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Corpus callosotomy in refractory idiopathic generalized epilepsy

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Summary

Rationale: A small percentage of patients with idiopathic generalized epilepsy (IGE) do not respond to medical therapy. Generalized tonic–clonic (GTC) seizures are especially debilitating and can be associated with severe injuries. The benefit, safety and effect of corpus callosotomy (CC) in patients with IGE have not been studied.
Methods: We reviewed patients with presumed IGE who underwent CC between 1991 and 2000. Criteria for selection included history, examination, brain imagining, interictal and ictal EEG. All patients had refractory and debilitating tonic–clonic seizures (GTCS) and had failed four or more antiepileptic drugs. Seizure frequency was calculated per month over the last year and pre-operative baseline was compared to last follow-up using paired *t*-tests. IQ, executive function, language and verbal, non-verbal memory and quality of life (QOL) was compared before and after surgery. Serial EEGs after surgery were reviewed.
Results: There were nine patients (seven men), mean age 37.9 (range: 22–49), mean IQ 87.3 (range: 75–107). All had anterior CC. Mean follow-up time was 5.4 years (range: 0.6–10.3 years). One patient died from sudden death in epilepsy 9 months after surgery. There was a significant reduction of GTC seizures from 6.3 to 1.1 ($p < 0.005$). Four patients had more than 80% and eight more than 50% reduction. Of five patients with absence seizures, two became seizure free and one had more than 80% reduction and two worsened slightly, and of three with myoclonic seizures one had more than 90% reduction. One patient had completion of the CC with improvement of myoclonus and absence seizures, but not of GTC seizures and suffered a disconnection syndrome. Another had right frontal focal resection without improvement after new seizures of focal onset. Cognitive testing showed a good outcome (improved or no change) in all cognitive domains. Post-surgical EEG showed new focal slowing and sharp waves. There was no change in QOL.

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Conclusion: CC can be effective in reducing GTC, absence and myoclonic seizures in patients with refractory IGE. These findings suggest that interhemispheric communication of the cerebral cortices plays an important role in the generation of seizures in IGE. Anterior CC appears safe while complete callosotomy has a risk of disconnection syndrome.

Introduction

Idiopathic generalized epilepsy (IGE) is typically characterized by either absence, myoclonic or generalized tonic–clonic (GTC) seizures.[1,2] These seizures occur in the absence of structural lesions or localized brain dysfunction and often are inherited in nature. Thus, normal intelligence is found, the neurological exam is normal, and brain imaging does not reveal any significant structural abnormalities. The EEG shows no focal abnormalities and is normal except for generalized 3 Hz or faster spike – wave and poly-spike – wave bursts. IGE is non-progressive in nature and begins in childhood or adolescence. While seizures are usually readily controlled by antiepileptic drugs (AED), some patients do not respond to therapy and continue to experience frequent seizures.[3] This is a serious problem, since GTC and myoclonic seizures may cause severe injuries.[4]

The relative contributions of the cerebral cortex and the thalamus in the physiopathology of generalized seizures have been hotly debated.[5–7] The cerebral cortex appears to be important in epileptogenesis and the corpus callosum has also been shown to play an important role in the synchronization and spread of seizures in the feline model of generalized seizures.[8] On the other hand, if seizures in IGE originate mainly from thalamus then interruption of the corpus callosum would not have major effect on seizures. In symptomatic generalized epilepsy (SGE), corpus callosotomy (CC) has been shown to be effective in decreasing the number of generalized seizures.[9] One report suggests that CC can ameliorate seizures also in IGE, but the diagnosis of IGE was not documented.[10] Search of Medline does not reveal any cases of refractory IGE were CC was attempted. With this case series we aim to report the effect of CC on seizure frequency, scalp EEG, neuropsychological integrity and quality of life.

Methods

Patients

Patients who were treated with anterior or complete CC for intractable epilepsy at Graduate Hospital and Thomas Jefferson University, Philadelphia, PA between 1991 and 2000 and who met the following criteria were included:

(1) GTC seizures (at a rate of one or greater per month) that were refractory to treatment with at least four drugs. Patients may have had absence or myoclonic seizures as well. Patients with atonic or tonic seizures were excluded.
(2) Interictal EEG had to show generalized spike-and-wave complexes (SWC) at a frequency of 3 Hz or greater, normal frequency of background (alpha rhythm), without focal interictal epileptiform activity or slowing during video and EEG monitoring.
(3) Ictal EEG had to show generalized onset of seizures during video and EEG monitoring.
(4) Normal neurological exam and absence of mental retardation (IQ $>$ 70).
(5) No definite risk factors for symptomatic epilepsy.
(6) Brain MRI had to show no focal abnormalities of the cerebral cortex. Diffuse cortical or cerebellar atrophy was not an exclusion criteria, since they can be a consequence of GTC seizures or medical therapy.[11,12]

Pre- and post-surgical data

Chart review was performed (for neuropsychological tests vide infra). Pre-operative evaluations in all patients included history and neurological examination, scalp video–EEG monitoring and brain MRI. Some of the subjects also had pre-operative Wada test and brain Fluorodeoxyglucose F18-positron emission tomographic (FDG-PET). The post-surgical evaluations included neurological examination, scalp EEG and brain MRI in all patients. A few of the patients had post-operative video–EEG monitoring with intracranial electrodes. The purpose was to investigate for an underlying focal abnormality exposed by the interruption of the anterior corpus callosum.

Acute surgical complications were investigated in the in-patient records and chronic complications from out-patient records and neuropsychological data.

Quality of life (QOL) — a score on a scale from 1 to 10, 10 being the best score possible and 1 the worst — was compared between pre-surgical and most recent post-surgical results.

We registered changes in the treatment after surgery, including modification of antiepileptic drugs, placement of vagal nerve stimulator (VNS) and additional epilepsy surgeries. Seizure frequency was plotted over time from last visit before the procedure until last follow-up. Also new types of partial seizures were noted and related to post-surgical scalp EEG results.

Seizure frequency before and after corpus callosotomy was based on seizures reported during office visits with treating neurologist. We defined pre-surgical seizure frequency as the average number of seizures per month the last year before surgery and post-surgical seizure frequency as the average number of seizures the last year up to the most recent visit (if post-surgical follow-up less than 1 year it was averaged over the number of months of follow-up). Although the focus was on GTC seizures, myoclonic and absence seizures were also compared before and after surgery, but only if these had occurred during the year before surgery.

Neuropsychological tests

Post-operative neuropsychological testing, performed 3 months—1 year after the procedure, was compared to pre-operative findings. The following cognitive domains were included:

(1) General cognitive functioning: verbal, performance and full scale intelligence (IQ scores from the Wechsler Adult Intelligence Scale, verbal IQ (VIQ), performance IQ (PIQ) and full scale IQ (FSIQ), respectively).
(2) Language skills: confrontation naming from the Boston Naming Test (BNT).
(3) Attention and working memory: Digit Span (DS) and Wisconsin Card Sorting Test—number of categories (WCST).
(4) Verbal memory: California Verbal Learning Test, total and delayed recall (CVLT-TL and CVLT-LD) and Logical Memory subtest of Wechsler Memory Scale-III, delayed recall score (LMD).
(5) Non-verbal memory: Visual Reproduction Subtest of Wechsler Memory Scale-III, delayed recall scores (VRD).

Scores that were one standard deviation (S.D.) below the mean for age-matched peers were regarded as impaired. A change of half a S.D. or more after surgery in any single test was interpreted as significant.

Statistics

Continuous variables were analyzed in a univariate fashion using the paired *t*-test. Categorical dichotomous variables were analyzed using the sign test.

Results

Pre-surgical data

Nine patients, seven men, all right handed, fulfilled entry criteria. Mean age at epilepsy onset was 12.5 years (range 2—18 years) and mean age at the time of surgery was 37.9 years (range 22—48 years). Three of the patients had a family history of epilepsy. Two patients reported previous febrile convulsions. All neurological examinations were normal.

All subjects had failed at least four AED, including valproate, phenytoin, carbamazepine and phenobarbital or primidone (see Table 1).

Table 1 summarizes the ictal and interictal scalp EEG.

Three patients had Wada test and all three were left hemisphere dominant for language.

FDG-PET was performed in three patients with no focal or lateralized findings in two while one (patient eight) had right temporo-parietal hypometabolism.[13]

Post-surgical data

The initial surgical procedure was anterior CC in all patients, sectioning the anterior two thirds of the corpus callosum. Post-operative brain MRI in each case confirmed the post-surgical changes to this area. One (patient three) had completion of the CC 3.5 years later, another (patient five) had right frontal resection 2.5 years later and two (patients four and nine) had vagal nerve stimulator (VNS) placed six and one years later, respectively, because of persistence of GTC seizures after the anterior CC.

Seven subjects had long-term post-CC video—EEG monitoring with subdural strip electrodes (all except patients two and six). These recorded from the dorsolateral and mesial frontal areas, parietal lobes as well as variable amounts of the temporal lobes. All recordings showed scattered bihemispheric spikes and spike—wave discharges of variable localization. No seizures were observed during the video—IEEG recordings and no focal resections were performed after the initial anterior CC based on the post-operative IEEG recordings.

There was transitory decrease in verbal output noted in most patients lasting typically only a few days, sometimes with associated motor neglect

Table 1 Pre-operative data

ID	MRI	Inter-ictal EEG	Ictal EEG	Seizure frequency	Previous medications
1	Cerebellar atrophy	3.5–5 Hz, S/W	GFA	G: 4.5	Carbamazepine, felbamate, gabapentin, phenobarbital, phenytoin, primidone, valproate
2	Normal	3.5–5 Hz, S/W	3.5 Hz, S/W, GFA	G: 7; A: 0; M: 2	Carbamazepine, clorazepate, ethosuximide, phenobarbital, phenytoin, valproate
3	Normal	3–7 Hz, poly-S/W, GPFA	GFA	G: 10; A: 30; M: 30	Carbamazepine, ethosuximide, felbamate, phenobarbital, phenytoin, valproate
4	Normal	4 Hz, S/W	GFA	G: 1.3; A: 0.5	Carbamazepine, felbamate, gabapentin, methosuximide, phenobarbital, phenytoin, primidone, valproate, zonisamide
5	Small white matter lesions	3–4 Hz, S/W	3.5 Hz, S/W, GFA	G: 3.5; A: 3	Carbamazepine, lamotrigene, phenobarbital, phenytoin, topiramate, valproate
6	Normal	3.5–5 Hz, S/W	GFA	G: 7; A: 0; M: 0	Carbamazepine, felbamate, meberal, phenobarbital, phenytoin, primidone, valproate
7	Cerebellar atrophy	4 Hz, S/W, GPFA	4 Hz, S/W, GFA	G: 1.5; A: 30; M: 0	Carbamazepine, ethosuximide, felbamate, lamotrigene, phenobarbital, phenytoin, topiramate, valproate
8	Mild diffuse atrophy	3.5 Hz, S/W	GFA	G: 10; A: 60; M: 30	Carbamazepine, gabapentin, mesantoin, phenobarbital, phenytoin, valproate
9	Mild diffuse atrophy	4–5 Hz, poly-S/W	4–5 Hz, poly-S/W	G: 12; A: 0; M: 0	Lamotrigene, phenytoin, primidone, topiramate, valproate, zonisamide

S/W: generalized spike and wave discharges; GFA: generalized fast activity; G: generalized tonic–clonic seizure; A: absence seizure; M: myoclonic seizure; seizure frequency refers to mean number of seizures during the last year prior to corpus callosotomy. See text.

Table 2 Demographic data

ID	Age	Sex	Epilepsy onset (years)	Family history	Febrile seizures	IQ
1	46	m	9	–	+	73
2	22	m	18	–	–	83
3	39	m	15	–	–	93
4	31	m	15	–	–	107
5	44	f	2	+	–	90
6	37	m	5	–	–	75
7	40	m	14	+	–	76
8	48	m	12	+	–	83
9	34	f	15	–	+	93

Patient 3 had only verbal IQ tested. All other scores are for full scale IQ. Please also see text.

affecting left leg. This was thought to be due to traction of the frontal lobe during surgery and always disappeared completely within a week. One (patient seven) had after anterior CC a prolonged disconnection syndrome lasting for 2 months affecting the left upper extremity. Patient three who had completion of CC after the initial anterior CC, suffered a permanent disconnection syndrome consisting of independent left arm and leg movements. In spite of this after the surgery he was able to live independently and work for the first time.

QOL measures were available for eight patients and seven patients had measures both before and after CC. The post-surgical scores showed a non-significant improvement (dependent paired *t*-test, $p = 0.057$). Mean pre-surgical and post-surgical scores were 4.1 (range 1–7, $n = 7$) and 6.5 (range 2–8, $n = 8$).

Two patients had new onset of partial seizures after CC: one (patient five) had secondarily GTC seizures documented by video and both scalp and intracranial EEG. She had right frontal lobe resection with up to now no further improvement. Another (patient seven) reported simple partial motor seizures until five months after the surgery, undocumented by video and EEG monitoring.

Routine scalp EEG after surgery showed new unilateral epileptiform discharges in seven patients and bilateral synchronous SWC in three patients, one without epileptiform activity and focal and/or bilateral frontal focal slowing in all patients (see Table 2). There was no correlation between lack of bilateral synchronous epileptiform discharges and better response to CC.

All patients remained on antiepileptic drugs after the surgery. Three were on one drug, four was on

Table 3 Outcome data

ID	EEG	Seizure frequency	GTC reduction (%)	Current medication	Follow-up (years)
1	S/W + focal spikes	G: 0	100	Carmazepine	4.3
2	Focal spikes	G: 2.8; A: 0; M: 2	60	Valproate	0.6
3	S/W + focal spikes	G: 1.2 (4); A: 82 (0); M: 30 (1)	88	Lamictal, valproate, phenobarbital	8.7
4	Focal spikes	G: 1.3; A: 0.5	0	Felbamate, lamotrigene (VNS)	6.6
5	Focal spikes	G: 1; A: 0	71	Phenobarbital, phenytoin, lamotrigene, phenytoin	2.5
6	S/W	G: 0.3; A: 0; M: 0	95		10.3
7	None	G: 0.6	61	Lamotrigene, topiramate	6.9
8	Focal spikes	G: 0.1; A: 5; M: 0.1	99	Lamotrigene	7.2
9	Focal Spikes	G: 2.7; A: 0; M: 0	78	Phenytoin	1.4

S/W: generalized spike and wave discharges; seizure frequency refers to mean number of seizures per month during last year; G: generalized tonic clonic seizure; A: absence seizure; M: myoclonic seizure. Patient 3 had later completion of corpus callosotomy, numbers in parenthesis are after completion. Patient 4 has VNS (vagus nerve stimulator), but with no change in seizure frequency since implant. See text.

two and two on three drugs. Six patients were on a drug that had not been tried prior to surgery. This was either lamotrigene or topiramate or both (see Table 3). These agents were not available at the time of surgery in any of the cases. Patient 4 had vagus nerve stimulator (VNS) implanted after surgery, but this had meanwhile been turned off due to lack of effect. Only minor medication changes occurred within the first post-surgical year, and none of the new medications noted at the last follow-up were started during this time. The first post-operative year was when the seizure reduction was most notable (see Fig. 1). We found no relationship between medication change and change in seizure frequency.

Mean pre-operative frequency of GTC seizures per month was 6.3 (range 1.3—12). One of the patients had only had GTC seizures. Eight of the subjects had a history of absence seizures and five within the year prior to CC with a mean frequency of 24.7 (range 0.5—60) per month. Six patients had a history of myoclonic seizures and three within the last year before surgery with a mean frequency of 20.6 (range 2—30) per month.

Post-operatively, the mean frequency of GTC seizures was 1.1 per month (range 0—2.5). Eight patients had more than 50% decrease of GTC seizures and five had more than 80% reduction. One patient was seizure free during the last year, but all patients had had at least one GTC seizure after surgery. There was a significant reduction of GTC seizures after surgery ($p < 0.005$). Fig. 1 shows the fluctuation of GTC seizure frequency over time in the post-operative period. Some patients were initially seizure free and then later recurred. The most notable decrease took place during the first post-operative year.

Among the five patients who reported absence seizures the year before surgery three improved (of these two became seizure free) and two worsened slightly (one became seizure free after completion of CC). Mean post-surgical frequency was 12.5 (range 0—82), a nonsignificant decrease.

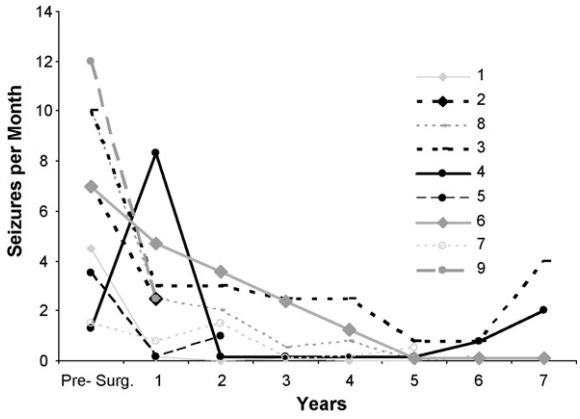

Figure 1 Post-surgical generalized tonic—clonic seizures. There is an impression that the greatest decrease in frequency of generalized tonic—clonic seizures occurred over the first few months and up to a year after corpus callosotomy. Seizures per month refers to the mean number over the last year (or maximal time) of follow-up. All ID numbers correspond to Tables 1—3 and the two individual cases.

Of the three patients with myoclonus the year prior to surgery one had more than 95% decrease while two stayed the same (one had more than 95% decrease after completion of CC).

Mean time from surgery until last follow-up was 5.4 years (range 0.6—10.3 years). One (patient two) died from sudden death in epilepsy (SUDEP) 9 months after the surgery, all others had follow-up of more than one year.

Neuropsychological data

See Table 4 for individual IQ results. FSIQ scores were within the range of normal, with an average of 87.5 (range 75—107), VIQ average 90.2 (range 73—128) and PIQ average 83.9 (range 70—95).

Table 4 Neuropsychological data

Test	Gen. Cog.: IQ (V/P)	Speech: BNT	Executive function		Verbal memory		Non-verbal memory: WMS-VRD
			WCST	Digit span	CVLT-TL/LD	WMS-LD	
Pre-surg. subjects	8 (9/8)	9	8	8	9/9	9	9
Pre-surg. non-impaired	6 (8/5)	2	8	2	5/3	4	7
Post-surg. subjects	4 (4/6)	6	5	5	5/5	6	6
Improved	2 (0/3)	4	1	0	3/3	3	3
No change	1 (3/3)	2	3	0	1/1	3	3
Worse	1 (1/0)	0	1	0	1/1	0	0

Neuropsychology testing results before and after corpus callosotomy. IQ (V/P) shows full scale IQ and verbal and performance IQ; WCST: Wisconsin Card Sorting Test (Categories); CVLT-TL/DR: California Verbal Learning Test-Total Learning and Delayed Recall; WMS-LD: Wechsler Memory Scale (III)-Long Delay; WMS-VRD: Wechsler Memory Score-Visual Reproduction Delayed. See text for details.

All patients had pre-operative testing while six had post-operative test results available. The cognitive areas with most impaired patients in the pre-operative testing were language and verbal memory while attention/working memory had mixed results and general cognitive functioning and non-verbal memory had a majority of non-impaired. Table 3 shows change in the neuropsychological test results after as compared to before surgery. Of 10 cognitive measures, 6 showed post-operative improvement, 3 stayed the same and 1 worsened. No individual showed a greater number of performance declines than good outcomes (improvement/no change). The BNT had a statistically significant improvement in mean scores on a dependent, paired *t*-test ($p < 0.05$).

Of the ten measures, three (Boston Naming, Digit Span, and Visual Reproduction) representing distinct cognitive domains (language, attention, and non-verbal memory) showed no decline. A Sign Test comparing the number of subjects who worsened or obtained good outcome (improved/no change) on each measure demonstrated that these three were the only measures that showed a reliable improvement across the two testing points (chi-square critical value = 3.84, d.f. = 1, $p < 0.05$).

Two individual cases

See Table 2 for additional information.

Patient 6

A 37-year-old right handed man began to experience absence, myoclonic and generalized tonic–clonic (GTC) seizures at the age of 5. The absence and myoclonic seizures disappeared at an early age (he thinks before 10 years old), but he continued having 5–12 GTC seizures per month despite treatment. There were several seizure related injuries, including an occipital skull fracture. Post-surgical full-scale IQ was 83. He had no new cognitive complaints. Prior to surgery he was unemployed and lived with his parents. At last follow-up he was living by himself and working full time.

Patient 4

A 39-year-old right handed man had his first GTC seizure during sleep at age 15. He developed frequent daytime GTC seizures without warning, absence seizures lasting up to 1 min with "freezing up" and alteration of consciousness and abrupt falls to the ground with brief alteration of consciousness which were associated with multiple injuries. He had mandibular fractures, loss of teeth, and a head injury with brain swelling. Seizure semiology did not change subsequent to injuries. Exam was remarkable for mild bilateral ptosis and decreased hearing after head trauma. After the anterior CC he had a total of nine GTC seizures in the next 4 years, and during two of these years there was only one GTC seizure per year. The absence seizures were unchanged. Prior to the surgery he was unmarried, living with his parents and working full time. At last follow-up he was married, had three children and worked full time. He reckoned his personality unchanged.

Discussion

This is the first systematic report on the effects of callosal section for refractory IGE in humans (10). We found a decrease in GTC, absence and myoclonic seizure frequency after CC, with eight of nine patients experiencing benefit. The benefit of surgery generally occurred immediately, although variability in response was noted over time. The finding of efficacy of CC in IGE is important since some patients with IGE remain refractory to medical treatment.

The finding that seizures in IGE are improved, but not abolished after CC could shed some light on the role of the corpus callosum in the pathophysiology of IGE. The rostral corpus callosum connects mainly the prefrontal, premotor and motor cortex[14] and these areas have particularly rich interhemispheric connections[15] representing the majority of fibers. The greater tendency for symmetric areas to develop from focal unilateral to bilateral synchronize discharges could be due to the topography of the callosal fibers.[16] Corpus callosum may form part of a circuit that is involved in IGE, in addition to thalamus and subcortical pathways (intraoperative electrocorticograms from patient undergoing corpus callosotomy have shown that spike and wave discharges are not conducted across the corpus callosum[17]). The importance of the thalamus as a pace maker of discharges was showed in 1947 by Jasper and Droogleever-Foruynn[18] who could provoke generalized spike and wave discharges by stimulating midline and reticular nuclei of the thalamus in the cat. Other examples of the pivotal role of thalamus in IGE, include the recording of spike and wave discharges in thalamus and cortex in a patient during absence seizure,[19] selective H_2O PET activation of the thalamus during absence seizures in a human subject,[20] thalamic decrease of NAA/Cr ratio on MR spectroscopy in patients with IGE as compared to other brain areas and normal controls,[21] and the action of ethosuximide, a drug specific for absence seizures, on low threshold T-type Ca(2+) current in thalamocortical and nucleus reticularis thalami (NRT) neurons. On the other

hand, evidence that the cerebral cortex can trigger similar seizures include absence seizures seen after symmetric bilateral application of a proconvulsant substance to the frontal cortex[22] and GTC seizures in the feline penicillin model.[23] Therefore, fibers of the anterior corpus callosum, could form part of a circuit in IGE that facilitates absence and GTC seizures by connecting the frontal lobe cerebral cortices to each other, but this circuit may not be necessary for seizures in IGE since there are subcortical pathways.

Most of our patients developed new focal discharges after the procedure and a minority had new onset partial seizures. This has previously been reported in patients with symptomatic generalized epilepsy with tonic and atonic seizures after callosotomy.[24,25]

The neuropsychological data and the clinical examinations do not reveal any permanent cognitive problems associated with anterior CC. Language improved significantly and in none of the cognitive domains did deterioration outnumber improvement or no change.

Permanent cognitive neuropsychological side effects of anterior corpus callosotomy reported in the past includes mutism in cases where language is poorly lateralized,[26] difficulty with new learning[27] and focal deficits in case of prior focal abnormalities.[28] Fortunately, these complications were not observed in our series.

Transient post-surgical non-dominant hemiparesis or hemineglect and mutism[29] also occurs, and was observed in most of our patients but these occurrences were brief. A permanent disconnection syndrome was only seen in one of the patients with completion of corpus callosotomy,[30] while one patient who had anterior corpus callosotomy reported a transient disconnection syndrome.

Since this review is retrospective, the results are open to other influences occurring in this period than the surgical procedure. Changes in medical treatment and other therapeutic interventions could have affected the seizure frequency after surgery. Several antiepileptic drugs known to be effective for generalized epilepsy became available only after the surgery in most of the cases, and six of the patients were on antiepileptic drugs at last follow-up which had not been tried before surgery. However, in all the cases, this medication was begun after the beneficial effect of the CC had occurred. In no cases did the new drugs abolish seizures. Therefore, although possibly beneficial, these medication changes do not account for the improvement. Likewise, patient four had VNS implant because of persistent seizures, but this did not help and the devise was turned off.

Lastly while our entry criteria aimed to exclude patients with symptomatic generalized epilepsy, a partial or cryptogenic mechanism cannot be completely excluded in our patients. Patient five (Table 2) presented with secondarily GTC seizures after the initial anterior CC with a focus in right frontal lobe, but further resection in this area has not helped up to now.

Use of CC for apparent refractory IGE depends on the selection of candidates and a careful patient counseling. First, a benign remitting epilepsy syndrome needs to be ruled out. Second, thorough trials of virtually all available antiepileptic drugs with potential efficacy in IGE need to be undertaken to prove intractability. Third, the seizure disorder needs to be associated with severe disability or potential for injury to justify undergoing a neurosurgical procedure. Both patient and physician need to keep in mind that anterior CC is primarily a palliative procedure and that a cure cannot be expected.

With these provisos, anterior CC is an effective and reasonably safe procedure in carefully selected patients with apparent, refractory IGE.

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