

EEG findings in shunted hydrocephalic patients with epileptic seizures

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The EEG findings in 40 shunted hydrocephalic patients tested after the onset of epileptic seizures are assessed. The salient point to emerge from the study is the higher frequency of anomalies, both specific (spikes, slow spikes, spike and wave complexes) and nonspecific (monomorphic and polymorphic slow waves) on the shunted hemisphere (19 cases) than on the unshunted side (8 cases). The lateralization of EEG anomalies on the shunted side in patients with no neurological deficits and no focal damage to the substance of the brain suggests that the EEG focus is in some way related to the presence of the shunt. Without denying the importance of the hydrocephalus etiology as a cause of epilepsy in shunted patients, we do think that, in some cases at least, epilepsy is to be regarded as a surgical complication.

Key-Words: Epilepsy – hydrocephalus – CSF shunts

Introduction

Epilepsy is a not uncommon finding in hydrocephalic patients who have undergone shunting procedures (in the main with ventriculo-atrial or ventriculoperitoneal shunts). Although the exact frequency is hard to assess, we shall not be far from the truth if we say that epilepsy occurs in about 30% of patients with hydrocephalus [2,5]. In a comprehensive follow-up at nine Italian neurosurgical centers epilepsy was found in 172 out of 910 cases [1].

Apart from the occasional case report [3], the neurosurgical literature tends to regard the epilepsy that accompanies hydrocephalus as disease-related or anyway connected with the etiology, ignoring the possibility that it may be considered, at least in part, as a complication of surgery.

The shunting procedure used for treating hydrocephalus involves the insertion of a catheter into the lateral ventricle passing through the substance of the brain and the rare reference in the neurophysiological literature to the subject does

not fail to point out that: "the trauma of catheter placement and the subsequent presence of a foreign body within the substance of the brain are potential causes of focal cerebral dysfunction" [4]. In view of these conflicting assumptions and to find out the relevance, if any, of this "potential cause of focal cerebral dysfunction" to the genesis of epilepsy, we decided to evaluate the EEG findings in a consecutive series of shunted hydrocephalic patients seen by us after the onset of convulsive manifestations.

Patients

The series comprises 40 shunted hydrocephalic patients, 23 males and 17 females, admitted to or attending the outpatient clinic of the Neurosurgery Division of the Santobono Children's Hospital, Naples, following an epileptic seizure. The etiology of the primary hydrocephalic condition that prompted the shunting procedure is stated in Table I. In 17 cases the shunt was peritoneal and

TABLE I. Etiology in 40 shunted cases of hydrocephalus with seizures

	No.	%
DEVELOPMENTAL	23	57.5
Encephalocele	2	
Spina bifida	7	
Aqueductal stenosis	11	
Dandy-Walker malformation	2	
Complex ventricular malformation	1	
INFLAMMATORY	7	17.5
HEMORRHAGIC	6	15
UNKNOWN	4	10
	40	100

in 23 atrial; in all cases the ventricular catheter was inserted into the right lateral ventricle through a burrhole in the occipitoparietal region. The mean age at the time of first shunting was 9.8 months with a range of 7 days to 9 years, 26 patients underwent reoperation one or more times with a mean of 2.1 reoperations per patient.

At the time of the convulsive episode with which this study is concerned the mean age of the patients was 4.7 years (range: 2 months to 20 years). 22 of them were at their first seizure and 18 had had one or more and were on anticonvulsants. The seizures started after the shunting procedure in all of the latter patients barring two, a girl of 4 1/2 years and another of 4 months, the former with postmeningitic hydrocephalus and the latter with triventricular hydrocephalus, both of whom had seizures before the operation. In the 38 patients whose seizures started after surgery the mean interval between the shunting procedure and the onset of seizure was 41.9 months with a range of 10 days to 11 years. The longest interval was in a 20 year old man operated on at the age of 9 years for triventricular hydrocephalus and a further 3 times for revision.

Table II lists the types of convulsions occurring in the 40 cases of the series. We have some reservations about the data, most of which are based on descriptions supplied by relatives, who may have missed earlier localized seizure. In 4 cases it was impossible to reconstruct the history and so no reliable classification could be made for them.

Table III gives the neurological and psychomotor status of our patients. CT scanning was done in 37 patients and the relevant conditions are listed in Table IV.

Electroencephalographic study

The EEGs were altered in all our patients. The ab-

TABLE II. Types of seizures

	No.	%
LOCALIZED:		
hemiclonic	7	
adversive	3	
complex	2	
generalized with localized onset	1	
Total	13	32.5
GENERALIZED:		
clonic	15	
tonic	3	
tonic-clonic	5	
Total	23	57.5
UNCLASSIFIED	4	10

TABLE III. Neurological and mental status of the patients

Normal*	14	35
Mild deficit	12	30
Medium grade	6	15
Severe		

* Patients with a neurological deficit secondary to spina bifida and with normal mental status have been included in this group.

TABLE IV. CT findings in 37 cases*

	No.	%
mild	8	21.6
Ventricular dilatation: medium grade	17	45.9
severe	1	2.7
Ventricular asymmetries	2	5.4
Dilatation subarachnoid spaces	4	10.8
Hypodense areas of parenchyma	2	5.4
Pericerebral CSF layers	4	10.8
Hyperdense pericerebral layers	1	2.7
Poroencephalia	1	2.7

* More than one abnormality present in some cases

normalities found were both nonspecific and specific. Both were found in some patients.

Nonspecific abnormalities

25 patients presented diffuse alteration of background activity expressed by a prevalence of delta and/or theta waves. Diffuse slowing was the only abnormality in 3 of them while this was accom-

TABLE V. EEG abnormalities in 40 cases*

	No.	%
Diffuse slow waves	25	62.5
Focal slow waves	13	32.5
Specific paroxysms:		
focal	17	42.5
multifocal	2	5
generalized	9	22.5

* More than one type found in some cases

panied by focal slowing in 7, by specific focal bursts in 11 and by generalized bursts in 4 patients.

In 8 patients the EEG was characterized by a focus of polymorphic slow waves, located on the right hemisphere in 5 and on the left in 3 patients. 5 other patients presented focal slow waves, rhythmic, monomorphic and on the right hemisphere in every case.

The site of the slow wave foci on the right hemisphere, that is, on the shunted side, was occipital in 3 cases, parieto-occipital in 3, temporo-occipital in one, parietal in one and temporal in one.

The occipital lobe was affected in only one of the cases with focal slow waves on the left hemisphere.

Specific abnormalities

Specific irritative abnormalities were found in 28 patients, 4 of whom also presented foci of slow waves. In 20 patients the abnormalities presented as focal paroxysms and in 8 as generalized paroxysms. Focal paroxysms varying in morphology from case to case (spikes, spike and wave complexes, slow spikes, both isolated and in bursts) appeared on the right or shunted hemisphere in 13 cases, on the left, or unshunted, hemisphere in 5 cases and on both in 2 cases.

The site of the focal paroxysms on the right hemi-

sphere was occipital in 4 cases and mainly temporal and/or parietal and/or central in the others. On the left hemisphere the site was occipital in only one case and fronto-central in two.

9 patients presented generalized paroxysmal abnormalities with discharges of spikes in 3, spike and wave complexes in 5 and poly-spike and wave complexes in one. One of these patients also presented specific focal abnormalities on the right hemisphere.

Table V summarizes the EEG findings in the 40 patients. With regard to the side of the focal abnormalities, both specific and nonspecific, the right hemisphere or shunted side was affected in 19 cases and the left or unshunted side in 8 cases.

Table VI correlates the EEG abnormalities found with the patients' neurological and psychomotor status.

Discussion

The connexion between epilepsy and etiology of hydrocephalus is certainly true and well documented.

Blauuw [2] reported an epilepsy frequency of 34% in his 323 shunted hydrocephalic patients, a frequency that correlates closely with the etiology since it ranged from 24% in his spina bifida group to 66% in his postmeningitic hydrocephalus group. It is clear from this datum that a hydrocephalic patient's chances of becoming epileptic are greater in all those conditions in which the cause of hydrocephalus is also the cause of structural changes in the cerebral parenchyma, as is the case of postinflammatory hydrocephalus above all.

While there is no doubt about the connexion between hydrocephalus and epilepsy, it is not applicable to all cases, in view of the fact that epilepsy may arise in patients who have no neurological deficits and whose psychomotor development is normal [9].

TABLE VI. Correlation between EEG findings and neuropsychic status

	Neuropsychic status				Total
	Normal	Mild deficit	Medium grade deficit	Severe deficit	
Focal abnormalities:					
right hemisphere	8	6	1	4	19
left hemisphere	2	3	2	1	8
Multifocal abnormalities	2				2
Generalized paroxysms	2	2	3	1	8
Diffuse slow waves		1		2	3

Note: The EEG findings have been classified according to the principal abnormalities.

The salient datum to emerge from our EEG study is the lateralization of focal abnormalities, specific or otherwise, on the shunted side.

Pampiglione and Laurence [8] reported EEG anomalies on the shunted hemisphere in 3 out of 8 shunted patients. Laws and Niedermeyer [6] found focal EEG abnormalities on the shunted hemisphere in 12 out of 18 shunted patients and no EEG lateralization in any of their unshunted hydrocephalus patients. Marossero et al [7] reported paroxysmal anomalies in 14 out of 33 shunted patients with lateralization on the shunted hemisphere in 11 of them. Graebner and Celestia [4] compared 26 shunted hydrocephalics with 21 unshunted patients in respect of EEG data: in the first group 11 patients presented abnormalities on the shunted side, which in their cases too was the right side, whereas only 2 of the unshunted patients presented EEG lateralization, the difference being statistically significant ($p < 0.01$). Taken together, these data broadly confirm what we found, namely that there is some correlation between focal EEG abnormalities and the side of the shunt.

This correlation finds further support in a longitudinal study conducted by Varfis et al [9] on 29 shunted hydrocephalic children followed clinically and electroencephalographically for 4 years: 19 of them presented an epileptogenic focus on the EEG close to the site of insertion of the ventricular catheter. 17 of these patients presented seizures. Another point demonstrated by these workers is that in the majority of cases the epileptic focus develops after two years have elapsed since the shunting procedure, often preceded by focal slow waves at the same site. Like Blauuw [2], Varfis et al [9] point up the connexion between epilepsy and etiology of the hydrocephalus, especially when associated focal cerebral lesions are present, but they also emphasize that "... in several cases no such focal lesion is clinically apparent, or even may be in both hemispheres, whereas an irritative

focus is present in 14 of the 23 cases without hemiparesis... this irritative focus is always located where the catheter was first inserted".

This datum is confirmed in our series by the fact that it was the patients without neurological deficits – apart, obviously, from those secondary to spina bifida – who presented EEG lateralization mainly on the shunted side. Likewise in cases with mild mental retardation without focal neurological deficits EEG lateralization was mainly on the shunted side.

The presence of focal anomalies on the shunted hemisphere in patients with no neurological deficits traceable to focal damage to the parenchyma suggests that, at least in these cases, the epilepsy may somehow be related to the surgical procedure or rather to the presence in the substance of the brain of the shunt catheter, responsible in its turn for the epileptogenic focus.

The anatomical lesion responsible for this focus is, according to Laws and Niedermeyer [6], a low grade infection or small hemorrhages and, according to Varfis et al [9], adhesion of the cerebral cortex to the dura mater.

Conclusions

On the strength of data published by other workers and of our own we think it safe to say that, at least in some cases, the epilepsy that occurs in shunted hydrocephalic patients may be regarded as an actual surgical complication. Obviously this is in no sense to be regarded as a criticism of a procedure that is in many respects indispensable in the treatment of childhood hydrocephalus. We nonetheless consider it important to draw attention to this possibility in the hope of encouraging even closer clinical and EEG surveillance of a condition, like that of the shunted child, potentially an epileptic risk.

Sommario

Sono stati valutati i reperti EEG di 40 pazienti idrocefalici portatori di derivazione liquorale extratecale controllati in seguito all'insorgenza di manifestazioni convulsive.

Il dato principale emergente dallo studio è rappresentato da una prevalenza di anomalie sia specifiche (punte, punte lente, punte onda) sia non specifiche (onde lente mono e polimorfe) sull'emisfero derivato (19 volte) rispetto a quello non derivato (8 volte).

La lateralizzazione delle anomalie EEG sull'emisfero ove è presente il catetere di derivazione in casi nei quali l'esame neurologico è normale e non sono presenti danni parenchimali focali fa ritenere che il focolaio EEG sia in qualche modo correlabile con la presenza della derivazione.

Senza negare l'importanza dell'etiologia come causa di Epilessia nei pazienti idrocefalici gli AA. ritengono che, almeno in una parte dei casi, l'Epilessia debba essere considerata come una complicanza chirurgica.

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