Arch Psychiatr Nervenkr (1982) 231 : 547–554

Archiv für Psychiatrie und Nervenkrankheiten
Archives of Psychiatry and Neurological Sciences

Temporal Slow Activity of the EEG in Old Age

A. Kazis, A. Karlovasitou, D. Xafenias

Aristotelian University of Thessaloniki, Department of Neurology, AHEPA Hospital, Thessaloniki, Greece

Summary. The aim of this work and the question which it will attempt to answer is to what extent temporal slow activity comprises a purely functional finding in old age and to which, if any, organic cerebral lesions it is related.

For this reason 2,035 in-patients, 60–92 years old, were divided into four groups on the basis of clinical and laboratory criteria: a) asymptomatic patients without neurological or C. T. findings (502); b) asymptomatic patients with neurological and/or C. T. findings (103); c) patients with clinical semeiology due to cerebrovascular accidents (1,230); and d) patients with clinical semeiology due to organic cerebral lesions other than vascular (200).

Recording frequency of temporal slow activity was evaluated in the total number of patients 26.8%, while in each group separately the values were 19.5%, 26.2%, 30.2%, and 24% respectively. The more frequent appearance of temporal slow activity on the left side (65.7%), its more frequent recording in patients with transient ischemic attacks (25.7%) in comparison with the remaining patients of the same groups without similar accidents (18.3%), its more frequent bilateral recording (60.8%), its lower correlation with the locations of the organic cerebral lesions (34.2%) and the more frequent appearance of generalized angiopathy in patients with temporal slow activity (79.4%) in comparison with the remaining patients without temporal slow activity (55%) support the view of the dependence of this electrographic element on the circulatory disturbances of the brain, which are frequently subclinical.

This view is further supported by the results of the study of 26 asymptomatic patients of the first group with temporal slow activity, of whom 34% suffered vascular accidents over a period of 10 years.

Key words: Temporal slow activity – Old age – Circulatory disturbances

Introduction

The slow rhythms which are recorded episodically in the temporal regions of aged individuals, unilaterally or bilaterally, are much debated: are they normal or abnormal phenomena, and with what conditions or diseases are they associated?

First presented at the South-East European Conference on Neurology and Psychiatry, 30 September–3 October 1981, Thessaloniki.
Offprint requests to: A. Kazis at the above address

0003-9373/82/0231/0547/$01.60

Obrist (1954), Obrist et al. (1962 and 1963) and Kooi et al. (1964), following a study of a great number of asymptomatic aged people, considered temporal slow activity to be a normal phenomenon. On the other hand, Gastaut et al. (1959) and later Gastaut and Poirier (1960), Bruens et al. (1960), Van der Drift (1961) and Kendel and Koufen (1970) considered temporal slow activity as an element indicating circulatory disturbances in the brain.

The purpose of the present work is to attempt to clarify the above difference in opinion by evaluating this electrographic element in aged and clinically asymptomatic patients, as well as in patients with a clinical semeiology.

Material and Methods

From approximately 20,000 EEG recordings of in-patients, 2,035 were chosen belonging to patients between the ages of 60 and 92 years (average age 72.5), of which 1,001 were men and 1,034 women. The patients were divided into two categories on the basis of their clinical semeiology at the time of their EEG recordings: 605 asymtomatic patients (298 men and 307 women, average age 70.8 years) who came to the hospital with subjective disturbances (dizziness, headache, vertigo, easy mental fatigue) without any neurological or psychiatric clinical semeiology (normal psychometric tests); and 1,430 patients (703 men and 727 women, average

Table 1

	Asymptomatic patients (605)		Patients with clinical semeiology (1430)		Total
	1st group (502) without neurological or C. T. findings	2nd group (103) with neurological and/or C. T. findings	3rd group (1230) vascular cerebral episode	4th group (200) other organic cerebral lesion (except vascular)	
Temporal slow activity (TSA)	98 (19.5%)	27 (26.2%)	372 (30.2%)	48 (24%)	26.8%
Unilateral (TSA)	40 (40.8%)	12 (44.5%)	147 (39.5%)	18 (37.5%)	39.2%
Bilateral (TSA)	58 (59.2%)	15 (55.5%)	225 (60.5%)	30 (62.5%)	60.8%
Prevalence of the TSA on the left side	70 (71.4%)	17 (63%)	238 (64.1%)	33 (64.6%)	65.7%
TSA ipsilateral to the lesion	–	8 (29.6%)	130 (34.9%)	15 (31.75%)	34.2%
TSA and generalized angiopathy	60 (61.2%)	18 (66.7%)	325 (87.4%)	30 (62.5%)	79.4%
Generalized angiopathy without TSA	142 (35.1%)	30 (39.5%)	582 (67.8%)	66 43.4%)	55%
TSA and transient ischemic attack	24.6%	30.8%	–	–	25.7%
TSA without transient ischemic attack	18.9%	15.5%	–	–	18.3%

age 73.4 years) with neurological semeiology (hemiparesis, hemianopsia, aphasia, paralysis of cranial nerves, etc.) or psychiatric semeiology (diminution of the higher mental functions, memory disturbance, etc.).

The patients in each category were divided into two groups on the basis of observations of their clinical progress and the neurological examination and X-rays, mainly computed tomography (C. T.) but also brainscan, and in special cases angiography. The asymptomatic patients of the first category were divided into those whose C. T. scans did not give any abnormal findings for their age (1st group, 502 patients) and those whose C. T. scans revealed abnormal elements, usually small ischemic infarctions (2nd group, 103 patients).

The 1,430 patients of the second category were divided into those whose clinical semeiology was due to a cerebral vascular accident, i.e. thrombosis, embolism or hemorrhage (3rd group, 1,230 patients), and those whose clinical semeiology was due to various organic cerebral lesions, other than vascular, i.e. tumors, degenerating diseases, inflammatory diseases of the brain, etc. (4th group, 200 patients).

These patients' EEG's were examined for signs of temporal slow activity. Recording frequency, as well as certain other parameters of temporal slow activity, was studied in the patients, both in total and separately in each group, so that comparisons could be more easily made.

More specifically we evaluated the percentage of the unilateral or bilateral (symmetrical or asymmetrical) appearance of temporal slow activity, its predominance in the left or right hemisphere and the coincidence of the EEG findings with the side where the organic lesion existed (for the last 3 groups of patients). In addition we studied the relation between temporal slow activity and general angiopathy (obvious arteriosclerosis of the fundal vessels, hypertension, coronary insufficiency, peripheral artery occlusion, other evidence of systemic vascular disease, etc.) both in the patients as a total and separately in each group.

The relation between temporal slow activity and transient ischemic attacks was studied specifically in 57 patients of the first group and 13 patients of the second group who had a similar past medical history. A limited number of patients (26) in the first group with temporal slow activity have been under clinical and laboratory observation for periods of 5 to 10 years.

Results

Table 1 shows the main results.

Group 1. Asymptomatic patients without neurological or X-ray findings (total: 502; men: 247; women: 255; average age: 70.6 years), Table 1.

a) Of the 502 patients 98 (19.5%) showed temporal slow activity, 40 (40.8%) unilaterally and 58 (59.2%) bilaterally (49 asymmetrically and 9 symmetrically, Fig. 1). In 28 of the 40 patients and 42 of the 58, i.e. a total of 71.4%, temporal slow activities in the left hemisphere only or with the left side predominating were seen.

b) Of the 502 patients 57 had a medical history which could be definitely attributed to a transient ischemic attack. Of these 57 patients 14 (24.6%) showed temporal slow activity, 9 unilaterally (64.3%) and 5 bilaterally (35.7%). In 10 of the 14 patients, angiography showed stenosis of the carotid, 8 of them ipsilateral to the temporal slow activity. Of the remaining 445 patients with no medical history of a transient ischemic attack 84 (18.9%) showed temporal slow activity, 39 unilaterally (46.4%) and 45 bilaterally (53.6%).

c) Of the 502 patients, 202 (40.2%) showed elements of a generalized angiopathy. In 60 of the 98 patients with temporal slow activity (61.2%), and 142 of the remaining 404 patients without temporal slow activity (35.1%), elements of a generalized angiopathy were evident.

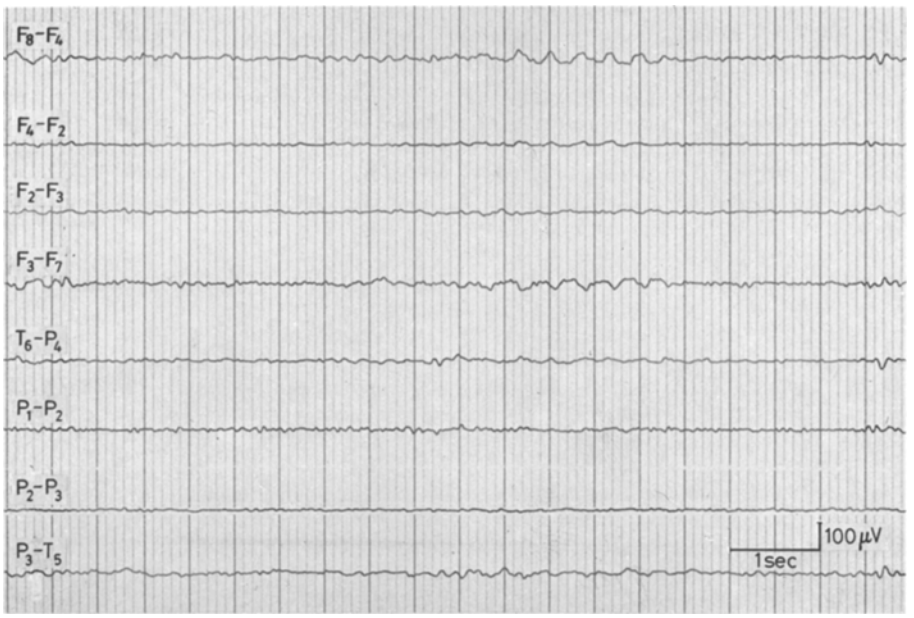

Fig. 1. Bilateral and symmetrical slow activity mainly in the temporal (midsylvian) regions, in a healthy 77-year-old subject (1st group). Calibration = 1 s/100 μV

d) Of the 26 patients with temporal slow activity who had been under observation for periods of 5–10 years, 6 suffered a cerebral accident (5 ipsilateral to the EEG irregularities) and 3 a myocardial infarct. Of the remaining 17, 10 showed an increase in the EEG irregularities and 7 showed unchanged EEG's.

Group 2. Asymptomatic patients with neurological and/or C. T. findings (total: 103; men: 51; women: 52; average age: 71.3 years), Table 1.

a) Of the 103 patients, 27 (26.2%) showed temporal slow activity, 12 (44.5%) unilaterally and 15 (55.5%) bilaterally (13 asymmetrically and 2 symmetrically). In 7 of the 12 patients and 10 of the 15, i.e. a total of 63%, temporal slow activity showed in the left hemisphere only or with the left side predominating. Of the 27 patients with temporal slow activity, 8 (29.6%) showed localization or predominance of the EEG irregularities in the area ipsilateral to the lesion. The remaining 19 patients (70.4%) showed temporal slow activity which cannot be related to the side or to the precise location of the lesion.

In this group 18.4% of the patients showed temporal slow activity not related to the organic lesion.

b) Of the 103 patients, 13 had a medical history which could be definitely attributed to a transient ischemic attack. Of these 13 patients, 4 (30.8%) showed temporal slow activity: 3 unilaterally and 1 bilaterally. In 2 of the 4 patients, angiography showed a stenosis of the carotid ipsilateral to the temporal slow activity.

Of the remaining 90 patients with no medical history of an ischemic attack, 14 (15.5%) showed temporal slow activity: 9 unilaterally and 5 bilaterally.

c) Of the 103 patients, 48 (46.6%) showed elements of a generalized angiopathy. From 18 of the 27 patients with temporal slow activity (66.7%) and 30 of the

remaining 76 patients without slow temporal activity (39.5%) there were elements of generalized angiopathy.

Group 3. Patients with clinical semeiology due to a cerebrovascular disease (total: 1,230; men: 601; women: 629; average age: 74.2 years), Table 1.

a) Of the 1,230 patients, 372 (30.2%) showed temporal slow activity, 147 (39.5%) unilaterally and 225 (60.5%) bilaterally, (188 asymmetrically and 37 symmetrically).

In 87 of the 147 patients and 151 of the 225, i.e. 64.1%, temporal slow activity in the left hemisphere only, or with the left side predominating was seen. Of the 372 patients with temporal slow activity, 130 (34.9%) showed localization or prevalence of the EEG irregularities ipsilateral to the area of the lesion. The remaining 242 patients (65.1%) showed temporal slow activity which could not be related to the side or to the precise location of the lesion. In this group 19.7% of the patients showed temporal slow activity not related to organic lesions.

b) Of the 1,230 patients, 907 (73.7%) showed elements of a generalized angiopathy. In 325 of the 372 patients with temporal slow activity (87.4%) and 582 of the remaining 858 patients without temporal slow activity (67.8%) elements of a generalized angiopathy were evident.

Group 4. Patients with clinical semeiology due to organic cerebral lesion other than vascular (total: 200; men: 102; women: 98; average age: 72.1 years), Table 1.

a) Of the 200 patients, 48 (24%) showed temporal slow activity, 18 (37.5%) unilaterally and 30 (62.5%) bilaterally (27 asymmetrically and 3 symmetrically).

In 11 of the 18 and 20 of the 30 patients, i.e. 64.6%, temporal slow activity showed in the left hemisphere only, or with the left side predominating. Of the 48 patients with temporal slow activity, 15 (31.25%) showed localization or prevalence of the EEG irregularities ipsilateral to the side of the lesion. The remaining 33 patients (68.75%) showed temporal slow activity which could not be related to the side or to the precise location of the lesion. In this group 17% of the patients showed temporal slow activity not related to organic lesions.

b) Of the 200 patients, 96 (43%) showed elements of a generalized angiopathy. 30 of the 48 patients with temporal slow activity (62.5%) and 66 of the 152 patients without temporal slow activity (43.4%) showed elements of a generalized angiopathy.

Discussion

Slow, irregular waves, usually bilateral, of various frequencies (2–6 c/s) and low to medium amplitude (usually up to 80 μV) are recorded episodically in the temporal (midsylvian) regions of a great number of aged people. This temporal slow activity, which is usually made up of three to ten elements, appears with equal frequency in both sexes, becomes more frequent as the individual gets older and is activated in half of the cases by hyperventilation (Gastaut et al. 1959). The transverse montage and the large distances between the electrodes, especially in par triangulation coronale recordings, favor the appearance of this electrographic element (Gastaut et al. 1949).

Silverman et al. (1955), evaluated that 25% of normal individuals over 60 years show temporal slow activity in routine EEG's. In a total of 605 clinically asymptomatic patients belonging to the first two groups, we evaluated the slow temporal activity at 20.7%. In the patients of the first group with a normal C. T., the percentage was evaluated at 19.5% while in the patients of the second group with findings on C. T. scan (usually small ischemic infarctions) the percentage was evaluated at 26.2%.

Gastaut et al. (1959) studied 66 patients with temporal slow activity and related them to organic cerebral syndromes, mainly vascular, with cases of generalized angiopathy. The investigators conclude that of the 51 patients with definite or very possible cerebrovascular disease and clinical semeiology, only 11 (21.6%) showed a correlation of lesion and EEG findings as far as location is concerned, while the remaining 40 (78.4%) did not show a similar correlation. The investigators' conclusion is that temporal slow activity is usually not connected with focal organic lesions but more generally with disturbances of cerebral blood flow, as a result of a localized (cerebral) or generalized angiopathy.

In the 372 patients of the third group, with temporal slow activity and confirmed cerebrovascular diseases, we found a correlation between lesion and EEG findings in 130 (34.9%), while in the remaining 242 patients (65.1%) there was no correlation. Almost similar were the findings in the patients of the second group, where the correlation between lesion and slow waves did not exceed 29.6%, and the patients of the fourth group, where the correlation between organic lesions and temporal slow activity did not exceed 31.75%. In the total number of patients with organic cerebral lesions the correlation between lesion and temporal slow activity was evaluated at 34.2%.

Our findings support the view of Gastaut et al. (1959), that is, in the greatest percentage of patients (65.8%) temporal slow activity is independent of the location of the lesion and is probably caused, as in healthy individuals, by disturbances in cerebral blood flow. This view is supported by the following finding: our patients with temporal slow activity showed a higher percentage of generalized angiopathy (79.4%) than the patients without temporal slow activity (55%), a difference of nearly 25%. It is known that generalized angiopathy due to stenosis of the cerebral arteries (arteriosclerosis) and decreased blood supply, which results in anoxia, causes bilateral slow waves, mainly in the anterior regions.

Something similar happens at the experimental stage during the breathing of an air mixture poor in oxygen when the blood saturation in oxygen approaches 70%; and at the clinical stage in serious congenital cardiopathy and serious cases of heart failure. In these cases the slow waves become less pronounced after the supply of oxygen or treatment of the heart failure. Carotid pressure also produces unilateral slow waves when the collateral circulation through the opposite carotid is not sufficient to cover the oxygen needs of the ischemic hemisphere.

These slow waves are found mainly in the temporal area because the collateral circulation through the median cerebral artery is more difficult than through the anterior cerebral, anterior communicating, posterior cerebral, and basilar arteries.

We believe that the mechanism governing the appearance of temporal slow activity in the patients of the present study is similar, and to document our view we list below our findings.

a) A higher recording percentage of temporal slow activity on the left side (65.7%), which cannot be attributed to more frequent attacks of the left vascular network in the brain but probably to the increased oxygen needs of the predominant hemisphere. Gastaut et al. (1959) evaluated this dominance at 70%.

b) A higher recording percentage of temporal slow activity in patients with a medical history of transient ischemic attacks (25.7%) in comparison with the remaining patients without a simular medical history (18.3%). The more frequent appearance of temporal slow activity unilaterally (66.7%) and its almost absolute correlation with the side of the narrowed carotid (in 10 of the 12 cases) comprise findings which were observed only in the specific group of patients with transient ischemic attacks, and support the view of an etiological relationship between temporal slow activity and local ischemia (anoxia).

c) The more frequent bilateral appearance of temporal slow activity (60.8%), which supports the view of a generalized circulatory disturbance in the brain.

d) Finally, the observation of a limited number of asymptomatic patients of the first group, which supports to some extent the vascular etiological basis of temporal slow activity, since 9 of the 26 patients (34%) had, over a period of 10 years, vascular accidents, 6 of them cerebral (5 times in correlation with the existing EEG disturbances). The remaining 17 patients (66%) are absolutely healthy, which supports a good prognosis of temporal slow activity in 2/3 of the asymptomatic aged patients without pathological findings in the axial tomography.

Kellaway (1979) also makes optimistic predictions regarding patients over 50 with temporal slow activity who were observed for 20 years without having any clinical semeiology. We believe that the encephalographist must mention in his report that "similar slow activity is recorded in normal individuals of this age" but must also keep in mind that temporal slow activity does not always comprise a specific finding of a "non-urgent" and "benign" circulatory disturbance, and that in about 1/3 of the cases it can indicate a definite focal organic cerebral lesion the nature of which can create serious diagnostic problems (Paillas et al. 1952).

The reason for this is that, as our material shows, the form of temporal slow activity does not usually help to distinguish the benign from the organic cerebral lesion, although in the latter case slow waves are more prominent and show a greater amplitude and a smaller frequency. For this reason, full cardiovascular control in these patients is necessary for the timely treatment of possible subclinical circulatory disturbances.

References

Bruens JH, Gastaut H, Giove G (1960) Electroencephalographic study of the signs of chronic vascular insufficiency of the sylvian region in aged people. Electroenceph Clin Neurophysiol 12 : 283–295

Gastaut H, Poirier F (1960) The electroencephalogram in cerebrovascular diseases. Neurology (Minneap) 14 : 110–111

Gastaut H, Paillas J, Gastaut Y (1949) L'exploration E.E.G. par les dérivations bipolaires à grande distance interélectrodes. Rev Neurol 81 : 525–528
Gastaut H, Bruens JH, Rogers J, Giove G (1959) Etude électroencéphalographique des signes d'insuffisance circulatoire sylvienne chronique. Rev Neurol 100 : 59–65
Kellaway P (1979) An orderly approach to visual analysis: parameters of the normal EEG in adults and children. In: Klass DW, Daly DD (eds) Current practice of clinical electroencephalography. Raven Press, New York, pp 69–147
Kendel K, Koufen H (1970) EEG Veränderungen bei cerebralen Gefäßinsulten des Hirnstamms. Dtsch Z Nervenheilk 197 : 42–45
Kooi KA, Gunever AM, Tupper CJ, Bagchi GK (1964) Electroencephalographic patterns of the temporal region in normal adults. Neurology (Minneap) 14 : 1029–1035
Obrist WD (1954) The electroencephalogram of normal aged adults. Electroenceph Clin Neurophysiol 6 : 235–244
Obrist WD, Busse EW, Eisdorfer C, Kleemeier RW (1962) Relation of the electroencephalogram to intellectual function in senescence. J Gerontol 17 : 197
Obrist WD, Sokoloff L, Lassen NA, Lane MH, Butler RN, Feinberg I (1963) Relation of EEG to cerebral blood flow and metabolism in old age. Electroenceph Clin Neurophysiol 15 : 610–619
Paillas JE, Bonnal J, Gastaut Y (1952) Syndrome électroclinique pseudotumoral d'origine vasculaire. Rev Neurol 86 : 160–165
Silverman AJ, Busse EW, Barnes RH (1955) Studies in the processes of aging: Electroencephalographic findings in 400 elderly subjects. Electroenceph Clin Neurophysiol 7 : 67–74
Van der Drift JHA (1961) Ischemic cerebral lesions. Angiology 12 : 401–418

Received January 15, 1982