Electroencephalographic changes associated with cerebellar hemisphere tumours

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The value of electroencephalography in the assessment of lesions in the region of the third ventricle and posterior fossa is limited, but certain patterns may serve to confirm clinical impressions. To assess the help that the electroencephalogram provides, a review of 60 cases with structural lesions in the region of the third ventricle or posterior fossa was undertaken. There appeared to be a relationship between the electroencephalographic patterns and the distribution of the lesions within those regions.

That there is a relationship between the cerebral rhythms and cerebellar lesions has been shown by many authors. An early report by Walker (1938) indicated that stimulation of the cerebellar hemispheres produced a change in the cortical resting rhythms from the motor areas of anaesthetized cats. Dow, Fernandez-Guardiola, and Manni (1962) have recently reported on cerebellar influences in cortical epilepsy in rats. It is apparent that the cerebellum is capable of inhibiting or activating the cerebri. The influences of the various cerebellar regions vary with the parameters of stimuli as well as with changes in the interplay and feedback from reticular and cerebral activity. Bagchi, Lam, Kooi, and Bassett (1952) expressed the opinion that electroencephalographic changes in association with posterior fossa tumours were sufficiently distinct to be of help in confirming the existence of a deep or posterior fossa lesion. They properly emphasized that careful correlation should be made with clinical data. A further study of this relationship was undertaken by Daly, Whelan, Bickford, and MacCarty (1953). They described the principal abnormality as being rhythmic, bilaterally synchronous slowing. Their data suggested that there was no absolute relationship between the anatomical site of the lesion and the distribution of the electroencephalographic abnormalities. They concluded that increased pressure within the third ventricle was of major importance in the genesis of this abnormality. The changes as they described them were helpful in making a distinction between lesions in the vicinity of the third ventricle or posterior fossa, and lesions involving primarily the convexity of the cerebral hemispheres. Support for their conclusions was offered by Dow (1956). He also was inclined to attribute the changes seen to obstructive effects and subsequent dilatation of the third ventricle. However, Liversedge and Emery (1961) described similar electroencephalographic changes in association with cerebellar degenerative lesions, presumably in the absence of any distension of the ventricular system. It was their contention that an alteration of the cerebello-central connexions was important in the production of the electrical patterns associated with posterior fossa lesions. More recently Hasegawa and Aird (1963) have reviewed their findings in subtentorial lesions. They too describe records with bilaterally simultaneous bursts of slow activity but indicate that 68% of recordings were normal, borderline, or only mildly abnormal. Their group with burst activity showed only a slightly higher incidence of increased intracranial pressure than that group without burst activity. According to them, subtentorial lesions ‘activate’ a ‘pacemaker’ mechanism.

METHODS

We reviewed and interpreted the electroencephalograms of 60 patients who had tumours involving the posterior fossa or third ventricle. The records reviewed were knowingly chosen on the basis that there was a tumour involving those areas, provided the record had been technically satisfactory and had been recorded before any surgical intervention. It was also required that there be histological confirmation of the tumour, either by operation or post-mortem examination. All of the lesions involved either the posterior fossa or third ventricle structures, alone or with associated involvement of other intracranial areas. A further division was made into two main subgroups of (a) involvement of cerebellar hemispheres, or (b) non-involvement of cerebellar hemispheres. In one-half of the cases we were able to confirm that there was direct or secondary involvement of the cerebellar

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Of the 60 patients, 32 had a structural lesion of the cerebellar hemispheres, and 28 had intact cerebellar hemispheres. Within these two groups there was a predominance of males, there being 22 in the group with involvement and 20 in the group without involvement. This is partially explained by the use of records from a veterans' hospital for part of the material. The age range was similar in the two groups, and the distribution of ages is compared in Table II.

### Table II

<table>
<thead>
<tr>
<th>Age Group (yr.)</th>
<th>Hemisphere Involved (N=32)</th>
<th>Hemisphere Not Involved (N=28)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0—15</td>
<td>11</td>
<td>11</td>
</tr>
<tr>
<td>15—30</td>
<td>4</td>
<td>4</td>
</tr>
<tr>
<td>30—60</td>
<td>9</td>
<td>8</td>
</tr>
<tr>
<td>60+</td>
<td>8</td>
<td>5</td>
</tr>
</tbody>
</table>

Within the two groups a further distinction was made between cases with or without evidence of distension of the third ventricle. Cases were included in the group with distension of the third ventricle provided this was confirmed by post-mortem or surgical studies, by contrast studies, or when there was clinical evidence of increased intracranial pressure and the anatomical location of the tumour was such that the third ventricle would, perforce, be involved. Clinical signs of increased intracranial pressure alone were not accepted as criteria as these could be produced by a blockage of flow from the lateral ventricles.

The electroencephalograms were all done on either a Grass model III eight-channel electroencephalograph or a 16-channel Grass model IV electroencephalograph. Seven electrodes were placed over each hemisphere. These were arranged to record from the frontal, motor, parietal, occipital, and anterior and posterior temporal regions bilaterally. The majority of recordings were done with a variety of bipolar montages, although in some instances common reference electrodes were used.

All the electroencephalograms were reviewed separately and in conference by the authors. No gross discrepancies arose as to the proper classification of records. There was a high degree of correlation between the original and review interpretations, although statistical methods were not used to substantiate this. The selection of cases for this study naturally may have introduced some bias. However, it is worth emphasizing that all records were reviewed without knowledge as to the specific nature and location of the disease process and without knowledge as to whether there was or was not an associated distension of the third ventricle.

The records were classified as either normal, arrhythmic, continuously dysrhythmic, or episodically dysrhythmic. The arrhythmic records were those that showed random slow forms (0·5–4 c.p.s.) of no predictable frequency, occurring asynchronously if bilateral, or confined to one or more foci. The continuously dysrhythmic records were those that contained frequencies ranging from 2 to 7 c.p.s. that were nearly continuous or only briefly intermittent. Included in this same category were those records which showed the more distinct types of abnormalities such as spikes, sharp waves, or a combination of spike and slow-wave activity. The episodically dysrhythmic records contained bursts of bilaterally synchronous or simultaneous high amplitude rhythmic slow activity with frequencies generally ranging around 2 to 4 c.p.s. These wave forms could predominate in one hemisphere or the other, and in addition were susceptible to inhibition by eye opening and augmentation by hyperventilation. These bursts were often in addition to other types of abnormalities. Such records are labelled burst dysrhythmia in Tables III, IV, and V. Figure 1 is an example of this type of episodic dysrhythmia and Fig. 2 of the more continuous type of dysrhythmia. It is worth noting, in connexion with the electroencephalogram illustrated in Fig. 2, that there was distension of the third ventricle in this patient. This type of dysrhythmia disappeared following the Torkildsen procedure in a fashion similar to the case described by Daly, Whelan, Bickford, and MacCarty (1953).

### Discussion

Tables III, IV, and V, in which the electroencephalographic patterns and pathological lesions are correlated, suggest that there is a pattern suggestive of involvement of the cerebellar hemispheres. An analysis of the relative incidence of burst records in the cerebellar hemisphere group versus the non-hemisphere group reveals a highly significant difference beyond the 0·001 level. Analysis of those cases with or without distension of the third ventricle reveals no significant difference. An analysis of the cerebellar hemisphere lesions with and without distension of...
FIG. 1. 'Burst' dysrhythmias. (a) A 9-year-old boy with a cystic astrocytoma of the right hemisphere. (b) A 54-year-old woman with an astrocytoma involving the left hemisphere and portions of the vermis.

FIG. 2. An example of the more continuous rhythmic slowing associated with distension of the third ventricle. These tracings are from a 26-year-old man with an epidermoid tumour in the third ventricle. The second portion of the tracings was made following ventriculostomy.
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the third ventricle suggests that the ideal circumstance for the production of these intermittent dysrhythmic type records is a combination of involvement of the cerebellar hemisphere plus some distension of the third ventricle.

Of those cases with burst-type dysrhythmias, in which anatomical investigation of the distribution of the lesions was possible, there appeared to be a common involvement of an area immediately lateral to the dentate nucleus in the cerebellar hemisphere, in addition to whatever other areas might be involved. In six cases with clear clinical signs suggesting neocerebellar disease, and where the anatomically confirmed lesion was primarily in the neocerebellum, typical burst-type dysrhythmic electroencephalograms occurred. These were in contrast to three cases with high pons or mid-brain involvement pathologically, in two of which there could be no question of involvement of the brachium conjunctivum, but in none of which was there the typical burst-type dysrhythmia. These findings are substantiated by Hasegawa and Aird (1963) who indicate that burst-type abnormalities were much less frequently seen with brain-stem lesions as compared to lesions in the cerebellum or extraparenchymatous regions such as the cerebellar-pontine angle.

The brachium conjunctivum represents the major efferent outflow of the cerebellum with many fibres originating from the cerebellar nuclei (Jansen and Brodal, 1940, 1954). Both the phylogenetically newer ventrolateral dentate nucleus as well as the phylogenetically older dorsal medial portion contribute fibres to it. Other contributions come from other cerebellar nuclei. Study of these structures in monkeys by Carpenter and Stevens (1957) does suggest some degree of lamination of these tracts within the brachium conjunctivum. Nevertheless, their relationship is so intimate it is probable that all fibre systems would be equally vulnerable to interruption by lesions in extracerebellar regions. Lesions within the cerebellar hemisphere conceivably could act more selectively, thus disrupting any balance of inhibitory and activating influences arising within the cerebellar centres and acting upon the thalamus or 'pacemaker' centres. Obviously this is only a possible mechanism suggested by our data and not conclusively established. Disturbances of other inhibitory and activating structures and relationships, such as occur with toxic states and convulsive disorders, produce similar electroencephalographic changes.

Our data do suggest that distension of the third ventricle favours the appearance of burst-type records, but that the essential feature is involvement of the cerebellar hemispheres. We do not claim that the distribution of the pattern helps to determine whether or not the cerebellar hemisphere is involved primarily or secondarily. The pattern is simply a non-specific one which arises following destruction of certain regions in the cerebellar hemispheres. From a clinical standpoint, this offers little additional help, although conceivably it aids in distinguishing between the deeper midline lesions in the baso-frontal and anterior third ventricle region from those involving posterior fossa structures primarily. In our series it was also of help in the assessment of metastatic diseases, such a pattern in the presence of obvious arrhythmic foci suggesting multiple lesions involving both supratentorial and infratentorial areas.

SUMMARY

The electroencephalograms of 60 patients with involvement of the third ventricle and/or posterior fossa by structural lesions have been reviewed. In those instances where there was a destructive pro-
cess involving the cerebellar hemisphere, a burst-type of intermittent dysrhythmic electroencephalogram appeared in 72%. The bursts tended to appear and end abruptly, being superimposed upon the pre-existing pattern. They were often suppressed by eye opening and accentuated by hyperventilation. The changes were of no lateralizing value. Such patterns appeared statistically to be related to lesions of the cerebellar hemisphere, although an associated distention of the third ventricle favoured their appearance. Distension of the third ventricle alone was a much less frequent cause. This pattern may be the result of a morphological abnormality which disrupts the normal relationships of the cerebellar-reticular-thalamic inhibitory and activating fibre pathways.

REFERENCES


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