pruvet and Ossen of 3 serious late complications in a series of 20 hemispherectomized patients with follow-up for periods ranging from 1 to 13 years. As a result of these 3 catastrophies in patients who had been doing well, the authors were concerned about the vulnerability of the remainder of the brain after removal of one hemisphere and speculated on the advisability of devising a stereotactic method of disconnecting the cortex of the bad hemisphere from the rest of the brain as an alternative to hemispherec-

From the Montreal Neurological Institute and Hospital.

Presented at the XVII Canadian Congress of Neurological Sciences, Toronto, June 1982 as the Penfield Award Lecture of the Canadian League Against Epilepsy.

Reprint requests to: Dr. Theodore Rasmussen, Montreal Neurological Institute and Hospital, 3801 University St., Montreal, Quebec H3A 2B4.

tomy. So far as I know, however, the matter was not pursued further.

The role of persistent intracranial bleeding in causing these late complications of hemispherectomy was described by Oppenheimer and Griffiths in their 1966 report of the Oxford hemispherectomy series. The importance of this late complication soon became apparent as reports from other centres appeared (Till, 1967; Brett, 1969; Falconer & Wilson, 1969; Wilson, 1970).

By 1969, 7 patients in the MNI hemispherectomy series had developed late pressure complications, appearing $4^1/_2$ to 10 years postoperative (Table 1). It should be noted and emphasized that this complication has rarely been reported earlier than $3^1/_2$ to 4 years after hemispherectomy. Three of these 7 patients (#3, 5 and 7) ultimately died as a result of this complication. Patient #1 made a partial, not very satisfactory recovery. The remaining 3 (patients #2, 4 and 6) made good recoveries after early and effective shunting procedures were carried out. Two more patients developed this complication in 1972 and 1973, one 20 years (#8) and the other 5 years postoperative (#9) (Table 1). Since 1973, 2 more of our hemispherectomy patients have developed this complication, 7 years and 24 years postoperative. This brings to 11 the number of patients in our hemispherectomy series who have developed this late complication to date.

These patients of our hemispherectomy series and the similar patients reported from other centers seem clearly to be examples of the syndrome of superficial cerebral hemosiderosis first reported by Noetzel in the German literature in 1940. Noetzel described the autopsy findings in 2 patients who had sustained repeated episodes of subarachnoid bleeding. The microscopic findings consisted of inflammatory reaction in the meninges with hemosiderin particles in macrophages, granular ependymitis, hydrocephalus and microscopic subpial necrotic lesions. This picture was reproduced in dogs by Iwanowski and Olszewski in Saskatoon in 1960 by the repeated subarachnoid injection of the dogs own hemolyzed blood or, more effectively, by the repeated subarachnoid injection of the dextran iron complex Imferon.

It seemed likely, therefore, that these late post-hemispherectomy complications were the result of gradual seepage of red cells into the hemispherectomy cavity over a period of several years, perhaps as a result of trivial jolts to the head or of abrupt normal changes of intracranial pressure due to coughing, sneezing, etc. In any event, there was a marked reduction of enthusiasm for hemispherectomy in many pediatric neurologic centers and also among many neurosurgeons, as reports of serious late complications of hemispherectomy continued to appear.

We had not seen this complication, however, in our series of 40 patients in whom, over the years, 2/3 or 3/4 of a cerebral hemisphere had been removed, and who had been followed for a minimum of 4 years after operation, comparable to the minimum time in which this late complication had developed in our 27 hemispherectomized patients (Table 2). The *median* follow-up period was also similar in the 2 groups. The 2 late complications that are listed in the

Table 1: Late Complications of Hemispherectomy

Pt. No.	Age yrs.	Operation & Complication Dates	Location of CSF Block	Shunt Procedure	Follow-up Data
1	$5^1/_2$	1953 - 1958	acqueduct and foramen of Munro	ventriculo-cisternal	partial recovery
2	7	1955 - 1962	acqueduct	ventriculo-atrial revised 1971 & 1972	good recovery neurologically stable
3	11	1955 - 1965	acqueduct and trigone of ventricle	opening of cisterns cavity-cisternal	partial recovery then death $1^1/_3$ yrs. later
4	16	1956 - 1966	acqueduct and foramen of Munro	ventriculo-atrial	good recovery neurologically stable
5	3	1956 - 1966	acqueduct	ventriculo-cisternal	partial recovery died in seizure 1967
6	7	1961 - 1969	acqueduct	cavity-atrial revised 1971	good recovery neurologically stable
7	7	1964 - 1969	acqueduct	cavity-atrial revised twice 1972	good recovery died of pneumonitis after last shunt revision
8	8	1952 - 1972	acqueduct and foramen of Munro	ventriculo-atrial	died of hemorrlogic complications of shunt
9	17	1968 - 1973	acqueduct and foramen of Munroe	ventriculo-peritoneal	good recovery

https://doi.org/10.1017/S0317167100044668 Published online by Cambridge University Press

subtotal hemispherectomy series were of a different order. One was really an early postoperative complication. In this patient a slight increase in intracranial pressure persisted beyond the early postoperative period and was never completely controlled despite periodic lumbar punctures and a shunt and several shunt revisions. CSF rhinorrhea finally developed leading to ventriculitis and death 7 years postoperative. The second patient developed a closed cyst in the removal cavity 9 years postoperative, which caused headache without neurologic deterioration. She died suddenly of brain stem herniation, verified by autopsy. Thus the syndrome of superficial cerebral hemosiderosis, which developed in 9 of our first 27 hemispherectomized patients, was not encountered in the series of 40 patients in whom a

Table 2: Late Pressure Complications of Large Brain Removals

Operation	No. of pts.	Late Increased Pressure Complication	Interval Between Operation & Complication	Duration of Follow-up
Hemispherectomy	27	9 pts. (33%)	4½-20 yrs. (median 8 yrs.)	5-20 yrs. (median 11 yrs.)
Subtotal hemispherectomy (removal of at least 3 lobes of the hemisphere)	40	2 pts. (5%)	7 yrs. 9 yrs.	4-26 yrs. (median 10 yrs.)

Table 3: Hemispherectomy for Seizures

1952 through 1972

Seizure free since discharge	13 pts. (48%)	16 pts. (59%)	23 pts. (85%)	27 pts. with follow-up data of 5-20 yrs. median 11 yrs.
Became seizure free after some early attacks	3 pts. (11%)			
Free 3 or more years then rare or occasional attacks	6 pts. (22%)	7 pts. (26%)		
Marked reduction of seizure tendency	1 pt. (4%)			
Moderate or less reduction of seizure tendency	4 pts. (15%)			
Died in 1st year of progressive encephalopathy	2 pts.			
Total	**29 pts.**			

https://doi.org/10.1017/S0317167100044668 Published online by Cambridge University Press

subtotal hemispherectomy had been carried out and the patients then followed for a minimum period of 4 years.

Apparently the preservation of a small portion of the bad hemisphere provided enough support to the rest of the brain to reduce its vulnerability to whatever mechanism was responsible for the gradual seepage of red cells into the large removal cavity and the resulting late development of the syndrome of superficial cerebral hemosiderosis, hydrocephalus and neurologic deterioration.

In 1968, therefore, we abandoned the technically gratifying and therapeutically effective operation of complete one stage hemispherectomy and routinely preserved the least epileptogenic 1/4 or 1/3 of the hemisphere in those patients who previously would have been considered candidates for complete hemispherectomy. This surgical tactic has effectively eliminated the late complication of superficial cerebral hemosiderosis with its symptoms of hydrocephalus and gradual neurologic deterioration (Rasmussen, 1973), but at the cost of a significant lessening of the effectiveness of the cortical excision in reducing the seizure tendency.

For example, as of 1972 (Table 3), 59% of our hemispherectomy patients had become and remained seizure free and 26% averaged no more than 1-2 attacks per year. Thus 85% had experienced a complete or nearly complete reduction in seizure tendency.

In patients who had 2/3 or 3/4 of the hemisphere removed over the years, the reduction in seizure tendency was not quite as satisfactory (Table 4), 45% seizure free compared to 59% in the hemispherectomy series, and 68% with a complete or nearly complete reduction of seizure tendency compared to 85% in the hemispherectomy series.

In recent years, therefore, we have once again altered our surgical tactics in dealing with patients with maximal or near maximal hemiplegia and severe medically refractory seizures. The change consists in disconnecting the remaining segment of the bad hemisphere from the rest of the brain by sectioning the white matter down to the medial pial surface. The remaining frontal or occipital pole is therefore separated from the corpus callosum and the upper brain stem, resulting in a functional complete but anatomical subtotal hemispherectomy. As of the end of 1980, this revised procedure has been carried out in 8 patients (Table 5). Six have had no seizures since discharge from hospital, and 1 has been seizure free for 5 years after having several brief minor absence attacks in his first postoperative year. The eighth patient, operated upon at 4 years of age, had

Table 4: Results of Subtotal Hemispherectomy for Seizures (1/5 to 1/3 of hemisphere preserved - operated 1936 through 1978)

Seizure free since discharge	15 pts. (26%)	26 pts. (45%)	39 pts. (68%)	57 pts. with follow-up data of 2-31 yrs. (median 13 yrs.)
Became seizure free after some early attacks	11 pts. (19%)			
Free 3 or more years, then rare or occasional attacks	11 pts. (19%)	13 pts. (23%)		
Marked reduction of seizure tendency	2 pts. (4%)			
Moderate or less reduction of seizure tendency	18 pts. (32%)			
Postoperative deaths	2 pts.			
No follow-up data	2 pts.			
Total	**61 pts.**			

https://doi.org/10.1017/S0317167100044668 Published online by Cambridge University Press

"chronic encephalitis" and preoperatively was unable to walk or stand alone due to constant epilepsia partialis continua involving the left face, arm and leg. At her last report, 6 years postoperative, she is attending regular school with the help of a tutor and her seizure tendency has been reduced to one or two minor attacks per day lasting 10-15 seconds and consisting of twisting of the mouth to the left, staring and arrest of activity.

The follow-up in these 8 patients is still relatively short, 2 to 6 years, but the incidence of late pressure complications should not differ from those in our larger series of patients, now 56 in number, who have had an anatomical subtotal hemispherectomy carried out, and have been followed for a minimum period of 4 years (Table 5). The 1 late complication listed in this series is the patient mentioned above who developed a closed cyst in the removal cavity and died as a result of brain stem herniation 10 years after operation.

We have another group of 72 patients who have had a hemi-hemispherectomy carried out, with about 1/2 of the bad cemisphere preserved and who have also been followed for a minimum period of 4 years (Table 5). One patient operated upon at age 12 years developed headaches, dizziness, poor memory and confusion 13 years postoperative. A diagnosis of low pressure hydrocephalus was made at her local hospital and a ventriculo-atrial shunt carried out with prompt relief of the symptoms and a stable neurological status for the subsequent 9 years. If her case represents a minimal degree of superficial cerebral hemosiderosis, it is the only instance of this complication in the 136 patients of these last 3 groups. This contrasts with a 35% incidence in our 31 anatomically complete hemispherectomy cases. (Table 5).

There is another intracranial pressure complication of large brain removals that should be mentioned. Although a low grade increase in intracranial pressure is commonly present for a week or 10 days in nearly all patients who have had large brain removals carried out, this usually resolves spontaneously, and is part of the normal postoperative course and is not a significant complication of the operation. In a few patients, however, the increase in pressure persists beyond the postoperative period as a result of inadequate absorption of CSF, secondary to the extensiveness of the removal of pia-arachnoidal absorbing surface (Table 6). This has occurred in 3 (9.7%) of our 31 patients with an anatomical complete hemispherectomy, in 5 (7.7%) of 65 patients with anatomical subtotal hemispherectomy and in 2 (2.2%) of 90 patients in whom about 1/2 the hemisphere has been removed. If this early postoperative increased pressure persists beyond the 3rd or 4th postoperative week, we have learned that a shunt should not be unduly delayed, even though the increased intracranial pressure is minimal.

Table 5: Late Intracranial Pressure Complications of Large Brain Excisions

Operations (1930 through 1980)	No. of pts.	Late pressure complication	Interval between operation and complication	Duration of follow-up
Anatomical complete hemispherectomy (1 stage.....24 pts.) (2,3 or 4 stages.....7 pts.)	31	11 pts. (35%)	4 1/2 - 24 yrs. (median 9 yrs.)	6 to 28 yrs. (median 17 yrs.)
Functional complete but anatomical subtotal hemispherectomy	8	0 1.6%	—	2 to 6 yrs. (median 3 1/2 yrs.)
Subtotal hemispherectomy 1/5 to 1/3 of hemisphere preserved	56	1 pt.	10 yrs.	4 to 31 yrs. (median 15 yrs.)
Hemi-hemispherectomy about 1/2 of hemisphere preserved	72	1 pt. (1.4%)	13 yrs.	4 to 36 yrs. (median 11 yrs.)

https://doi.org/10.1017/S0317167100044668 Published online by Cambridge University Press

Table 6: Early Intracranial Pressure Complications of Large Brain Excisions

Operations (1930 through 1980)	No. of pts.	Early pressure complication
Anatomical complete hemispherectomy (1 stage.....24 pts.) (2,3 or 4 stages.....7 pts.)	31	3 pts. (9%)
Functional complete but anatomical subtotal hemispherectomy	8	0 } 7.7%
Subtotal hemispherectomy 1/5 to 1/3 of hemisphere preserved	57	5 pts. } 7.7%
Hemi-hemispherectomy about 1/2 of hemisphere preserved	90	2 pts. (2.2%)

The effectiveness of hemispherectomy in reducing medically refractory seizure tendencies in patients with hemiplegia has held up well through the years. Our current statistics (Table 7), with the functional complete but anatomical subtotal hemispherectomy cases included, are essentially the same as those of the 1972 analysis (Table 3). Fifty-four per cent have become and remained seizure free, and 84% have had a complete or nearly complete reduction of seizure tendency. The median follow-up period of these 37 patients who survived the first postoperative year, is now 15 years.

Although the 6 patients, classified as having a moderate or less reduction of seizure tendency, have had too many seizures to qualify for either of the two categories just above, as we have defined them, all have derived significant benefit. Two of these 6 have had only about 1-2% as many attacks compared to the preoperative rate, 2 have had about 5-10% and the remaining 2 about 50% of the preoperative rate. It is of interest to note in passing that in 3 of these 6 patients, there is good clinical evidence the residual seizures are still arising from the side of the operated hemisphere. In 2 of these 6 patients there is clinical or EEG evidence the residual seizures are arising in the remaining, good, hemisphere. There is no clinical or EEG information available as to the localization of this residual seizure tendency in the remaining 6th patient.

Table 7: Results of Hemispherectomy for Seizures operated 1952 through 1979

Seizure free since discharge	17 pts. (46%)	20 pts. (54%)	31 pts. (84%)	37 pts. with follow-up data of 2-28 yrs.
Became seizure free after some early attacks	3 pts. (8%)			
Free 3 or more years, then rare or occasional attacks	10 pts. (27%)	11 pts. (30%)		
Marked reduction of seizure tendency	1 pt. (3%)			
Moderate or less reduction of seizure tendency	6 pts. (16%)			
Died in 1st postoperative year	2 pts.			
Total	**39 pts.**			

https://doi.org/10.1017/S0317167100044668 Published online by Cambridge University Press

This latest revision of the 55 year old operation, hemispherectomy, enables the patient with a maximal or near maximal infantile type hemiplegia and a severe medically refractory seizure tendency to have the full benefits of a functionally complete hemispherectomy, so extensively documented over the past 30 years, most recently by Verity et al. (1982), along with the safeguards against both early and late pressure complications that are provided by a subtotal or hemi-hemispherectomy. The technical aspects of this revised procedure consist first in the removal of the central portion of the hemisphere above the fissure of Sylvius, carrying the excision anteriorly to a level just in front of the rostrom of the corpus callosum and posteriorly to a level just behind the splenium of the corpus callosum. On the medial surface the excision is carried down to the cingulate gyrus, thus protecting the two anterior cerebral arteries during this phase of the removal. After removal of this central segment of the brain, the white matter of the remaining portion of the frontal lobe is sectioned just in front of the rostrom of the corpus callosum down to the medial pial surface. Similarly the white matter of the remaining portion of the posterior parieto-occipital region is sectioned just behind the splenium of the corpus callosum, also down to the medial pial surface. Thus the remaining frontal and posterior parieto-occipital brain regions are disconnected from the unoperated hemisphere and from the brain stem. The cortex of the cingulate and subcallosal gyri can then easily and safely be removed completely by suction. Next the temporal lobe is removed back to the level of the posterior line of excision. Finally the hippocampus is removed from its pial bed and the choroid plexus removed from the temporal horn and trigone of the ventricle as close to the foramen of Munro as possible.

The risk-benefit ratio of the use of this revised, functional complete but anatomical subtotal hemispherectomy, in my opinion, is strongly in favor of its more wide spread use in those unfortunate seizure patients with a maximal or near maximal hemiplegia and a complete or high grade homonymous hemianopsia and whose seizures constitute a significant handicap in regard to schooling and psychosocial development, despite an adequate trial of appropriate antiepileptic medication. The earlier the good hemisphere is spared the nociferous effect of continual bombardment of high amplitude epileptiform discharges, the more effectively development can take place in all areas, motor, sensory, intellectual and psychosocial (Carlson, et al. 1968).

When the patient still has a useful visual field, it is usually wise to preserve the function of the posterior third of the hemisphere, including the posterior half of the temporal lobe to avoid adding an homonymous hemianopsia to the patient's neurologic deficits. If a significant seizure tendency persists, however, and does not soon show evidence of decreasing, reoperation and disconnection of the remaining portion of the hemisphere should not be unduly delayed. The resulting hemianopsia is nearly always much less of a handicap, particularly in young children, than continuing seizures and bombardment of the rest of the brain by the epileptiform discharges.

The patient who has only a mild or moderate hemiparesis despite having an extensively damaged and epileptogenic hemisphere poses a much more difficult therapeutic problem. In this patient population, neither the clinical attack pattern nor the epileptiform EEG discharges give as accurate guidance to the neurosurgeon as is the case in more restricted types of focal epilepsy. More quantitative methods are particularly needed in these patients to assess what might be called the second and third order of localizational aspects of epileptogenesis. After the primary localization has been determined, where the attacks begin, the second order localization consists of determining how much of the cortex contiguous to the site of origin of the seizure discharge must be recruited into action to produce a clinical seizure, and then, thirdly how much of this potentially epileptogenic cortex must be removed to satisfactorily reduce the seizure tendency. However, until new techniques improve the accuracy of these second and third order localizational aspects of epileptogenesis, in this patient population it seems wise to remove as much of the obviously damaged cortex as can be done without undue risk of increasing the patient's existing neurologic deficit, even though the attack pattern and the cortical EEG may suggest that a more restricted cortical resection might be adequate.

But that is a story for another day, as are the many interesting neurophysiological, neuroanatomical, neuropathological, electroencephalographic, neuropsychological and socio-economic aspects of hemispherectomy that lie outside the narrow focus of this report of the development of our surgical tactics to retain the benefits of hemispherectomy for patients with hemiplegia and medically refractory seizures and still protect them from the late development of superficial cerebral hemosiderosis and its associated neurologic deterioration, hydrocephalus and sometimes death.

References

Brett, E. (1969). Second thoughts on hemispherectomy in infantile hemiplegia. Develop. Med. Child Neurol. 11:374-376.

Carlson, J., Netley, C., Hendrick, E.B. and Pritchard, J.S. (1968). A re-examination of intellectual disabilities in hemispherectomized patients. Tr. Amer. Neurol. Assn. 93:198-201.

Dandy, W. (1928). Removal of right cerebral hemisphere for certain tumors with hemiplegia: preliminary report. J.A.M.A., 90:823-825.

Falconer, M.A. and Wilson, P.J.E. (1969). Complications related to delayed hemorrhage after hemispherectomy. J. Neurosurg. 30:413-426.

Gros, C. and Vlahovitch, B. (1955). L'hémisphérectomie cérébrale. Imprimerie Causse, Graille et Castelnau. Montpellier, 113 pp.

Iwanowski, I. and Olszewski, J. (1960). The effects of subarachnoid injections of iron containing substances on the central nervous system. J. Neuropath. and Exper. Neurol. 19:433-448.

Krynauw, R.A. (1950). Infantile hemiplegia treated by removing one cerebral hemisphere. J. Neurol. Neurosurg. Psychiat. 13:243-267.

Laine, E. and Gros, C. (1956). L'hémisphérectomie. Masson et Cie, Editeurs, Paris. 134 p.

Laine, E., Pruvet, P. et Osson, D. (1964). Résultats éloignés de l'hémisphérectomie dans les cas d'hémiatrophie cérébrale infantile génératrice d'épilepsie. Neuro-Chirug. 10:507-522.

Lhermitte, J. (1928). L'ablation complète de l'hémisphère droit dans les cas de tumeur cérébrale localisée compliquée d'hémiplégie: la décérébration supra-thalamique unilatérale chez l'homme. Encéphal. 23:314-323.

McKenzie, K.G. (1938). The present status of a patient who had the right cerebral hemisphere removed. Proc. Amer. Med. Assn, Chicago 111:168.

McKissock, W. (1953). Infantile hemiplegia. Proc. Royal Soc. Med. 46:431-434.

Noetzel, H. (1940). Diffusion von Blutfarbstoff in der innerne Randzone und äusseren Oberfläche des Zentralnervensystems bei subarachnoidaler Bluting. Arch. f. Psychiat. 111:129-138.

Oppenheimer, D.R. and Griffith, H.B. (1966). Persistant intracranial bleeding as a complication of hemispherectomy. J. Neurol. Neurosurg. Psychiat. 29:229-240.

Ransohoff, J.C. (1954). Hemispherectomy in the treatment of convulsive seizures associated with infantile hemiplegia. Res. Publ. Ass. Nerv. Ment. Dis. 34:176-195.

Rasmussen, T. (1973). Postoperative superficial hemosiderosis of the brain, its diagnosis, treatment and prevention. Trans. Am. Neur. Assn. 98:133-137.

Till, K. (1967). Hemispherectomy for infantile hemiplegia. Dev. Med. Child. Neurol. 9:773-774.

Verity, C.M., Strauss, E.H., Moyes, P.O., Wada, J.A., Dunn, H.G., and Lapointe, J.S. (1982). Long term follow-up after cerebral hemispherectomy: neurophysiologic, radiologic and psychological findings. Neurology 32:629-639.

White, H.H. (1961). Cerebral hemispherectomy in the treatment of infantile hemiplegia: review of the literature and report of 2 cases. Confinia neurol. 21:1-50.

Wilson, P.J.E. (1970). Cerebral hemispherectomy for infantile hemiplegia. A report of 50 cases. Brain 147-180.

https://doi.org/10.1017/S0317167100044668 Published online by Cambridge University Press