Temporal-lobe Changes Associated with the Syndromes of Basilar-vertebral Insufficiency: An Electroencephalographic Study

B. M. PHILLIPS,* M.B., M.R.C.P.

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The syndrome of basilar-vertebral insufficiency has come to be recognized over recent years as a common cause of recurrent transient disturbances of brain-stem function. Kubik and Adams (1946) suggested that variation in the neurological state in patients with basilar occlusion could be explained on a haemodynamic basis. Denny-Brown (1953) and Millikan and Siekert (1955) emphasized the syndrome of basilar-vertebral insufficiency and described cases confirmed at necropsy.

Diagnostic aids are on the whole unsatisfactory, for the demonstration of stenosis or occlusion of the basilar artery by vertebral angiography is a procedure not without risk in arteriosclerotic patients (Hauge, 1954). Deterioration after vertebral angiography is not uncommon and has been attributed to vasospasm in the basilar-vertebral territory or, alternatively, to small particles of thrombus being dislodged from the surface of atheromatous plaques. Neurological deterioration following the retention of contrast medium in the small vessels of the brain, such as described by Northfield and Russell (1937), where Thorotrast was the medium employed, is less likely to occur with the improved contrast media now being used. Retrograde subclavian angiography obviates the necessity for puncturing the vertebral artery, and with this technique the origin and course of the right vertebral artery is demonstrated. Unfortunately, adequate filling of the basilar artery along its whole length is difficult to achieve using the subclavian route, and, furthermore, filling of the upper basilar via the posterior communicating and other collateral vessels makes the interpretation of the angiogram difficult (see Case 2). Bauer, Sheehan, and Meyer (1961) studied 71 patients with cerebrovascular disease, using percutaneous carotid angiography and either subclavian angiography or brachial catheterization without serious complication. It was the paucity of simple diagnostic aids that prompted the present investigation into the electroencephalographic changes associated with brain-stem vascular syndromes.

A number of reports have appeared in the literature describing a variety of changes in the electroencephalogram associated with vascular disease of the brain stem. Watson and Adams (1951) described the case of a patient with upper basilar thrombosis whose fluctuating level of consciousness was accompanied by the development of slow-wave abnormalities, subsequently reverting to normal. Roger, Roger, and Gastaut (1953) concluded from their studies on 36 patients with brain-stem vascular disease that the electroencephalographic changes could be subdivided into (*a*) modification of background activity, which included fast α-rhythms, β-activity, and spikes; and (*b*) slow waves, either unilateral or bilaterally situated. Markovich (1958) found no significant abnormality among the 12 cases he studied. Tucker (1958) reviewed four cases in detail and three of them showed clear-cut temporal-lobe abnormalities, while the fourth showed a more diffuse hemisphere disturbance with bilateral slowing in the range 3–5 c/s. Meyer, Leiderman, and Denny-Brown (1956) found bi-occipital slowing in 4 out of 13 cases, although after the use of a tilt-table to lower the systemic blood-pressure the abnormal records totalled nine with foci in the occipital and posterior temporal regions. Bauer, Wechsler, and Meyer (1961) studied the effect on the electroencephalogram of carotid compression, with and without rotation of the head, in a group of patients with vertebral-artery disease proved by angiography. In 10 cases the resting records were normal, while the remaining eight showed diffuse theta waves as the principal abnormality. With compression and rotation of the head seven cases showed altered records, the induced changes being predominantly temporal and parietal. The occurrence of seizures and the intensification of signs and symptoms with this procedure make it unsuitable as a method of revealing electroencephalographic abnormalities in patients with brain-stem vascular disease.

* Registrar to the Department of Neurology, St. George's Hospital, London. Present address: National Hospital for Nervous Diseases, Queen Square, London.

Method and Results

The patients in the present series were all admitted to Atkinson Morley's Hospital for investigation in either the neurological or the neurosurgical department. In each case a clinical diagnosis of basilar-vertebral insufficiency was made on the history of recurrent episodes of brain-stem disturbance culminating in a number of instances in basilar thrombosis. The criteria for diagnosis used in these patients have been outlined by Williams (1961), and a more detailed review is made by Williams and Wilson (1962); it is therefore not necessary to enlarge on the clinical features.

The electroencephalograms used in the present study were routine recordings made on an 8-channel Ediswan apparatus. Hyperventilation was the only activation procedure employed unless otherwise indicated. Recordings from 40 patients were reviewed and 19 were judged to be within normal limits; many of the patients had repeated records. In 13 patients the principal abnormality was slow-wave activity (2–6 c/s) over the temporal lobes. Bitemporal slowing was seen in nine cases, of which five were asymmetrical. This asymmetry has been noted by previous authors, and Meyer *et al.* (1956) suggested that this may be related to the frequent asymmetry observed in the posterior communicating arteries, which play an important

Electroencephalographic Findings

Case	Age	Electroencephalographic Abnormalities
1	73	4-5 c/s slowing over anterior and middle temporal regions. Diffuse β-activity
2	45	Paroxysmal bitemporal slowing 2–3 c/s
3	43	Bitemporal slowing. Anterior L > R 3–5 c/s
4	53	Bitemporal slowing, R > L 2–3 c/s
5	38	Bitemporal slowing 6–7 c/s
6	62	Bitemporal slowing R > L 6–7 c/s
7	65	Bitemporal slowing, L > R 3–6 c/s
8	60	Bitemporal slowing, R > L 2–3 c/s
9	71	Mild bitemporal slowing 5 c/s
10	55	Left temporal focus 2–3 c/s
11	60	Left temporal slowing 4 c/s
12	48	Right anterior temporal slowing
13	70	Low-voltage 7 c/s in left temporal region
14	72	Diffuse dysrhythmia with low voltage on left
15	64	Generalized β-activity with 6 c/s waves over left hemisphere
16	50	Diffuse slowing over left hemisphere.
17	61	Bilateral 5–6 c/s waves over both hemispheres most marked in the anterior temporal regions. Paroxysmal frontal slow waves 2–3 c/s
18	61	Bilateral 6 c/s over parietal temporal regions
19	71	Diffuse 2–6 c/s over both hemispheres
20	68	Bifrontal and anterior temporal slow waves 3–5 c/s
21	66	Generalized β-activity with bilateral 5 c/s R > L

part in the maintenance of adequate blood-flow to the upper brain stem in the presence of impaired basilar flow. In four cases the temporal-lobe disturbance was strictly unilateral. The remaining abnormal records in the main showed more diffuse abnormalities over both hemispheres, with no particular preponderance over the temporal leads. In three cases β-activity was prominent throughout the recordings. Details of the electroencephalographic findings are given in the Table.

Case 7

This 65-year-old man, a known hypertensive, began to complain of transient attacks of unsteadiness, slurred speech, and tingling of the lips five weeks prior to admission to hospital. Each episode lasted for five minutes, and between the attacks he was asymptomatic. Four weeks after the onset he began to notice weakness of his right arm and, in addition, bulbar disturbances became evident, inasmuch as he had increasing difficulty with swallowing and articulation.

On examination at the time of admission he was dysarthric, with a partial left trigeminal, right lower facial weakness, nystagmus on lateral gaze, and bilateral palatal weakness. There were bilateral cerebellar and pyramidal signs in the limbs. Radiographs of the skull and chest were normal, as were the findings on lumbar puncture. The electroencephalogram showed well-marked bitemporal slowing in the range 3–6 c/s.

A clinical diagnosis of basilar insufficiency was made, and, as the signs were progressing, anticoagulants were begun. The patient died suddenly seven days after admission in spite of an initial clinical improvement.

Necropsy findings confirmed the clinical diagnosis, for the basilar artery was seen to be completely occluded by recent ante-mortem thrombus, and on sectioning the basilar and vertebral arteries severe atheromatous changes were seen. Patchy areas of ischaemic necrosis were present in the pons, tegmentum, and middle cerebellar peduncles.

This case was quoted in greater detail by Williams and Wilson (1962) in their paper on basilar insufficiency.

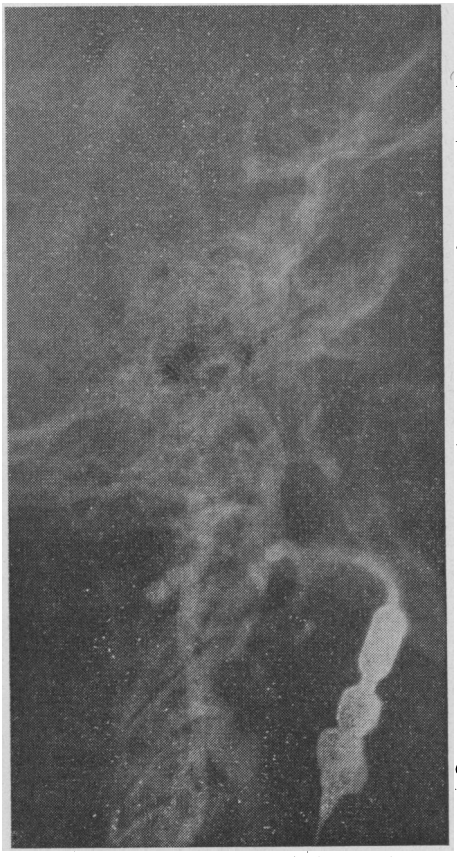

FIG. 1.—Case 2. Right subclavian angiogram showing the distal segment of the basilar artery filling via the posterior communicating artery.

Case 2

A 45-year-old woman had been known to have hypertension from the age of 25, following her second pregnancy. The presentation in this case consisted of recurrent episodes of total blindness lasting from two to three minutes. One week after the onset a transient loss of consciousness was followed by complete visual loss of 30 minutes' duration. On examination soon after this episode no abnormal signs were found and she was then asymptomatic for a further 10 days, when she awoke to find that her speech was slurred and her gait unsteady.

On admission to hospital three weeks after the onset she showed marked emotional lability. Speech was slurred and bilateral facial weakness, left trigeminal paresis, nystagmus, and poverty of tongue

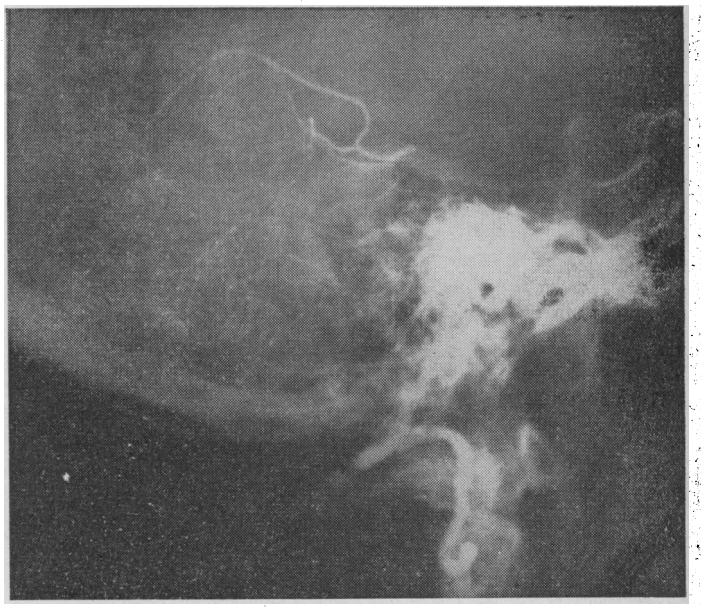

FIG. 2.—Case 2. Lateral projection of left vertebral angiogram showing complete basilar occlusion. The posterior inferior cerebellar artery can be seen filling.

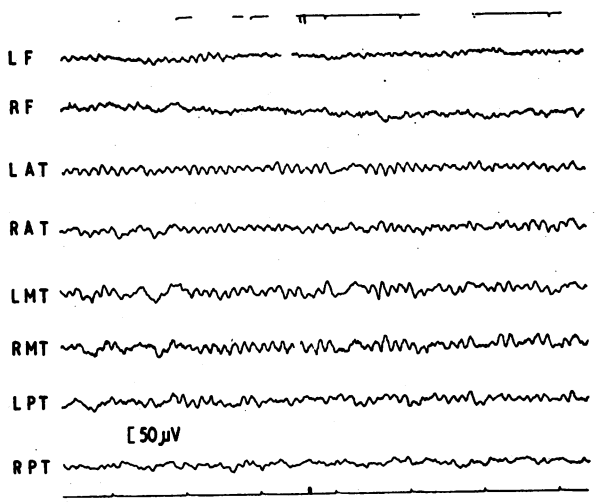

FIG. 3.—Case 2. Electroencephalogram soon after admission, showing temporal slowing which occurred paroxysmally throughout the recording.

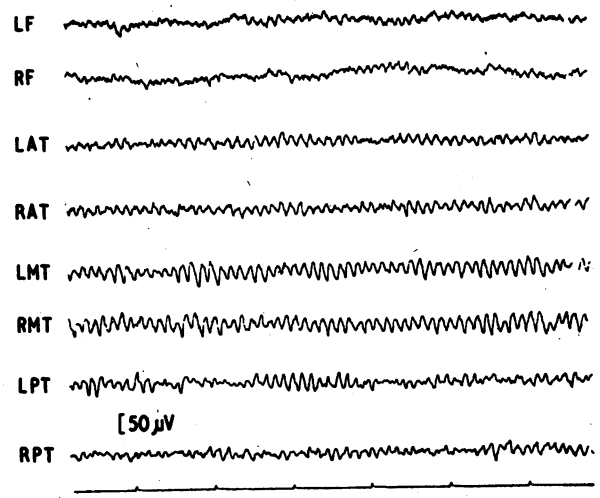

FIG. 4.—Case 2. Electroencephalogram two months after the onset.

movements were present. Examination of the limbs revealed bilateral ataxia and pyramidal signs, more in evidence on the right.

Radiographs of the skull and chest were normal, as was the cerebrospinal fluid. A right subclavian angiogram showed that the right carotid and vertebral arteries were normal, but filling of the proximal third of the basilar artery was not obtained. The distal segment of the basilar artery could be seen to be filling via the posterior communicating arteries (Fig. 1). A left vertebral arteriogram carried out later confirmed complete proximal basilar occlusion (Fig. 2). An electroencephalogram recorded soon after admission showed paroxysmal slowing 2–3 c/s in both temporal regions (Fig. 3). Over the course of the next two months dramatic improvement in the neurological signs occurred, although emotional lability was still in evidence. The electroencephalogram returned to normal within days. Fig. 4 shows a recording made two months after the onset.

Case 17

A hypertensive man aged 61 suddenly developed slurring of speech followed by weakness of the left limbs while working in his garden one year prior to admission. This episode lasted approximately four minutes and was not accompanied by sensory disturbance or headache. After this incident he remained well for 11 months, when on reaching the top of a long flight of stairs he developed an acute onset of pain in the chest which lasted for an hour. Later that day he had a second episode of pain, this time lasting for 15 minutes; on this occasion it was accompanied by slurring of speech and weakness of his left limbs, which cleared up in a few minutes. Two weeks later he experienced palpitations which lasted for 15 minutes; at this time he was described as being confused, but no other details of his neurological state are available. During the next two weeks he had two further episodes of tachycardia, each accompanied by a deterioration in his mental state and slurring of speech.

On examination at the time of admission he was mildly confused and dysarthric. There was bilateral nystagmus and lower left facial weakness, but the right trigeminal weakness and absent corneal

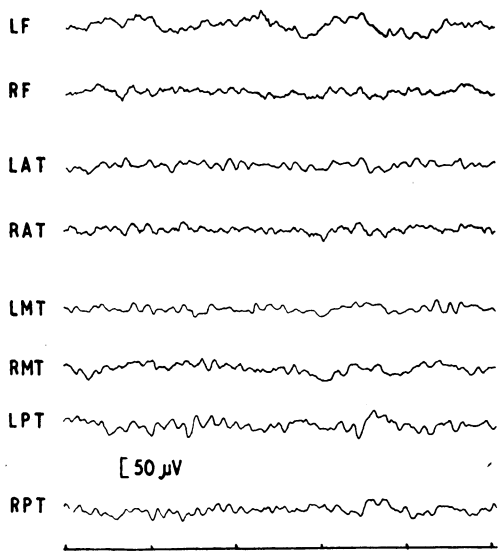

FIG. 5.—Case 17. Electroencephalogram showing slow-wave changes over the temporal lobes.

response previously noted had returned to normal. Pyramidal signs were apparent in all four limbs, the weakness being more in evidence on the left. Co-ordination was impaired bilaterally but there were no sensory signs.

Skull and chest *x*-ray films were normal. The E.C.G. confirmed the presence of a recent myocardial infarct. The W.R. and Kahn test were negative and the findings on lumbar puncture were within normal limits. The electroencephalogram (Fig. 5) showed bilateral slow-wave activity in the range 5–6 c/s, more marked over the temporal leads. The records of this patient also showed paroxysmal frontal slowing 2–3 c/s. No angiograms were carried out and he was transferred to another hospital; unfortunately no follow-up was possible in this case.

Case 22

This case is of particular interest in that it demonstrates how little the electroencephalogram may be affected by pontine lesions and that where changes are observed it is reasonable to suppose they are brought about, in many cases, by clinically less obvious lesions at a higher level.

A normotensive woman aged 56 had a year's history of symptoms suggesting a mild degree of basilar-vertebral insufficiency. On examination there were no abnormal physical signs apart from a systolic bruit over both carotids.

X-ray films of the skull and chest were normal and the findings on lumbar puncture non-contributory. Stenosis of the left internal carotid artery was revealed by a left carotid arteriogram, but a

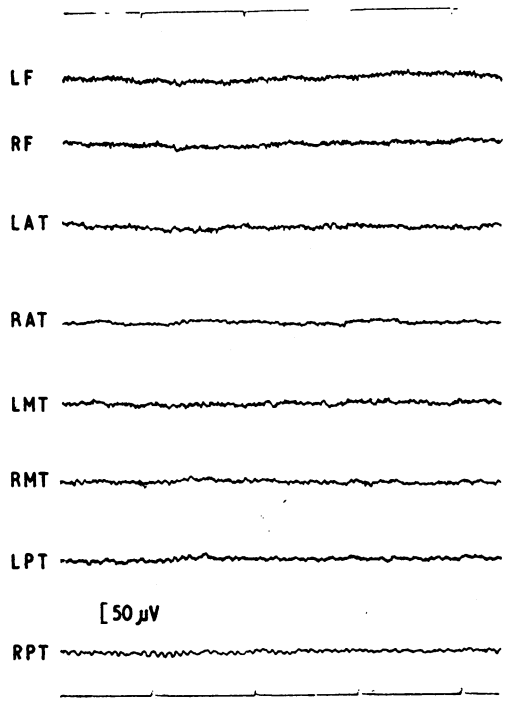

FIG. 6.—Case 22. Normal electroencephalogram two weeks after pontine infarction.

right subclavian arteriogram was entirely normal. Operation was deferred on the grounds that her symptomatology could not be attributed to the left carotid stenosis and that there were no abnormal neurological signs. Ten days after discharge from hospital she experienced an acute onset of dizziness, following which she became progressively more drowsy. On examination eight hours after the onset she was unconscious but responded to painful stimuli. The pupils were unequal, the left being constricted and poorly reactive to light. Initially there was some improvement although she remained anarthric, with severe limb weakness. Pneumonia eventually led to death two months after admission.

Electroencephalograms recorded before and after the terminal illness were normal (Fig. 6). At necropsy old cavitated softenings were demonstrated in the basi-pontis, producing extensive destruction (Fig. 7), attributed to atherosclerotic lesions in the vertebral arteries. The basilar artery showed only minimal atherosclerotic changes.

Discussion

From this and previous studies there can be no doubt that brain-stem ischaemic syndromes are accompanied by electroencephalographic changes in a significant number of cases. Over 25% of the present series showed temporal-lobe abnormalities, which is to be expected from the territory supplied by the basilar-vertebral system. The sites of lesions or ischaemic areas responsible for the production of abnormal rhythms are for the most part above the level of the pons. Lindsley,

Bowden, and Magoun (1949) found in experimental animals that lesions made in the medulla and pons were without marked effect on the electroencephalogram, whereas with mesencephalic or diencephalic lesions the low-voltage fast activity was replaced by abnormal slower rhythms. The changes produced by brain-stem lesions are attributed to damage to the reticular activating system. Thalamic lesions may contribute to the electroencephalographic abnormality, since the posterior cerebral arteries supply the posterior part of the thalamus. A further possibility is that electroencephalographic abnormalities may arise in the temporal lobes as a direct result of reduced blood-flow through the posterior cerebral arteries, which supply most of the inferior and lateral aspects of the temporal lobes. Typical temporal-lobe attacks have been described in association with basilar-vertebral insufficiency but were not encountered in the present series.

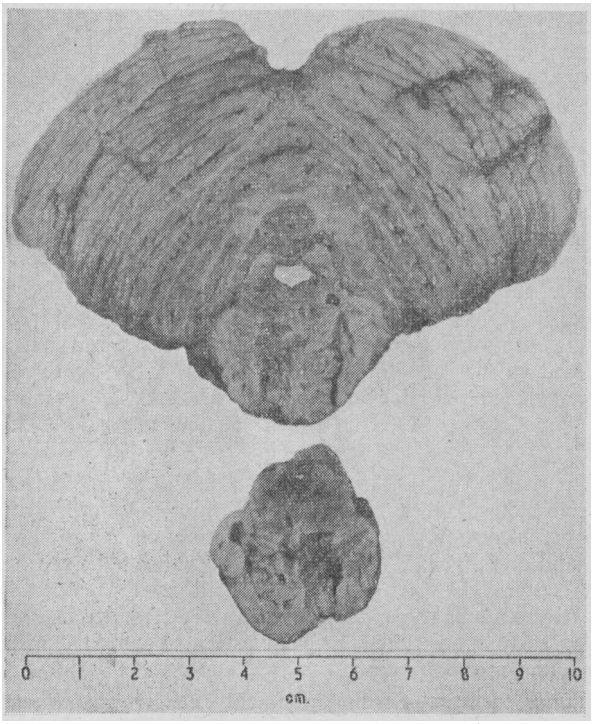

FIG. 7.—Case 22. Necropsy specimen showing pontine cavitation.

As a diagnostic aid the electroencephalogram is limited, mainly because many of the changes are non-specific, and, furthermore, a high percentage of the records are normal—50% in the present series. The fact, however, that the electroencephalogram is normal may to some extent signify that the diencephalic and mesencephalic structures have not suffered severe damage from ischaemia. It is in this type of case that further investigation into the state of the extracerebral vessels may be warranted, particularly in view of the high incidence of stenotic lesions in the proximal parts of the vertebral arteries (Schwartz and Mitchell, 1961; Hutchinson and Yates, 1957). Occasionally difficulty arises in the differentiation of vascular syndromes from space-occupying lesions in the posterior fossa; in this type of case the electroencephalogram is not of much help. Where basilar-vertebral insufficiency is suspected the danger of hypotension has to be borne in mind, particularly when air-encephalography is contemplated. The importance of hypotension is stressed, for in nine of the cases studied in the present series symptoms dated from the onset of myocardial insufficiency—for example, Case 17.

Summary

The electroencephalographic changes associated with the major and minor syndromes of basilar-vertebral insufficiency are reviewed in 40 cases. Nineteen of the recordings were assessed as being within normal limits. Among the abnormalities, temporal-lobe disturbances were the commonest finding—13 out of a total of 21 abnormal records. The importance of recognizing the occurrence of focal disturbances in the electroencephalogram in association with the syndrome of basilar-vertebral insufficiency is stressed, for angiography and air-encephalography are poorly tolerated in these patients. The high incidence of normal records and the non-specific nature of many of the abnormalities limit the value of the electroencephalogram, but nevertheless in some cases it provides useful additional information.

I should like to thank Dr. Denis Williams for his advice and encouragement, and I am also grateful to Mr. Wylie McKissock and the late Dr. J. Hamilton-Paterson for permission to study cases under their care. Electroencephalographic recordings were carried out by Miss M. Davitt.

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