SUBTENTORIAL TUMORS AND OTHER LESIONS: AN ELECTROENCEPHALOGRAPHIC STUDY OF 121 CASES[1]

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A number of investigators (Daly *et al.* 1953; Steinmann and Tönnis 1953; Pimenta *et al.* 1954; Corsino *et al.* 1954; Negri 1955; Simek and Stein 1955; Broglia and Postir 1956; Kreindler *et al.* 1956; Van der Drift 1957; Dumermuth 1958; Hess 1958) have focused their attention specifically on EEG evidence of posterior fossa lesions since our earlier reports (Bagchi *et al.* 1951, 1952; Bagchi and Bassett 1952). Questions in regard to electro-clinical correlations, EEG lateralizing signs, and mechanisms of "distant discharges" have been raised. This interest underscores the need for further analysis of EEG and clinical findings in a large series of surgically verified cases in order to delineate the range of variability and to identify EEG similarities from case to case which may be helpful in localization and differential diagnosis. Also, because of the location of these tumors in relation to cerebellar and brain stem systems intimately related to cerebral functions, a unique opportunity is offered to analyze the effect of disruption of these systems on electrocortical events. Such an analysis is obviously complicated, however, by secondary phenomena, *i.e.*, increased intracranial pressure, tentorial herniation, vascular changes and alterations in electrolyte balance, so that critical evidence may be offered only by statistical analysis of large groups of cases that had thorough EEG and clinical examinations or by individual examples in which the complicating factors appear negligible.

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METHODS

121 subjects with verified infratentorial lesions (113 tumors) were studied. 65 were males, 56, females. Average age was 33 years with a range of 1 to 66 years. Twenty patients were less than 13 years of age. Data about the following symptoms and signs were tabulated for each patient: headache, nausea and vomiting, decreased visual acuity, blurring and diplopia, gait disturbance, mental impairment, cranial nerve involvement, nystagmus, cerebellar signs, sensory deficit, pyramidal tract involvement, and signs of increased intracranial pressure including papilledema, ventricular dilatation, spreading of sutures, increased spinal fluid pressure, sellar erosion, and increased dural tension at operation. Duration of illness was estimated from the onset of the initial symptom referable to the lesion. Routine skull X-rays were available for 74 cases, ventriculograms for 90, arteriograms for 20, pneumoencephalograms for 4 and autopsy findings for 28. All lesions were verified by surgical or postmortem examination. The types of lesions represented are shown in Table I.

95 of the patients were examined with a Grass 8 channel electroencephalograph. 26 of the cases were studied early in the series with Grass 3 and 6 channel machines. A routine study (8–10 scalp leads) was carried out in 7, a routine with additional leads in 16, a semilocalizing study in 3 (12 – 14 leads), and a full localizing tracing in 95 (19 or more leads and as many as 26 monopolar and bipolar short and long distance montages). Lead placements, montages and reasons for adequate work-up (which is often most essential), have been previously described (Bagchi 1955a, b; Small *et al.* 1961). A so-called monopolar "posterior fossa" work-up

TABLE I

Types of subtentorial lesions

	No.
Astrocytoma	22
Acoustic neurinoma	14
Medulloblastoma	13
Metastatic carcinoma	12
Meningioma	11
Ependymoma	8
Hemangioblastoma	8
Glioma	7
Spongioblastoma polare	4
Leptomeningeal sarcoma	3
Pinealoma	2
Sarcoma	2
Stenosis of aqueduct	2
Cyst	2
Cholesteatoma	2
Papilloma	1
Abscess	1
Teratoma	1
Miscellaneous	6
Total	121

(Bagchi *et al.* 1952; Bagchi 1955b) was performed in 69 cases for sampling episodic biparasagittal bursts on the one hand and lateral bursts of both sides on the other for their asynchronous appearance (interareal parasagittal-lateral variability). All records were satisfactory. Artifact was moderate in 12 tracings, marked in 1. The series was collected between 1942 and 1959.

Background activity was tabulated as dominant (more than 75%) alpha, beta, theta or delta; alpha activity plus medium or high voltage fast activity (fast activity above 25% but less than 50%); mixed alpha and theta (theta above 25%, but less than 50%); mixed theta and delta (delta above 25% but less than 50%); mixed alpha and delta (delta above 25% but less than 50%); mixed alpha and beta (beta above 25%), and mixed theta and beta. Bilaterally synchronous episodic burst activity (over 50% increase in relation to background voltage) was placed in a separate category. This was subdivided into alpha, theta and delta (including masked and abortive spike and wave forms) ranges and anterior and posterior locations. Occasionally it was difficult to distinguish between background and burst activity. Appearance of burst activity 4 – 6 times in a record with a duration of less than 1 sec each was termed minimal (1); 7 – 10 times, moderate (2); and more than 11 times, marked (3). Interhemispheric shifting bursts of alpha, theta and delta between homologous regions (sometimes called independent bursts in the literature but not to be confused with diffuse random discharges) was evaluated separately for frontal, anterior and interaural temporal, parietal, posterior temporal and occipital regions, using the same classification of degree as with synchronous bursts. Overall dominance of one hemisphere in terms of episodic amplitude increase and/or wave-length decrease of burst or background activity was also noted down separately. If the difference between the two hemispheres in these respects was less than 5 per cent it was termed none, 5–25 per cent minimal, 25–50 per cent moderate, and over 50 per cent marked. Emphasis of biposterior or bianterior disturbance, and antero-posterior variability and parasagittal lateral variability (asynchronous appearance of bursts) were also graded as minimal, moderate and marked. Overall emphasis of one hemisphere in terms of episodic amplitude increase and/or wave length decrease when occurring *contralateral* to the side of the lesion, and also depth diagnosis by EEG without structure identification given preoperatively or retrospectively were further entered in the tabulation. Statistical treatment (chi square) of data was generally based on groupings contrasting those cases with moderate or marked abnormalities with the remainder.

TABLE II

Background EEG patterns in subtentorial lesions

	No.	%
Alpha	18	14.9
Alpha and beta	20	16.5
Alpha and medium and high voltage beta	5	4.1
Alpha and theta	31	25.6
Alpha and delta	0	0
Beta	1	0.8
Theta	18	14.9
Theta and beta	9	7.4
Theta and delta	14	11.6
Delta	5	4.1
Total	121	100.0

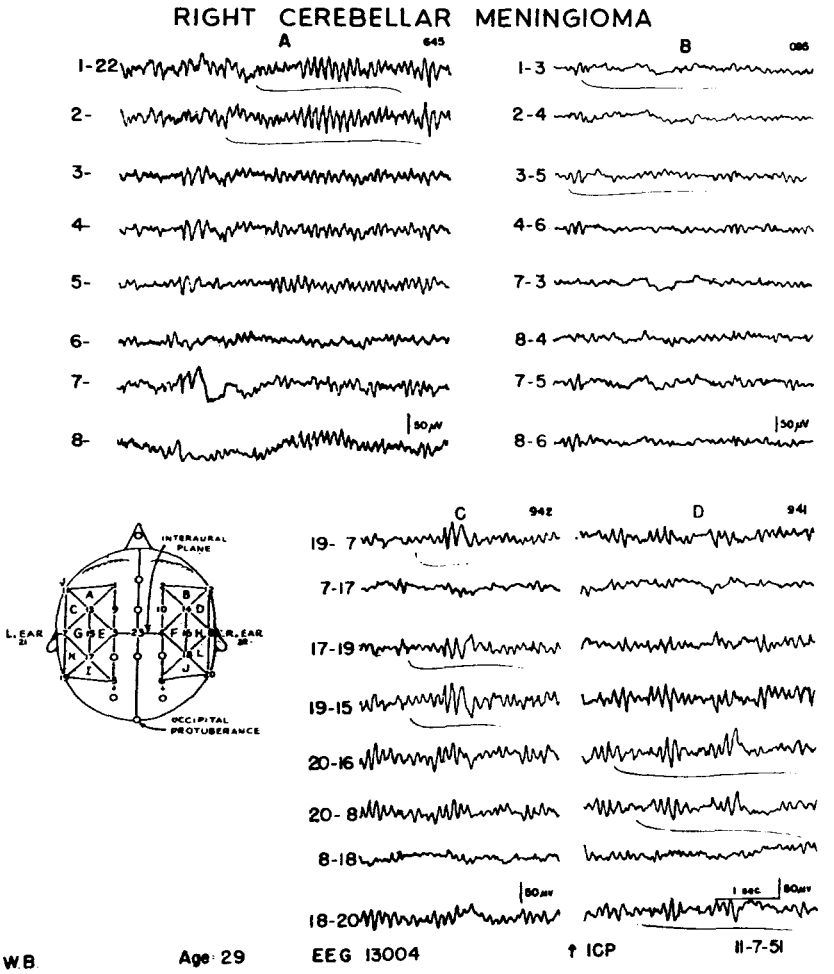

Fig. 1

W. B. Right cerebellar meningioma arising from the tentorium. Bioccipital headaches 2 months, visual blurring, diplopia, decreased visual acuity 2 weeks, ataxic gait, no mental changes, marked bilateral papilledema, right facial paresis, lateral nystagmus, right more than left. Ventricular dilatation (symmetrical). IV ventricle could not be evaluated. Sharp angle of aqueduct of Sylvius.

Panel A: displacement of voltage of alpha to biprefrontal region, underlined (lines 1 and 2), single sharp theta in left temporal (line 7) in monopolar recording. Panel B: low to medium voltage delta undulations slightly more on left, underlined (lines 1, 3, 5, 7). Panels C and D: alpha bursts on left (lines 1, 3 and 4), alpha burst shift to right (lines 5, 6, 8). The shift occurs within 10 sec. Note alpha background in spite of marked papilledema. Overall findings in this case were not marked. In different epochs, including triangulations, shifting alpha bursts and rare theta signs, also biprefrontal alpha bursts in absence of certain clinical conditions (see page 196), were preoperatively interpreted as not being consistent with a superficial lesion and as giving inconclusive evidence of a deep midline condition. Only on review of the EEG record later was left contralateralization determined.

RESULTS

EEG characteristics

Only 3 records were normal, 18 were classified as borderline. Of the remaining 100 cases (82.6%), 31 were classified as mildly abnormal, 55 moderately abnormal, and 14 markedly abnormal.

Characteristics of the basic background

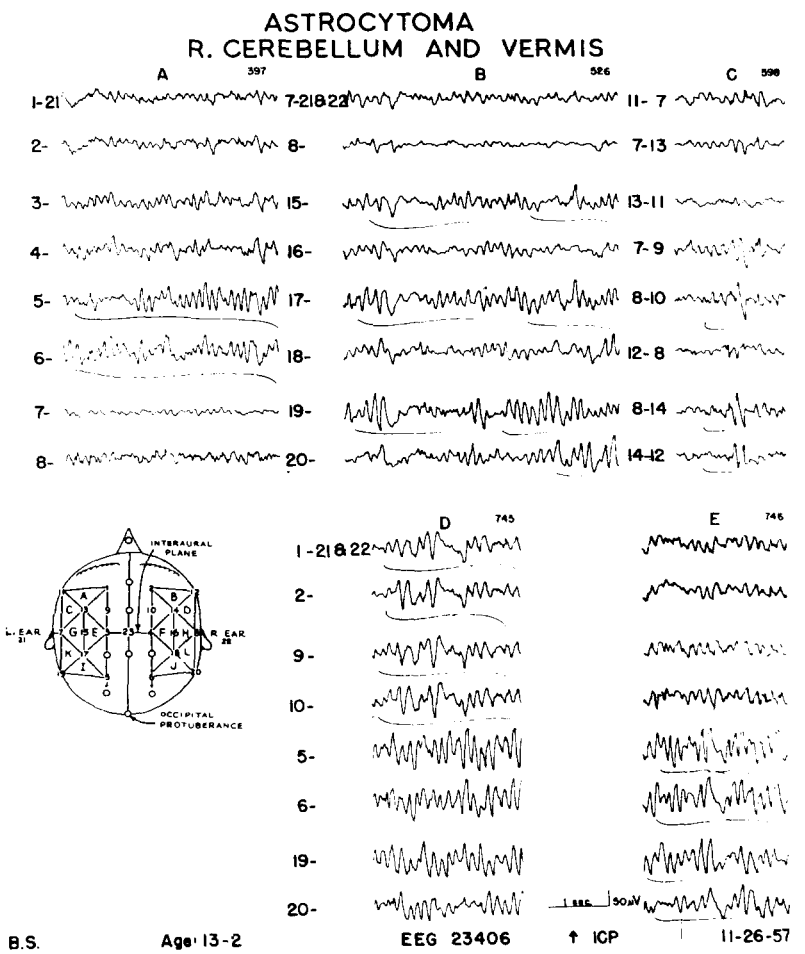

Fig. 2

B. S. Large solid astrocytoma in right cerebellum and vermis. About a year's history of frontal headache, nausea and vomiting, diplopia for 4 days, horizontal nystagmus, also rotary nystagmus with upward gaze, other cranial nerves intact, slight blurring of the temporal disc but no papilledema, bilateral tinnitus and some dizziness but no vertigo, increased or normal cerebrospinal fluid pressure at different times. No sensory or pyramidal tract findings, mild dyssynergia of right hand. Bilateral internal carotid arteriogram normal. EEG (11–26–57) suggested a deep lesion (see later). Patient was discharged as posterior fossa brain tumor suspect to return in four weeks, though at one time the possibility of pseudo-tumor cerebri was raised. In Boston, 3 months previously, ventriculogram, EEG and other studies were reported normal. Patient was operated on elsewhere two weeks following discharge from this Hospital when her condition got worse with the operative findings mentioned above (courtesy: Dr. A. W. Farley, Saginaw, Mich.).

Panel A: masked or patent thetas and deltas in occipitals, underlined (lines 5 and 6). Panel B: alpha bursts and delta undulations, on left, underlined (lines 3, 5 and 7). Panel C: spike bursts mostly on right-shift (lines 4, 5, 7 and 8). Panels D and E: bianterior alpha bursts with single sharp delta having superimposed waves and slow decay (lines 1, 2, 3 and 4) without biposterior bursts and similar biposterior bursts without bianterior bursts within 10 sec from the same scalp leads and interconnected ear reference leads (lines 5, 6, 7 and 8). This phenomenon has been termed biantero-posterior variability. These and other signs with left sided statistical emphasis (not adequately shown here) were preoperatively considered, in absence of certain clinical conditions (page 196), as being inconsistent with an upper convexity lesion and consistent with a deep lesion including one in posterior fossa probably on the midline and/or right (contralateralization).

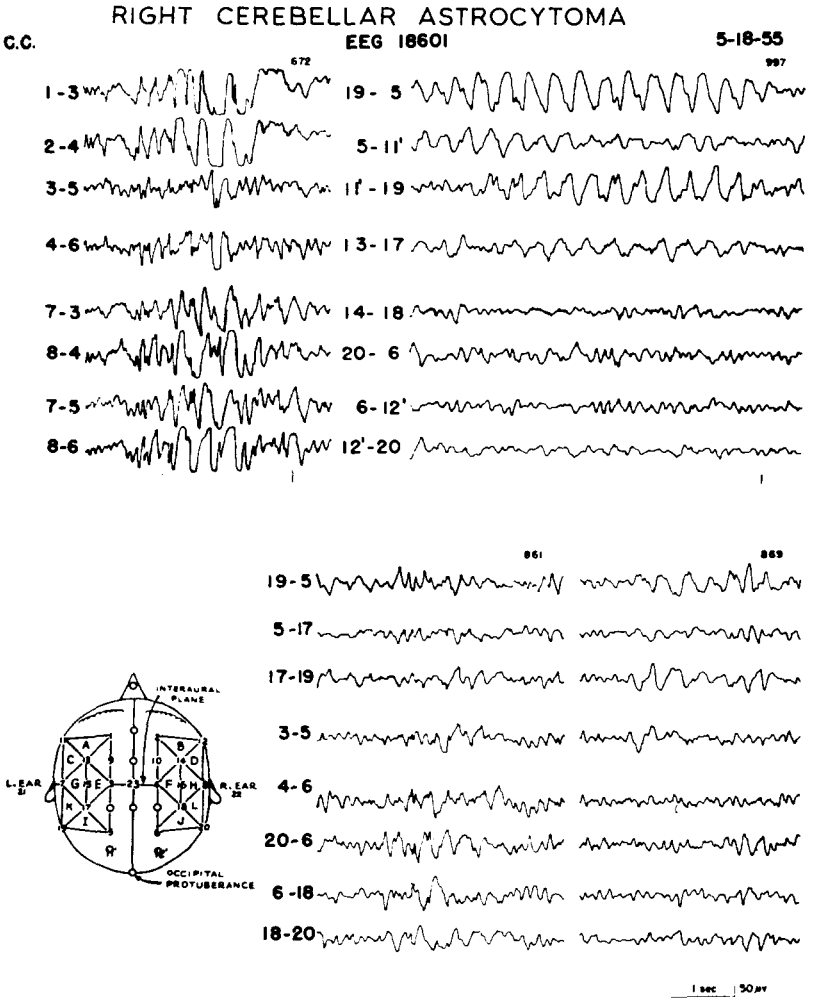

Fig. 3

C. C. Right cystic cerebellar astrocytoma, golf ball size, away from midline, extending through incisura. Age 13. Nausea and vomiting, headaches 3 months, staggering, no cranial nerve signs, no nystagmus, no pyramidal tract or sensory signs. Papilledema not known. Symmetrical ventricular dilatation. Deformation of posterior III ventricle. Increased cerebrospinal fluid pressure and protein. Dural tension at operating room not known.

Panel 1: bilateral sharp theta-delta bursts with some abortive spike wave formations. Panel 2: long 3/sec bursts in left posterior quadrant (lines 1, 2, and 3), not seen in right posterior quadrant (lines 6, 7 and 8). Panel 3: some bilaterality but mainly right posterior quadrant high voltage theta shift in right posterior triangle (lines 5, 6, 7 and 8). Panel 4: left posterior quadrant delta emphasis in left posterior triangle (lines 1, 2 and 3). In absence of certain clinical conditions (see page 196) a large proportion of bilateral bursts, anteriorly and posteriorly, with overall left sided emphasis and right sided shift was interpreted before operation as suggesting the non-existence of an upper convexity lesion and the presence of a deep lesion, including one in subtentorial structure, probably on the right (contralateralization).

patterns are listed in Table II. Specific abnormal features of the records are classified in Table III. Prior to surgery and/or postmortem and regardless of neurological signs, 69.4 per cent of the cases were correctly considered on the basis of EEG characteristics (see discussion page 196) to

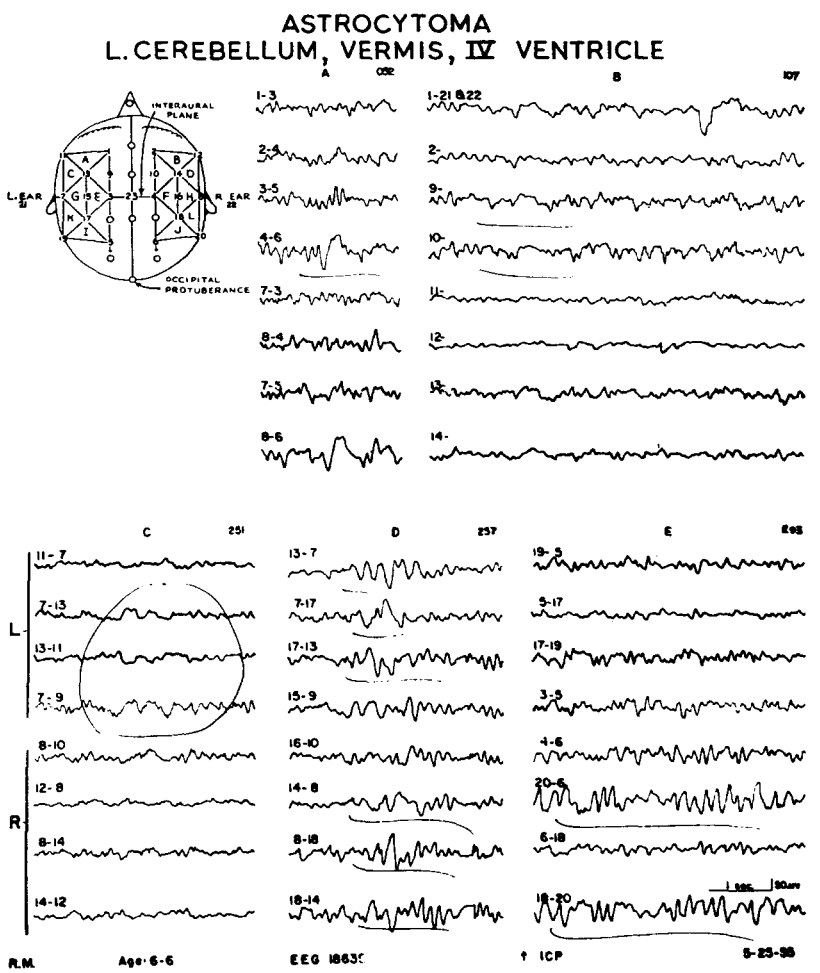

Fig. 4

R. M. Left cerebellar and midline astrocytoma. Occipital headache, vomiting for one year, lack of pep, marked bilateral papilledema, questionable disturbance in gait and coordination, no sensory signs, nystagmus, pyramidal tract signs, other cranial nerve signs. Separation of sutures, dilatation of the ventricular system, deviation of IV ventricle to right, anterior deviation of aqueduct of Sylvius. Herniation of tonsils to C_2. Panel A: right occipital abortive spike-waves, underlined (lines 4 and 8). Panel B: bipremotor serial theta-delta and sharp discharges (lines 3 and 4). Panel C: left sided deltas, ringed in left antero-lateral triangle and additional linkage (lines 2 to 4). Panel D: multilobar complex bilateral sharp theta-alpha-single delta bursts. Panel E: strong right posterior triangle (right posterior temporal lead) mixed theta-delta bursts (lines 6 and 8) very much less in corresponding triangle on left (lines 1 and 3). In absence of certain clinical conditions (page 196) these and other findings not shown were preoperatively considered inconsistent with an upper convexity lesion and consistent with a deep lesion, including one in posterior fossa probably on midline and left (contralateralization).

be consistent with a deep level disturbance or lesion, including one in the posterior fossa and not in the upper convexity. This is exclusive of 9.1 per cent with retrospective depth diagnosis. A false cerebral localization (homolateral or contralateral temporal) was made in 10 per cent of the cases in which EEG depth signs were absent or minimal.

Minimal, moderate or marked incidence of bilaterally synchronous bursts of frequencies in

alpha, theta and delta ranges (Fig. 1, 2, 3 and 4) occurred in 110 cases (90.9%) (separately indicated in Table III). These were observed slightly but not significantly more often anteriorly (84.3% *vs.* 74.4%). Alpha and theta bursts tended to be more prominent anteriorly, delta bursts posteriorly.

TABLE III

Characteristic EEG abnormalities in subtentorial lesions ($n = 121$)

	No.	%
Synchronous biposterior patterns		
Sharp alpha bursts	18	14.9
Sharp theta bursts	50	41.3
Mixed theta-delta bursts	53	43.8
Delta bursts	65	53.7
Synchronous bianterior patterns		
Sharp alpha bursts	43	35.5
Sharp theta bursts	66	54.5
Delta bursts	54	44.6
Non-synchronous shifting hemispheric bursts between homologous regions		
Alpha or spikey bursts	28	23.1
Theta	53	43.8
Delta	59	48.7
Frontal regions	44	36.4
Interaural temporal regions	73	60.3
Parietal regions	23	21.7[1]
Occipital regions	36	29.8
Posterior temporal regions	39	38.6[2]
Overall theta-delta disturbance		
Anterior	83	68.6
Posterior	74	61.2
Anterior-posterior variability	55	45.5

[1] Of 106 cases studied with lateral parietal leads.
[2] Of 101 cases studied with posterior temporal leads.

Alpha, theta and delta bursts shifting between homologous areas of the hemispheres occurred in 92 of the patients (76.0%; separately indicated in Table III). This was most commonly identified as theta or delta activity although shifting alpha or spikey bursts were not uncommon (Fig. 2, 3 and 4). Shifting abnormalities were particularly prone to occur between temporal areas. Variability between bianterior and biposterior parasagittal (Fig. 2) regions was noted in 55 cases (45.5%). If moderate to marked burst activity was present posteriorly, similar activity was present anteriorly in 61.8 per cent of the cases. In the remaining cases considerable disparity existed between the degree of anterior and posterior disturbance. In 47 cases EEG abnormality of any type was more marked anteriorly; in 37 cases it was more prominent posteriorly.

EEG findings and clinical variables

It was found that in extensive recordings unilateral overall emphasis of burst activity in terms of episodic wave-length decrease and/or voltage increase frequently emerges, even though shifting interhemispheric emphasis is common (Fig. 1, 2, 3 and 4). The one-sided EEG emphasis or dominance occurred in 112 of the 121 cases (Table IV). The relationship between overall EEG emphasis and tumor site is more frequently contralateral than ipsilateral. This contralateralizing emphasis of EEG signs was found in 37 of 50 (74.0%) laterally situated parenchymatous cerebellar tumors, with or without midline involvement. If all lateral posterior fossa tumors are considered (adding cerebellopontine angle tumors, other

TABLE IV

EEG bursts in subtentorial lesions ($n = 121$)

Overall emphasis (Voltage increase and/or wave length decrease)	Location of lesion									
	Cerebellar			*Brain stem*			*Extraparenchymatous*[1]			
	R, RM	L, LM	M	R, RM	L, LM	M	R, RM	L, LM	M	
Left	27	5	6	2	0	12	9	6	2	69
Right	5	10	6	0	4	12	2	3	1	43
None	1	2	2	0	0	1	1	2	0	9
Totals	33	17	14	2	4	25	12	11	3	121

[1] Includes 18 cerebellopontine angle neurinomas.
R – right-sided, RM – right-sided and midline, L – left-sided, LM – left-sided and midline, M – midline.

extraparenchymatous tumors and tumors of the medulla, pons and IV ventricle), the percentage was only slightly less (55 of 79, 69.6%). The possibility that such a distribution would occur by chance was less than 0.01 ($X^2 = 9.8$). If all tumors, both lateral and midline ($n = 121$), are considered the statistical significance of the relationship between EEG lateralization and lesion site remains beyond the 0.01 level of confidence. EEG lateralization also tended to be opposite the side of cerebellar neurological signs (18 of 28, 64.3%) and of lower cranial nerve signs (24 of 35, 68.6%). Patients with lateralizing sensory signs were few but these were ipsilateral to the EEG abnormality in 6 of 7 cases. No relationship emerged for pyramidal tract signs. For all cases with unilateral or unilateral and midline tumors, the EEG lateralizing emphasis was left sided in 49, right in 24. Thus, the right sided preponderance of EEG abnormalities reported by Van der Drift (1957) in his entire series is not borne out.

It was more common for overall homolateral EEG emphasis to occur with cerebellopontine angle tumors (8 out of 18) than with lateral parenchymatous cerebellar (9 out of 50) lesions ($X^2 = 5.3$, $P < 0.05$). The five lateral tumors occurring in patients without signs of increased intracranial pressure all had contralateralizing EEG emphasis. Three of the tumors invaded the pons and right cerebellar hemisphere (2 astrocytomas, 1 glioma). The two remaining tumors involved the right cerebellar hemisphere only (1 bronchogenic carcinoma, 1 meningioma).

All patients but one with ipsilateral EEG emphasis had increased intracranial pressure. The exception had secondary cerebral involvement by the tumor, ipsilateral to the cerebellar lesion. Unfortunately, midline tumors ($n = 39$) also have shown lateralized EEG emphasis (Table IV). The reason for this is obscure. The question as to whether they were precisely midline tumors or had undetected or unreported lateral extension causing such EEG emphasis could not be answered.

Cerebellopontine angle and IV ventricle tumors tended to produce lesser degrees of abnormality than primary cerebellar lesions. Of the 3 normal EEG's, 2 occurred with cerebellopontine angle tumors. Primary pontine gliomas in the absence of increased intracranial pressure may show only minor EEG alteration.

Inspection of Table V reveals that clinical evidence of mental disturbance was appreciably related to degree of EEG abnormality. The relationship between duration of symptoms and

TABLE V

Clinical variables *vs.* EEG classification in subtentorial lesions

EEG	Symptoms less than one year			Mental signs		Increased intra-cranial pressure		Large tumor			Malignant tumors			Extra-parenchymatous tumors	
	n	*No.*	%	*No.*	%	*No.*	%	*n*[1]	*No.*	%	*n*[2]	*No.*	%	*No.*	%
Normal	3	0	0.0	0	0.0	2	66.7	3	2	66.7	3	1	33.3	1	33.3
Borderline	18	9	50.0	3	16.7	13	72.2	16	13	81.3	15	9	60.0	5	33.3
Mildly abnormal	31	18	58.1	7	22.6	27	87.1	27	21	77.8	31	14	45.2	7	22.6
Moderately abnormal	55	41	74.5	23	41.8	50	90.9	43	38	88.4	52	33	63.5	9	17.3
Markedly abnormal	14	6	42.9	9	64.3	13	92.9	13	10	76.9	12	8	66.7	2	16.7
	121	74	61.2	42	34.7	105	86.8	102	84	82.6	113	65	57.5	24	21.2
$P(X^2)$*	= 0.05 (3.8)			< 0.01 (9.6)		NS		NS			NS			NS	

[1] Cases with size known.

[2] Tumors only.

* Four fold X^2, normal, borderline, mildly abnormal *vs.* moderately and markedly abnormal.

NS - Not significant 0.05 level of confidence.

degree of EEG abnormality also was significant, although at a lower level of confidence. Unexpectedly, a significant relationship between signs of increased intracranial pressure and degree of overall EEG alteration was not readily demonstrable. However, the minor but consistent trend in the percentages of cases with evidence of increased intracranial pressure for the five EEG categories should be noted. Tumor size, malignancy and extra- or intraparenchymatous location of the tumor were not significantly related to overall EEG abnormality.

Table VI relates significant degrees of background slowing, bianterior and biposterior synchronous burst activity, and shifting interhemispheric emphasis with selected clinical variables. Mental signs appear to relate more with background slowing than with the other EEG patterns whereas increased intracranial pressure tends to be associated with bianterior burst activity. Patient age did not contribute significantly to these relationships. Patients with symptoms of less than 1 year duration (suggesting a relatively rapidly advancing or critically situated lesion)

TABLE VI

EEG signs *vs.* clinical variables in subtentorial lesions

			Background slowing		$P(X^2)$	Bianterior bursts		$P(X^2)$	Biposterior bursts		$P(X^2)$	Shift		$P(X^2)$
Symptoms less than one year			*No.*	%		*No.*	%		*No.*	%		*No.*	%	
(All cases)	Yes	(74)	51	68.9	NS	34	45.9	NS	39	52.7	< 0.05 (4.0)	19	25.7	NS
	No	(47)	26	55.3		17	36.2		16	34.0		12	25.5	
< 13[2] (20)	Yes	(16)	15	(94.5)		5	(31.3)		12	(75.0)		2	(12.5)	
	No	(4)	4	(100.0)		3	(75.0)		3	(75.0)		2	(50.0)	
Mental signs														
(All cases)	Yes	(42)	32	76.2	<0.05 (4.4)	22	52.4	NS	19	45.2	NS	14	33.3	NS
	No	(79)	45	57.0		29	36.7		36	45.6		17	21.5	
< 13 (20)	Yes	(7)	7	(100.0)		4	(57.1)		5	(71.4)		2	(28.6)	
	No	(13)	12	(92.3)		4	(30.8)		10	(76.9)		2	(15.3)	
Increased intracranial pressure														
(All cases)	Yes	(105)	70	66.7	NS	49	46.7	<0.05 (5.2)[1]	47	44.8	NS	29	27.6	NS[1]
	No	(16)	7	43.8		2	12.5		8	50.0		2	12.5	
< 13 (20)	Yes	(16)	15	(93.8)		8	(50.0)		13	(81.2)		4	(25.0)	
	No	(4)	4	(100.0)		0	(0.0)		2	(50.0)		0	(0.0)	
Large lesions														
(Cases with	Yes	(84)	53	63.1	NS	35	41.7	NS	39	46.4	NS	19	22.6	NS
size known)	No	(18)	10	55.6		6	33.3		8	44.4		6	33.3	
< 13 (16)	Yes	(14)	14	(100.0)		6	(42.9)		11	(78.6)		2	(14.3)	
	No	(2)	2	(100.0)		2	(100.0)		2	(100.0)		2	(100.0)	
Malignant tumors														
(All tumors)	Yes	(65)	44	67.7	NS	27	41.5	NS	28	43.1	NS	17	26.2	NS
	No	(48)	27	56.2		17	35.4		23	48.0		12	25.0	
< 13 (20)	Yes	(11)	10	(90.9)		4	(36.7)		7	(63.6)		1	(9.1)	
	No	(9)	9	(100.0)		4	(44.4)		8	(88.9)		3	(33.3)	
Extra-parenchymatous tumors														
(All tumors)	Yes	(24)	10	41.7	< 0.01 (5.8)	10	41.7	NS	8	33.3	NS	4	16.7	NS[1]
	No	(89)	61	68.5		34	38.2		43	48.3		25	28.1	
< 13 (20)	Yes	(0)	0	(—.–)		0	(—.–)		0	(—.–)		0	(—.–)	
	No	(20)	19	(95.0)		8	(40.0)		15	(75.0)		4	(20.0)	

NS - Not significant 0.05 level of confidence

[1] With Yates correction

[2] Cases under 13 years of age.

were found to exhibit moderate to marked biposterior burst activity more often than patients with symptoms of longer duration. This was not so in deep supratentorial cerebral lesions (Small *et al.* 1961). The relationship appears to be of borderline significance, however, inasmuch as if children are not included the X^2 does not quite attain the 0.05 level of confidence. Biposterior burst activity occurred significantly more often in children than adults. Patients with extraparenchymatous tumors were not as likely to have background slowing as those without. However, this relationship appears to reflect largely the relative frequency of background slowing in children as compared with adults coupled with the lack of occurrence of extraparenchymatous tumors in children.

DISCUSSION

Common characteristics of EEG disturbances associated with subtentorial lesions suggest that inclusion in the differential diagnosis of infratentorial tumors or other processes may be warranted in patients having some of these EEG depth signs:

1. Anterior, posterior or diffuse bilaterally synchronous bursts of delta, theta, alpha.
2. Shifting interhemispheric bursts between homologous regions.
3. Parasagittal bianterior-biposterior variability.
4. Parasagittal lateral variability.
5. Overall unihemispheric emphasis of wave length decrease or voltage increase.
6. Antero-posterior genuine (not instrumental) phase difference.

A specific diagnosis of infratentorial abnormality is not possible from the EEG evidence alone inasmuch as these signs may occur in other conditions as pointed out previously (Bagchi *et al.* 1952), and also in deep cerebral lesions (Bagchi 1952, 1955a, b; Small *et al.* 1961). However, although EEG signs of an infratentorial lesion may be duplicated in patients with epilepsy, cerebral concussion or contusion, inflammatory disease, degenerative processes, hypertensive encephalopathy, extra-cerebral diseases, etc., these may not be under serious consideration in individual cases. If they are, depth diagnosis by EEG becomes uncertain or inapplicable. The differential diagnosis rather has frequently been between a posterior fossa lesion and such conditions as a psychiatric disorder, Ménière's syndrome or acute labyrinthitis, frontal lobe tumor and pseudo-tumor. In specific instances, the EEG findings may clearly favor an infratentorial process over the other possibility. In some cases, a posterior fossa lesion was the primary diagnosis but the clinical findings were minimal or equivocal. EEG evidence consistent with a posterior fossa lesion led to further definitive studies and final correct diagnosis.

Predominance of EEG abnormality contralateral to the side of the cerebellar lesion has been reported in sporadic cases or in small groups by a number of workers (Rheinberger and Davidoff 1942; Hoefer *et al.* 1946; Cohn 1949; Daly *et al.* 1953; Pimenta *et al.* 1954; Streifler and Feldman 1952; Broglia and Postir 1956). Contralateral emphasis of the EEG abnormality occurred far beyond the chance level in our series, confirming and extending the significance of these and our earlier observations. This suggests that increased intracranial pressure, which would usually exert bilaterally symmetrical effects, cannot be solely responsible for lateralizing EEG abnormalities existing in these patients. That increased intracranial pressure may actually obscure the issue of contralateralizing emphasis of EEG signs is suggested by these two observations: (*a*) all (18) patients with *ipsilateral* EEG emphasis had signs of increased intracranial pressure except one, and (*b*) 5 cases with unilateral cerebellar tumors without significant evidence of increased intracranial pressure had EEG signs well lateralized to the opposite cerebral hemisphere. The lack of evidence of a major relationship between signs of increased intracranial pressure and degree of EEG abnormality (Table IV) also tends to discount secondary supratentorial pressure effects as being of prime importance in the matter of contralateralizing EEG emphasis.

It is our belief that the preponderance of abnormal signs over the cerebral hemisphere opposite the side of the cerebellar lesion reflects an alteration of neural activity in crossed pathways. Experimentally, predominant contralateral cerebral effects of electrical stimulation of

neocerebellar structures have been described (Walker 1938; Capraro and Gualtierotti 1940; Kreindler *et al.* 1957) although homolateral responses have also been mentioned. Walker's evidence indicates that the cerebello-dento-rubro-thalamic pathway is necessary for contralateral motor cortex effects. In this connection the demonstrated ascending connections (cat) between the cerebellar nuclei and the centrum medianum nucleus should be noted. Projections have been described from the interposed nucleus to center median and central medial and lateral nuclear groups of the internal medullary lamina (Cohen *et al.* 1958; McMasters and Russell 1958). The disturbance of balance between cortico-ponto-cerebellar influences on the one hand and cerebello-thalamo-cortical influences on the other may also be a factor in this contralateralization (Dr. Elizabeth C. Crosby, personal communication). The blocking of afferent inflow at lower level may also play a role in causing generalized EEG disturbance, but a question may be raised about this explanation in cases where clinical sensory signs are not disturbed ($n = 114$).

Snider (1956) has suggested that ascending connections mediated cerebral "activation" by cerebellar electrical stimulation and pointed out that widespread cerebellar areas discharge into centrum medianum, centralis medialis, centralis lateralis, medial thalamic, and reticular nuclei. On the other hand electrical stimulation has not invariably evoked an "activation" pattern. Cooke and Snider (1953) observed, in rare instance, the production of slow waves in the electrocorticogram of cat with cerebellar electrical stimulation. Wetzel and Snider (1957) have recorded changes in the scalp EEG during electrical stimulation of the cerebellum in man. Frequency and amplitude changes varied depending upon the cerebellar site stimulated.

Other mechanisms responsible for abnormal cerebral events undoubtedly play significant roles. Daly *et al.* (1953) have emphasized that of increased intracranial pressure, believing that its effect is produced through pressure on the walls of the third and lateral ventricles. Such a concept, however, fails to explain those cases with signs of increased intracranial pressure without significant EEG changes. Furthermore, the effect of increased intracranial pressure may not only be on the walls of the third and lateral ventricles, but within the posterior fossa as well. Such secondary events as early ascending tentorial herniation, compression of blood vessels, edema, and alteration of electrolyte balance are in all probability significant although their exact roles are, as yet, not fully defined (Steinmann and Tönnis 1953).

Cordeau (1959) has reported that "monorhythmic frontal delta" activity was lateralized to the side of the cerebellar lesion in 5 of 8 cases. This appears to conflict with our findings. However, we have likewise noted that overall EEG contralateralizing emphasis is not an invariable finding, not appearing particularly in cases with signs of increased intracranial pressure or with marked EEG abnormalities. It should be further emphasized that in our series frequency and amplitude differences in all homologous cerebral areas of both rhythmic and arrhythmic patterns were utilized as "lateralizing signs." Recognition of this and of differences in sampling length, electrode number, and montage set-up may be important in comparing our results with other studies.

SUMMARY AND CONCLUSION

121 cases of verified infratentorial lesions, including 113 tumors, have been studied qualitatively and statistically in relation to a large number of clinical and EEG variables. Six significant and about two dozen non-significant relationships between the variables have been noted. For example, there is significant relationship between bianterior bursts and increased intracranial pressure (IICP) but none between degree of EEG abnormality and IICP; there is a significant relationship between degree of EEG abnormality and mental signs but none between the former and malignancy or extraparenchymatous tumors or tumor size.

An attempt has been made to answer certain questions raised in the literature. In line with our previous report, EEG signs of depth diagnosis without structure identification, but ruling out upper convexity lesion, have been confirmed in 78.5 per cent of cases within the limits of certain clinical reservations. The EEG depth signs which

have less clarity than upper convexity focal EEG signs include bilaterally synchronous bursts of all types, including hypersynchronous alphas, anteriorly, posteriorly or both; interhemispheric shifting bursts between homologous areas, commonly temporal; overall emphasis of voltage increase and/or wave length decrease of the bursts in one hemisphere; parasagittal bianteroposterior variability; parasagittal lateral variability. That some of these signs may be sometimes missed without long sampling and adequate montages has been pointed out. False localization to the cerebrum in absence of EEG depth signs occurs in 10 per cent of the cases. About 83 per cent of the lesions show various types, degrees, and distributions of EEG abnormality.

Statistically valid relationship has been established between overall contralateral EEG emphasis (73%) in lateral infratentorial lesions, with or without midline involvement. Conformity in terms of lateralization between EEG and some neurological signs, and the clarity of EEG contralateral signs in cases without signs of increased intracranial pressure have been noted in addition. Contralateral EEG emphasis has been found often to occur more in parenchymatous lesions than in extraparenchymatous lesions. The mechanism of generalized disturbance and of contralateral emphasis has been discussed.

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