VASCULAR MALFORMATIONS ASSOCIATED WITH TEMPORAL LOBE EPILEPSY*

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Several varieties of cerebral vascular malformations have been described.9 The cirroid or racemose angioma is the most common type and is familiar to most neurosurgeons.15 An infrequent vascular anomaly, the so-called “cryptic hamartoma,” consists of a small collection of vessels usually concealed in the depths of a sulcus.3,8,14 The hemangioma calcificans is a rare variety characterized by calcification in the temporal lobe.1,11,17

Most vascular malformations are related to a high incidence of epilepsy.9 Their frequent occurrence in the sylvian and rolandic areas results in sensory and motor seizures and occasional psychomotor epilepsy.15 These lesions, however, account for less than 5 per cent of the total pathology in selected series of psychomotor epilepsy.5,10

Usually vascular lesions are detected early in the course of preoperative studies of seizures.16 The present cases, however, were not discovered despite an intensive investigation. They represent an incidence of 11.4 per cent in a series of 88 temporal lobectomies performed on patients with psychomotor epilepsy.

PREOPERATIVE DATA

The significant clinical data are shown in Table 1. The sex distribution was 6 males and 4 females. The average age at onset of seizures was 18 years. Epigastric aura occurred in 60 per cent of the cases as compared to 36 per cent in the entire series of 88 cases.

All patients experienced psychomotor epilepsy and, in addition, 5 had generalized clonic-tonic seizures. Momentary confusion was common postictally. One patient experienced postictal aphasia.

The past histories were negative except for head trauma in Case 2, possible encephalitis in Case 6 and syphilis in Case 10.

Neurologic findings were usually within normal limits. Mental retardation occurred in 3 patients and was associated with left hemiparesis and atrophy in Case 9. Chorioretinitis was present in Case 10. Cerebrospinal fluid pressures and proteins were all normal.

The pertinent neuroradiologic data are shown in Table 2. Roentgenograms of the skull were normal except for ballooning and thinning of the right temporal squama in Case 1 and right hemicranial smallness in Case 9.

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Pneumograms usually showed some degree of general or temporal-horn dilatation. A cyst of the septum pellucidum was present in Case 1. In retrospect, Case 3 showed a slight deformity of the right anterior temporal horn. Carotid angiography was performed on 5 patients and all were within normal limits.

An analysis of the electrographic data is presented in Table 3. In 7 cases the preoperative electroencephalograms revealed unilateral temporal-lobe involvement. Five of them showed epileptiform activity well localized over the anterior portion of the right temporal lobe and 1 over the posterior portion. The sixth showed epileptiform activity over the anterior portion of the left temporal lobe.

Three out of 10 patients showed independent bitemporal epileptiform activity with maximal right-side involvement in 2 and left in 1. Electrographic evidence of rather widespread extratemporal involvement occurred in 2 of these cases.

The epileptiform activity in all instances consisted of high-voltage triphasic or diphasic sharp-wave discharges occurring sporadically or, less frequently, organized into brief bursts. They were very well localized over the temporal region in all records.

In the postoperative electroencephalograms there was a rather marked decrease in the amount of abnormalities, but all patients continued to show remaining epileptiform activity. Two of the unilateral cases became bitemporal.

Individual cases will now be presented.
<table>
<thead>
<tr>
<th>Case No.</th>
<th>Skull X-ray</th>
<th>Pneumoencephalogram</th>
<th>Carotid Angiogram</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Asymmetry</td>
<td>Focal Calcification</td>
<td>Pineal Shift</td>
</tr>
<tr>
<td>1</td>
<td>R. temporal squama thin and bulging, Fossa large</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>2</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>3</td>
<td>None</td>
<td>None</td>
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</tr>
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<td>4</td>
<td>None</td>
<td>None</td>
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<tr>
<td>5</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>7</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>8</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>9</td>
<td>R. hemi cranium small, Temporal fossa equal</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>10</td>
<td>None</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>Case No.</td>
<td>Background Activity</td>
<td>Focal Non-Paroxysmal</td>
<td>Focal Paroxysmal</td>
</tr>
<tr>
<td>---------</td>
<td>---------------------</td>
<td>----------------------</td>
<td>------------------</td>
</tr>
<tr>
<td>1</td>
<td>Normal alpha and diffuse 6-7/sec.</td>
<td>Irregular 4-6/sec. r. frontal &amp; temporal</td>
<td>Numerous sharp waves—RT-F8 (Pgt. T4)</td>
</tr>
<tr>
<td>2</td>
<td>Abnormal alpha with depression over r. hemisphere</td>
<td>2.5-5/sec. irregular slow waves—r. temporal</td>
<td>Sharp waves—RT-F8 (Pgt2)</td>
</tr>
<tr>
<td>3</td>
<td>Normal alpha</td>
<td>None</td>
<td>Sharp waves A2, F8, Pgt (maximal r.t. inferior)</td>
</tr>
<tr>
<td>4</td>
<td>Normal alpha</td>
<td>Irregular 4-7/sec. waves—anterior l. temporal</td>
<td>Sporadic sharp waves and spikes—RT-F7 (T2)</td>
</tr>
<tr>
<td>5</td>
<td>Normal alpha</td>
<td>None</td>
<td>None</td>
</tr>
<tr>
<td>6</td>
<td>Very little alpha, mainly diffuse slow waves</td>
<td>Diffuse slow waves—maximal r. hemisphere</td>
<td>Sharp and high voltage slow l. F7, T3, C3, Independent focus RT (F8, T6), maximal l. temporal</td>
</tr>
<tr>
<td>7</td>
<td>Poorly developed</td>
<td>Slow waves—r. temporal</td>
<td>Bursts diphasic sharp waves—RT (F8, T6)</td>
</tr>
<tr>
<td>8</td>
<td>Normal alpha and diffuse bilateral 2-7/sec.</td>
<td>3.5-6/sec. irregular slow waves—r. temporal</td>
<td>Frequent sharp waves—LT maximal F7, Pgt, occasional RT sharp wave</td>
</tr>
<tr>
<td>9</td>
<td>Normal alpha</td>
<td>4-6/sec. irregular slow waves—r. (some l.) temporal</td>
<td>Sharp waves—maximal RT (T4, F8) resting, Sharp waves LT (sleep)</td>
</tr>
<tr>
<td>10</td>
<td>Normal alpha</td>
<td>4-6/sec. irregular slow waves—r. temporal</td>
<td>Sharp waves—maximal RT posterior</td>
</tr>
</tbody>
</table>
CASE REPORTS

Case 3. A 33-year-old right-handed white female was in good health until aged 22 years when she experienced her first cerebral seizure. The seizures were characterized by an epigastric aura, automatism or generalized clonic-tonic movements and no postictal deficit. She averaged 2–4 seizures per month.

Neurological findings, cerebrospinal fluid pressure, protein and roentgenograms of the skull were normal. Pneumograms revealed slight dilatation and deformity of the right temporal horn. Electroencephalograms showed a right anterior inferior temporal-lobe focus.

During surgical resection of the right temporal lobe a hard, circular mass, measuring 1 by 1 by 1 cm., lying just beneath the cortex of the second temporal gyrus approximately 2.0 cm. from the temporal tip, was encountered and excised. The cortex overlying this lesion was elevated and brownish in color. There was sclerosis around the tip of the ventricle.

Postoperative electroencephalograms showed bitemporal epileptogenic foci. The patient has had fewer seizures in the 49 months postoperatively.

Comment. The malformation in this case (Fig. 1) is typical of the intracerebral variety. The right-sided anterior location and unilateral electrographic abnormalities were characteristic.

![Fig. 1. Case 3. Gross appearance of intracerebral type of lesion.](image-url)
Case 7. A 46-year-old right-handed white female was in good health until aged 24 years, when she experienced her first seizure. The seizures, occurring 2–3 times each month, were preceded by an epigastric aura. They were psychomotor in type without postictal deficit.

Neurologic findings, cerebrospinal fluid pressure and protein, roentgenograms of the skull and pneumograms were within normal limits. Electroencephalographic abnormalities were localized to the right anterior and middle temporal lobe.

No gross lesion was found during resection of the right temporal lobe. A meningocortical vascular lesion, measuring 0.5 by 0.5 by 0.5 cm., was discovered in the depth of a lateral temporal sulcus upon examination of the surgical specimen.

Postoperative electroencephalograms showed right temporal activity. The patient has been seizure-free for 44 months.

Comment. This particular case exemplifies the small meningocortical variety of vascular malformation. Clinically, no variations were found between patients with this lesion and patients with intracerebral vascular lesions.

Case 9. A 45-year-old right-handed white male was in good health until aged 3½ years when he experienced his first seizure. The seizures were not preceded by a warning. They were left-sided and Jacksonian motor in type. No postictal sensory or motor deficit occurred. At the age of 4 years a left hemiparesis was noted. This progressed to a left hemiatrophy and was associated with mental retardation. He also experienced automatisms and periodic changes in personality.

Neurologic examination revealed mental retardation, left hemiparesis, hemiatrophy and hyperreflexia. The cerebrospinal fluid protein and pressure were normal. Roentgenograms of the skull revealed a small right hemicranium. Pneumograms showed general ventricular dilatation which was greater on the right side. Right carotid angiogram was normal. Electroencephalograms disclosed bitemporal foci active on the right side.

During surgical resection of the right temporal lobe, a medium-sized sylvian plexiform arteriovenous malformation was seen (Fig. 2). This collapsed after ligation of two arteries arising from the middle cerebral group. The temporal lobe was atrophic and sclerotic.

Postoperative electroencephalograms showed a right temporal-lobe focus. During the 12 months following surgery, the patient has had an occasional seizure.

Comment. This case demonstrates an instance in which the malformation affected extratemporal areas of the brain. The location of the lesion in the sylvian fissure adjacent to the temporal lobe and precentral gyrus explains the combination of two different seizure patterns.

The following case is not included in this series but represents an example of sensory epilepsy resulting from a calcified arteriovenous malformation in the right parietal lobe.

Case P.L. A 19-year-old right-handed white female had experienced cerebral seizures since the age of 13 years. The seizures were characterized by a sensation of "an electrical shock" in the left wrist followed by numbness which spread up into her shoulder and then down into the left leg. Following this the patient would become unconscious for several minutes and, upon awaking, experience a 2- or 3-
minute period of left hemihypalgesia and hemiparesis. Neurologic examination disclosed no abnormalities.

Roentgenograms of the skull revealed a calcified lesion in the right parietal area. Clinical history, Mantoux skin test, roentgenograms of the chest, cervical-node biopsy, gastric and cerebrospinal fluid cultures for A.F.B. and guinea pig inoculations were all negative for tuberculosis. Roentgenograms of the skull taken in June 1946 and in April 1951 were compared and revealed increased density of calcification but no increase in size of the lesion. Electroencephalographic abnormalities were localized to the right parietal area.

At operation a yellow-white, hard circular mass, measuring 2 by 1.5 by 0.8 cm., in the right postcentral gyrus was completely removed (Fig. 3). Microscopic examination revealed an arteriovenous malformation with intense gliosis and calcification. The patient was followed for 2 years and during that time remained seizure-free.

**PATHOLOGIC ANALYSIS**

These vascular malformations may be divided into two categories: intracerebral and meningocortical. In this series the intracerebral variety occurred in 6 out of 10 cases. Fig. 4 represents a schematic drawing of the various lesions.

(a) **Intracerebral Lesions.** With the exception of Case 4, all of the 6 intracerebral lesions occurred in the right temporal lobe. Three were located in

Fig. 2. Case 9. Sylvian meningocortical malformation.
the mesial aspect of the lobe and 3 in the lateral portion. All were in the anterior 5.0 cm. of the temporal lobe.

Grossly this lesion consisted of a hard, circular, well-demarcated, greenish-brown mass surrounded by sclerotic white matter. Usually two or three medium-sized arteries entered the mass to serve as nutrient vessels. The average size of the lesions was 1 by 1 by 1 cm. The temporal lobe was sclerotic in all cases and in 2 cases, grossly atrophic.

Microscopically the intracerebral lesion consisted of irregular, dilated, vascular channels with typical and atypical elastic membranes. Venous channels were more numerous (Figs. 5, 6 and 7). The veins showed fibrous thickening of their walls. Venous thrombosis was present in several areas and in other areas partial and complete recanalization existed. Fibroelastic cushions and deposits of calcium in arterial walls were present but less frequently than the venous changes (Fig. 8).

A basophilic hyaline material was deposited between vessels while calcium was deposited concentrically in the lumen of the vessels. In Case 1 there was an area of bone formation (Fig. 9). In Case 4 numerous calcospherites were scattered throughout the brain around the malformation
Fig. 4. Schematic drawing of the various lesions. Lateral and cross sectional views are shown.

(Fig. 10). Granules of hemosiderin, areas of encephalomalacia and marked gliosis were present in the cerebral tissue at the periphery of the main lesion (Figs. 11 and 12).

(b) *Meningocortical Malformations.* Two of the 4 lesions in this category were of medium size and 2 were very small. All these lesions occupied the lateral surface of the temporal lobe in its anterior 5.0 cm. Three were located in the right lobe and 1 in the left (Fig. 4).

The medium-sized lesions consisted of a profuse conglomeration of tortuous, dilated veins lying in the sylvian fissure. In Case 9 two medium-sized arteries arising from the middle cerebral artery entered the inferior surface of this venous plexus (Fig. 2). Marked sclerosis and atrophy of the temporal lobe were present. In Case 10 the sylvian plexiform malformation exhibited ramifications over the frontal and temporal lobes (Fig. 13).
Fig. 5. Case 1. Abnormal vessels in an intracerebral lesion. Hematoxylin and cosin, X20.

Fig. 6. Case 6. Abnormal vessels with cavernous appearance. Gomery, X95.
Fig. 7. Case 2. Abnormal vessels in an intracerebral malformation showing marked thickness of the walls of the vessels. Hematoxylin and eosin, ×170.

Fig. 8. Case 1. Fibroelastic cushion in a vessel with deposits of calcium. Hematoxylin and eosin, ×150.
Nutrient vessels from the middle cerebral group supplied the abnormality. The temporal lobe was sclerotic.

The malformations in the other 2 cases consisted of small collections of vessels located in the depths of a lateral temporal sulcus. No significant atrophy was present in these 2 cases.

Microscopically, irregular, dilated vascular channels were present, and

Fig. 9. Case 1. Isolated area of bone formation. Hematoxylin and eosin, ×65.

Fig. 10. Case 5. Calcospherites. Hematoxylin and eosin, ×90.
again veins were more numerous. These channels consisted of vessels from
the meninges and extended into the superficial cortical layers. The tissue
between vessels was fibrous connective tissue. Venous thrombosis with
partial or complete recanalization was present but less frequently than in
the intracerebral variety.

Deposition of calcium was rarely present and in no instance was there
evidence of osteogenesis, calcospherites or encephalomalacia. Deposition
of hemosiderin was a prominent feature. The parenchyma of the brain
directly adjacent to the malformation was intensely gliotic and the neurons
therein exhibited ischemic changes.

The follow-up analysis, shown in Table 4, is not long enough to determine
the final results. The longest follow-up is 49 months and the shortest was 2
months. To date 5 patients are seizure-free and the remainder have fewer
attacks.

DISCUSSION

Various gross and/or microscopic abnormalities have been associated
with temporal lobe epilepsy. Severe mesial or “incisural” sclerosis secondary
to herniation of the mesial temporal structures through the incisura tentorii
during partuition, and various malformations of the temporal lobe such as
nodular cortical atrophy, porencephaly, microgyri, Ammon’s sclerosis and
Fig. 12. Case 4. Localized area of encephalomalacia in cerebral tissue around malformation. Hematoxylin and eosin, $\times 220$.

Fig. 13. Case 10. Medium-sized plexiform meningoocortical malformation with ramifications over the frontal and temporal lobes.
### TABLE 4

**Follow-up analysis**

<table>
<thead>
<tr>
<th>Case No.</th>
<th>Months Postop.</th>
<th>Deaths</th>
<th>Electrographic Status</th>
<th>Seizure Status</th>
<th>Neurologic Status</th>
<th>Personality Status</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>10</td>
<td>0</td>
<td>Less active, but became bitemporal</td>
<td>Marked decrease</td>
<td>Normal</td>
<td>Marked visual and somatic perceptual illusions</td>
</tr>
<tr>
<td>2</td>
<td>10</td>
<td>0</td>
<td>Less active but persisting r. temporal focus</td>
<td>Seizure free</td>
<td>Normal</td>
<td>Unchanged</td>
</tr>
<tr>
<td>3</td>
<td>49</td>
<td>0</td>
<td>Less active, but became bitemporal, maximal l. temporal</td>
<td>Definite decrease in frequency and severity</td>
<td>Normal</td>
<td>Improved</td>
</tr>
<tr>
<td>4</td>
<td>6</td>
<td>0</td>
<td>Decreased but persisting l. temporal focus</td>
<td>Seizure free</td>
<td>Normal</td>
<td>Unchanged</td>
</tr>
<tr>
<td>5</td>
<td>0</td>
<td>Died immediately from brainstem infarction and intracranial hemorrhage</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>6</td>
<td>0</td>
<td>Died in immediate postop. period from generalized bleeding. Afibrinogenemia</td>
<td>—</td>
<td>—</td>
<td>—</td>
<td>—</td>
</tr>
<tr>
<td>7</td>
<td>44</td>
<td>0</td>
<td>Decreased but persisting r. temporal focus</td>
<td>Seizure free</td>
<td>Normal</td>
<td>Unchanged</td>
</tr>
<tr>
<td>8</td>
<td>4</td>
<td>0</td>
<td>Decreased but persisting bitemporal foci</td>
<td>Seizure free</td>
<td>Normal</td>
<td>Improved</td>
</tr>
<tr>
<td>9</td>
<td>12</td>
<td>0</td>
<td>No longer bitemporal. Persisting l. temporal focus</td>
<td>Decreased frequency</td>
<td>Unchanged (l. hemiparesis persists)</td>
<td>Unchanged</td>
</tr>
<tr>
<td>10</td>
<td>2</td>
<td>0</td>
<td>Decreased but persisting r. temporal focus</td>
<td>Seizure free</td>
<td>Normal except for old chorioretinitis</td>
<td>Unchanged</td>
</tr>
</tbody>
</table>

Amygdalo-uncinate sclerosis subsequent to neonatal, paranatal, or postnatal anoxia or vascular insufficiency have been designated as epileptogenic. Meningocortical cicatrix resulting from open craniocerebral trauma or brain abscess and severe gliosis secondary to contusion of the anterior
temporal lobe incurred with closed head injuries account for a high percentage of cases of psychomotor epilepsy.\textsuperscript{6} Large arteriovenous malformations, neoplasms, tuberculomas, and viral or bacterial infections such as nonspecific viral encephalitides, measles encephalitis, syphilitic meningovascular disease and pyogenic meningoencephalitis account for a lesser number of cases of temporal lobe epilepsy.\textsuperscript{5,6,10}

Despite this impressive array of abnormalities, no conclusive statements can be made in regard to their relation to epilepsy. Several series have shown that the presence of identical lesions in various areas of the temporal lobe was not accompanied by psychomotor epilepsy.\textsuperscript{12,16} Therefore, factors other than these gross and/or microscopic lesions are necessary to produce epilepsy. At the present time, however, the documentation of all temporal lobe lesions associated with psychomotor epilepsy is important in order to correlate them with future data.

No signs or symptoms suggestive of focal mass lesion were present in the history, seizure pattern, or neurologic examination. The roentgenograms of the skull, cerebrospinal fluid examination and contrast studies usually did not reveal signs of increased intracranial pressure or of a focal mass lesion. Conversely, in several instances, a general dilatation of the temporal horn on the side of the lesion resulted in a clinical impression of a localized atrophic process rather than a mass lesion. Likewise, in the examples of small focal neoplasms of the temporal lobe reported by other investigators, preoperative suspicion of a mass lesion was infrequent and, in some cases, local dilatation of the temporal horn suggested an atrophic, sclerotic process rather than a tumor.\textsuperscript{4} The seizure pattern and electrographic abnormalities served to localize and lateralize the epileptogenic process preoperatively. These cases, therefore, cannot be differentiated from any other case of psychomotor epilepsy in which no definite etiologic factors have been established.

There are certain similarities between the intracerebral malformation and hemangioma calcificans. This latter entity was characterized by calcification visible in roentgenograms of the skull, anatomic location in the right temporal lobe in 5 out of 6 cases, and temporal lobe epilepsy.\textsuperscript{1,17}

In the present series, calcification was never visible radiologically but was usually present microscopically. Likewise, all patients experienced temporal lobe epilepsy and 5 out of 6 lesions were located in the right temporal lobe.

The possibility that these lesions are the same is suggested by calcification, tendency to sclerosis and intracerebral location. The degree of calcification may represent either metabolic variations or differences in duration.\textsuperscript{1,17}

The meningeocortical type of lesion may be compared, both grossly and microscopically, to the cryptic hamartoma.\textsuperscript{8,14} Clinically, however, certain differences exist that are quite important.

In a total series of 20 patients shown to have a cryptic hamartoma, only
2 experienced cerebral seizures. The cryptic hamartoma usually presents in a young adult with sudden, often fatal, spontaneous intracranial hemorrhage. These two features, infrequent seizures and tendency to hemorrhage, are juxtaposed to the clinical course of the 4 meningocortical cases presented in this paper.

The investigative approach may be primarily responsible for the variation. In the series of cryptic hamartoma, autopsy material was examined in an effort to explain nontraumatic spontaneous intracerebral hemorrhage. The cases presented in this paper resulted from examination of surgical specimens. These lesions should not be considered neoplastic but rather as congenital vascular malformations. No instance of mitosis, pleomorphism, invasion or other signs of malignancy was present. They were, however, usually mass lesions and could be considered progressive in regard to destruction of surrounding cerebral tissue. The cycle of thrombosis, hemorrhage, calcification and sclerosis perpetrates a continuing destruction of the immediately adjacent, previously normal cerebral tissue. This continuing process is not only recognizable through the microscope but is illustrated radiologically in Case P.L., in whom a progressive increasing density of calcium deposited was followed for 5 years by roentgenograms of the skull. This process most likely explains the accompanying atrophic changes seen grossly and by contrast studies in certain cases in this series.

Early surgical removal, as with other intracranial mass lesions, is preferable. The alleviation of seizures in certain cases and the termination of further damage to the brain justifies a surgical approach for this particular malady.

SUMMARY

Ten congenital cerebral arteriovenous malformations of the temporal lobe representing an 11.4 per cent incidence in a series of 88 temporal lobectomies were found coincident with psychomotor epilepsy. They were divided into intracerebral and meningocortical types. The former was compared to hemangioma calcificans and the latter to cryptic hamartomas. Despite adequate investigation, none of these lesions was diagnosed prior to surgical exposure. Their relationship to epilepsy remains unsettled.

The authors wish to express their gratitude to Dr. Jan Cammermeyer, Dr. Kristof Abraham and Mr. Fred Meiller for their assistance in the neuropathologic, electrographic and photographic aspects of this paper.

REFERENCES

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VASCULAR LESIONS AND TEMPORAL LOBE EPILEPSY


DISCUSSION

Dr. J. Peter Murphy: I would like to congratulate Dr. Edgar on his interesting and scientific paper and ask him three brief questions. The first is: In proceeding with surgery, does he incise the temporal lobe before undertaking standard removal, in order to determine whether some of the vascular lesions he described are present or not?

Second: If he finds this type of condition, does he proceed to remove the rest of the temporal lobe in standard fashion anyway?

Thirdly: Does he feel that the electrographic pattern when this specific lesion is causing psychomotor epilepsy is more discrete in terms of spiking and more obvious than it is in the cases of psychomotor epilepsy, in which after removal of tissue not much, if anything, is found on microscopic examination?

Dr. Theodore Rasmussen: These are an interesting group of patients and are a good example of the grab bag of lesions that one finds in exploring patients with focal epilepsy.

I would like to ask Dr. Edgar two questions. One, I wonder if he would elaborate a bit on their concept of the etiology of these lesions and the reason for the surprisingly widespread atrophic and gliotic changes that are so often present in association with these small focal lesions. It seems to me if these changes are secondary to vascular events in the vicinity of this focal lesion that the atrophic area should be concentrated around them. We have seen these atrophic changes throughout the entire temporal lobe at some distance from the small
angioma. The gross and microscopic appearance of these atrophic areas is not too dissimilar to the cases that we ascribe to birth compression and incisural sclerosis.

Second, I would like to ask whether or not the authors have turned up, in addition to these lesions that were grossly visible, similar lesions that were evident only on microscopic examination. There were several in which we saw nothing in the gross removal of the specimen, and even when the specimen was cut we saw nothing, but on microscopic examination of one of the blocks, lesions were seen quite similar to these. Since marked atrophic changes and gliosis of the temporal lobe were present in addition, one must consider the possibility that these changes rather than the tiny angioma itself were responsible for the seizures.

Dr. Robert Edgar: Dr. Murphy, the subpial suction dissection technique was utilized in each case. Dissection was ordinarily started in the second temporal convolution near the vein of Labbé. Masses such as these vascular malformations were readily identified as discolored, hard areas in the substance of the brain. In one instance a surface abnormality consisting of localized cortical atrophy and brownish discoloration overlay the vascular mass.

The extent of temporal lobe resected was usually dependent upon persisting electrographic abnormalities rather than on the type of lesion present. In most instances the anterior temporal lobe, including the mesial structures, was resected 4.5–5.0 cm. posterior to the temporal tip. In left-sided cases resection was rarely performed beyond the vein of Labbé.

In the present series the electrographic abnormalities were indistinguishable from those in other nonvascular cases of temporal lobe epilepsy. An analysis of the electrocorticograms in this group revealed only one instance of relatively focal spike and wave discharge.

Dr. Rasmussen, we have observed areas of focal encephalomalacia in the temporal lobe several centimeters from the primary vascular lesion. The tendency of these vascular masses to produce sclerosis and thrombosis of the vessels is responsible for these widely disseminated microscopic changes. Usually these changes are most severe in the parenchyma of the brain which is directly applied to the vascular lesion. As a matter of fact most of these masses were surrounded by a pseudcapsule which consisted of dense, calcified, gliotic brain. The gross expression of these microscopic lesions was a general atrophy of the entire temporal lobe.

We have observed microscopic vascular malformations. Two of the meningocortical vascular malformations were quite small and were detected only when the surgical specimen was sectioned in the pathological laboratory. In addition there were several cases of microscopic vascular lesions which were considered to be acquired rather than congenital. These included one case of unexplained periarteritis and several cases of severe arteriosclerosis of the superficial cerebral vessels. All were in individuals who were 40 years of age or younger.

We feel that these vascular malformations are congenital in origin. It would be difficult to account for such a localized abnormality of this histologic type on the basis of an atrophic scar or sclerotic process secondary to abscess, trauma or hemorrhage.