Is the EEG really normal in lacunar stroke?

L J Kappelle, A C van Huffelen, J van Gijn

Abstract

Electroencephalograms of 12 patients with a recent lacunar infarct, 12 patients with a recent cortical infarct and 12 control patients were studied without previous information about the clinical features and the corresponding CT scan findings. EEG assessment included both visual and computerised analysis, in both the eyes closed and the eyes open condition. The specificity and the sensitivity of the EEG in the diagnosis of lacunar infarction in this study were both 0.8 (95% confidence limits 0.5-1). The positive predictive value of diagnosing a lacunar infarct on the basis of the EEG was 0.7 (95% confidence limits 0.4-0.9). The chance-corrected coefficient of agreement (kappa) between CT scanning and EEG was 0.75. Thus, in contrast to the results of previous studies, most EEGs of patients with recent lacunar infarction show rather specific abnormalities.

Lacunar infarcts are small ischaemic lesions in the deep regions of the brain, caused by an occlusion of a small perforating artery.¹ Fisher described the correlation between the different clinical syndromes and the anatomico-pathological features of these lesions more than twenty years ago.¹⁻³ As long as these lesions could be confirmed only at necropsy, an electroencephalogram (EEG) without specific abnormalities, in the presence of more or less specific clinical features, was considered obligatory for the diagnosis of a lacunar infarction.⁴⁻⁷ However, when brain imaging techniques such as CT and later MRI became available and these small ischaemic lesions could be visualised during life, large series were published about the relationship between clinical features and CT scanning in patients with lacunar infarcts.⁸⁻¹² As the EEG was no longer mandatory for the diagnosis of lacunar infarction, only a few studies about this application were published afterwards,¹³⁻¹⁹ all showing normal or non-specific results. No attention was given, however, to subtle asymmetries in alpha and mu rhythms, nor to the increased possibilities of detecting small EEG abnormalities by computer analysis in patients with minor ischaemic lesions.²⁰⁻²⁴

In several clinical series and review articles about lacunar infarcts, the EEG is therefore still considered to be normal or non-specifically abnormal.^{4-9 13-19 23-34} This study was done to assess the EEGs of patients with recent clinical

and CT scan evidence of a lacunar infarct both visually and with the aid of spectral analysis, and to compare the results with those in patients with cortical infarcts and in normal controls.

Patients and methods

Three different groups of patients, aged between 39-85 years, were studied, each consisting of 12 patients. The first group, mean age 67 years, consisted of patients with recent clinical evidence of a lacunar syndrome, according to the criteria described by Bamford.³⁵ Pure motor hemiparesis was diagnosed in seven patients, sensorimotor stroke (combined motor and sensory deficit on clinical testing) in three patients, and ataxic hemiparesis (pyramidal as well as cerebellar signs on the same side) in two patients. The patients showed no evidence of cortical dysfunction, visual field defect or clinical features of brain stem deficit. In all 12 patients, CT scans showed an appropriate lacunar infarct in the internal capsule, basal ganglia or corona radiata, without other abnormalities (Fig 1). The second group, mean age 67 years, included patients with recent clinical and radiological features of a cortical infarct. Patients with lacunar or cortical infarcts had shown clinical features for at least 24 hours.

Figure 1 CT scan of a 66 year old woman who suffered from a right-sided pure motor hemiparesis one week before. A lacunar infarct in the left corona radiata is visible.

University
Department of
Clinical
Neurophysiology and
Neurology,
University Hospital
Utrecht, The
Netherlands
L J Kappelle
A C van Huffelen
J van Gijn

Correspondence to:
L J Kappelle,
University Departments of
Clinical Neurophysiology
and Neurology,
University Hospital Utrecht,
Heidelberglaan 100,
3584 CX Utrecht,
The Netherlands

Received 10 March 1989 and
in revised form 11 July 1989.
Accepted 25 July 1989

Figure 2 (a) EEG recording of the same patient as Figure 1, three days after onset of the neurological deficit. An abundant mu rhythm in the left central region (C3) is visible.

Figure 2 (b) Power spectrum of the F_3-C_3 and F_4-C_4 derivations of the same EEG as Figure 2(a), showing the asymmetry of the mu rhythm in eyes closed as well as eyes opened condition. On the left side (F_3-C_3 , solid line) the power of the mu rhythm is much larger than that of the right side (F_4-C_4 , dotted line). Note the preponderance of the lower mu frequencies.

Table 1 Results of EEG assessment CT scan findings

EEG-assessment	CT Scan			Total
	Lacunar infarct	Cortical infarct	Normal	
Lacunar infarct	10	2	2	14
Cortical infarct		10		10
Normal	2		10	12
Total	12	12	12	36

None of them had previously suffered from a stroke. The third group (controls), mean age 60 years, were patients without a history or clinical signs of cerebrovascular disease, and with normal CT scans. All patients were selected irrespective of the use of medications or of the technical quality of the recordings.

All EEGs were recorded within 10 days of the onset of the clinical feature, on 16 or 21 channel instruments with the use of 10–20 system of electrode placement. Both bipolar and referential montages were used. Spectral analysis was performed on four pairs of homologous derivations in the eyes closed as well as the eyes open condition. Statistically significant left-right asymmetries in the spectra and spectral peaks of alpha and mu rhythm, together with their -3 dB values, were presented automatically.²² All EEGs were visually assessed by one observer (ACvH), who knew the structure of the study, but who had no information about the clinical features or the CT findings in individual patients. The presence of unilateral slow activity locally or more diffusely together with an ipsilateral

decrease of the alpha rhythm was a reason for diagnosing cortical infarcts. A lacunar infarct was established on the basis of the absence of such slow activity and the presence of asymmetries of the alpha rhythm (including frequency, amplitude, prevalence, localisation, spreading or reactivity to opening of the eyes) and/or asymmetries of the mu-rhythm (including frequency, amplitude, prevalence, localisation or reactivity to somatosensory stimulation) (Fig 2a, b). The EEG results were summarised on standard forms. The conclusion as to which of the three groups the EEG belonged to, including the side of the lesion, was made twice: first after the conventional EEG had been judged and again after the spectral analysis had been taken into account.

Results

Visual EEG assessment alone led to the correct diagnosis and to the correct side of the lesion in eight of the 12 patients with lacunar infarcts. After assessing both the conventional and quantitative EEG this figure rose to 10 of the 12 patients (table 1). The EEG recordings in two patients with lacunar infarcts were wrongly judged as normal (sensitivity 0.8; 95% confidence limits 0.5–1). Conversely, the EEGs of two patients with cortical infarcts and of two patients in the control group were misjudged as belonging to the group of patients with lacunar infarcts (specificity 0.8; 95% confidence limits 0.5–1). In each group 10 EEGs were assessed correctly. As a result the positive predictive value of the EEG in detecting lacunar infarcts

Table 2 EEG findings and interpretations of two different groups of patients with clinical and CT scan evidence of lacunar or cortical infarcts. (+ = present, – = absent, l = lacunar infarct, c = cortical infarct, n = normal)

Clinical and CT scan diagnosis	EEG	Patients											
		1	2	3	4	5	6	7	8	9	10	11	12
Lacunar infarct	Days between onset of stroke and recording	6	3	5	10	9	4	3	4	3	4	5	4
	Asymmetric delta activity	–	–	–	–	–	–	–	–	–	–	–	–
	Asymmetric alpha rhythm	–	–	–	–	–	+	+	+	+	+	+	+
	Asymmetric mu rhythm	–	–	+	+	+	–	+	+	+	+	+	+
	Interpretation	n	n	l	l	l	l	l	l	l	l	l	l
Cortical infarct	Days between onset of stroke and recording	2	1	4	4	3	2	3	4	2	2	3	5
	Asymmetric delta activity	–	–	+	+	+	+	+	+	+	+	+	+
	Asymmetric alpha rhythm	–	+	+	+	+	+	+	+	+	+	+	+
	Asymmetric mu rhythm	+	+	+	+	+	–	–	–	–	–	–	–
	Interpretation	l	l	c	c	c	c	c	c	c	c	c	c

was 0.7 (95% confidence limits 0.4–0.9). In view of the patient selections, one out of three EEG diagnoses might be correct on the basis of chance alone. If these effects were taken into account by means of the coefficient kappa, the agreement between CT and EEG was 0.75 (maximal agreement is 1.0). The decisive EEG findings in patients with lacunar and cortical infarcts are summarised in table 2. The timing of the EEG recording did not influence the results: the EEGs that were misclassified were not recorded later, within the study period of 10 days, than the others (table 2). We did not find a relationship between the EEG findings and the precise location of the lacunar infarct on the CT scan in the basal ganglia, internal capsule or corona radiata.

Discussion

Lacunar infarcts, if at all symptomatic, usually cause typical clinical features known as lacunar syndromes.³⁶ Nevertheless, lacunar syndromes may occasionally be caused by lesions other than lacunar infarcts^{5 8 10 11 25–28 35} and confirmation by ancillary studies is often important.

This study clearly demonstrates that lacunar infarcts can give rise to subtle EEG abnormalities, in contrast to previous studies.^{4–9 13–19 25–34} The EEG is reasonably specific and sensitive in diagnosing lacunar infarcts, although the 95% confidence limits are wide because of the small number of patients studied. The positive predictive value of the EEG diagnosis “lacunar infarction” in this group of patients was 0.7 (95% confidence limits: 0.4–0.9), and if the *a priori* probability is taken as 0.3 (not excluding the clinical features) the diagnostic gain is 0.4.

Asymmetries in the alpha and especially in the mu rhythm are important in diagnosing lacunar infarcts, whereas focal slow abnormalities that were the main focus of attention in previous studies,^{4 13–19} were lacking. Most lacunar infarcts could be identified after visual assessment of the EEG. However, computer analysis increased the sensitivity because minor asymmetries in the alpha and mu rhythms that escaped visual assessment could also be demonstrated. The use of medication, the age of the patients and the quality of the EEG recording play a prominent role in determining whether EEG abnormalities can be correctly identified,^{22 24} but from a pragmatic point of view we did not select our patients according to these factors. The time interval between the onset of the clinical features and investigations is another important factor, as most abnormalities on the EEG are found immediately after the onset of the clinical features,^{20 22–24} whereas CT scans are usually normal during the first few days.^{37 38} It is therefore likely that electroencephalography confirms a lacunar infarct at an earlier stage than the CT scan. Lacunar infarcts cannot be verified with CT in about one third of clinically suspected cases.^{11 12 28 35} As our patients all had an appropriate lacunar infarct on the CT scan, we can only speculate on the value of the EEG in

patients with lacunar stroke and normal CT scans.

Finally, we wish to stress that CT scanning or magnetic resonance imaging remain the preferred diagnostic methods if confirmation is necessary in patients with lacunar syndromes. The EEG, however, may be helpful in some cases where scanning facilities are not available. Further studies are needed to establish the value of the EEG in patients with lacunar stroke and normal CT scans.

We thank Dr E A C M Sanders for his constructive comments and Mrs M Schipper and Mrs E Budelman-Verschuren for their secretarial help.

- 1 Fisher CM. Lacunes. Small deep cerebral infarcts. *Neurology* 1965;15:774–84.
- 2 Fisher CM. The arterial lesions. Underlying lacunes. *Acta Neuropath (Berlin)* 1969;12:1–15.
- 3 Fisher CM. Capsular infarcts. The underlying vascular lesions. *Arch Neurol* 1979;36:65–73.
- 4 Caplan LR, Young RR. EEG findings in certain lacunar stroke syndromes. *Neurology* 1972;22:403.
- 5 Fisher CM, Curry HB. Pure motor hemiplegia of vascular origin. *Arch Neurol* 1965;13:30–44.
- 6 Caplan LR. Lacunar infarction: a neglected concept. *Geriatrics* 1976;31:71–5.
- 7 Haferkamp G. Das krankheitsbild des lakunären hirnininfarktes. *Akt Neurol* 1977;4:115–20.
- 8 Mohr JP, Caplan LR, Melski JW, Goldstein RJ, Duncan GW, Kistler JP, Pessin MS, Bleich HL. The Harvard Cooperative Stroke Registry: A prospective registry. *Neurology* 1978;28:754–62.
- 9 Weisberg LA. Lacunar infarcts. Clinical and computed tomographic correlations. *Arch Neurol* 1982;39:37–40.
- 10 Pullicino P, Nelson RF, Kendall BE, Marshall J. Small deep infarcts diagnosed on computed tomography. *Neurology* 1980;30:1090–6.
- 11 Nelson RF, Pullicino P, Kendall BE, Marshall J. Computed tomography in patients presenting with lacunar syndromes. *Stroke* 1980;11:256–61.
- 12 Donnan GA, Tress BM, Bladin PF. A prospective study of lacunar infarction using computerised tomography. *Neurology* 1982;32:49–56.
- 13 Yanagihara T, Houser OW, Klass DW. Computed tomography and EEG in cerebrovascular disease. *Arch Neurol* 1981;38:597–600.
- 14 Primavera A, Folis E, Romagnoli P, Ruffinengo U, Barolini A. The EEG in lacunar strokes. *Stroke* 1984;15:579–80.
- 15 Zorzon M, Masè G, Iona LG, Cazzato G. Electroencefalogramma ed infarti lacunari. *Riv Neurol* 1986;56:293–9.
- 16 Falcone N, Fensore C, Lanzetti A, Lanzarino LG, Nappo A, Nicolai A, Tomasi G. Considerazioni cliniche e correlazioni EEG-TC negli infarti lacunari. *Riv Neurol* 1986;56:396–410.
- 17 Schaul N, Green L, Peyster R, Gotman J. Structural determinants of electroencephalographic findings in acute hemispheric lesions. *Ann Neurol* 1986;20:703–11.
- 18 Janati A, Kidwai S, Balachdran S, Dang MT, Harrington D. A comparative study of electroencephalography and computed axial tomography in recent cerebral infarction. *Clin Electroencephal* 1987;18:20–5.
- 19 Macdonell RAL, Donnan GA, Bladin PF, Berkovic SF, Wriedt CHR. The electroencephalogram and acute ischemic stroke: distinguishing cortical from lacunar infarction. *Arch Neurol* 1988;45:520–4.
- 20 Van Huffelen AC, Poortvliet DCJ, van der Wulp CJM, Magnus O. Quantitative EEG in cerebral ischemia. Parameters for the detection of abnormalities in “normal” EEGs, in patients with acute unilateral cerebral ischemia. In: Lechner H, Aranibar A, eds. *EEG and clinical neurophysiology*. Excerpta Medica, Amsterdam 1980: 125–30.
- 21 Pfurtscheller G, Sager W, Wege W. Correlations between CT-scan and sensorimotor EEG rhythms in patients with cerebrovascular disorders. *Electroenceph Clin Neurophysiol* 1981;52:473–85.
- 22 Van Huffelen AC, Poortvliet DCJ, Van der Wulp CJM. Quantitative electroencephalography in cerebral ischemia. Detection of abnormalities in “Normal” EEGs. In: Pfurtscheller G, Jonkman EJ, Lopes da Silva FH, eds. *Brain ischemia: quantitative EEG and imaging techniques*. Progress in brain research. Amsterdam: Elsevier 1984: 3–28.
- 23 Köpruner V, Pfurtscheller G, Auer LM. Quantitative EEG in normals and in patients with cerebral ischemia. In: Pfurtscheller G, Jonkman EJ, Lopes da Silva FH, eds.

- Brain Ischemia: Quantitative EEG and imaging techniques. Progress in brain research.* Amsterdam: Elsevier 1984: 29-50.
- 24 Jonkman EJ, Van Huffelen AC, Pfurtscheller G. Quantitative EEG in Cerebral Ischemia. In: Lopes da Silva FH, Storm van Leeuwen W, Remond A eds. *Clinical applications of computer analysis of EEG and other neurophysical signs.* Amsterdam: Elsevier 1986:205-37.
 - 25 Weisberg LA. Computed tomography and pure motor hemiparesis. *Neurology* 1979;29:490-5.
 - 26 Rascol A, Clanet M, Manelfe C, Guiraud B, Bonafé A. Pure motor hemiplegia: CT study of 30 cases. *Stroke* 1982;13:11-7.
 - 27 Soisson T, Cabanis EA, Iba-Zizen MT, Bousser MG, Laplane D, Castaigne P. Hémiplegie motrice pure et tomodesitométrie. *J Neuroradiol* 1982;9:304-22.
 - 28 Loeb C, Gandolfo C, Mancardi GL, Primavera A, Tassinari T. The lacunar syndromes: a review with personal contribution. In: Lechner H, Meyer JS, Ott E, eds. *Cerebrovascular disease: research and clinical management.* Amsterdam: Elsevier 1986;1:107-56.
 - 29 Donnan GA, Zapf P, Doyle AE, Bladin PF. CSF enzymes in lacunar and cortical stroke. *Stroke* 1983;14:266-9.
 - 30 Dobkin BH. Heparin for lacunar stroke in progression. *Stroke* 1983;14:421-3.
 - 31 Mohr JP. Lacunes. *Stroke* 1982;13:3-11.
 - 32 Mohr JP. Lacunes. *Neurol Clin* 1983;1:201-21.
 - 33 Dickmann E, Müller E. Lakunäre Hirninfarkte. *Z Gerontol* 1985;18:222-5.
 - 34 Caplan LR, Stein RW. Small Vessel Disease. In: *Stroke A Clinical Approach.* London: Butterworths 1986:167-77.
 - 35 Bamford J, Sandercock P, Jones L, Warlow C. The natural history of lacunar infarction: The Oxfordshire Community Stroke Project. *Stroke* 1987;18:545-51.
 - 36 Fisher CM. Lacunar strokes and infarcts: a review. *Neurology* 1982;32:871-6.
 - 37 Calandre L, Gomara S, Bermejo F, Millan JM, Del Pozo G. Clinical-CT correlations in TIA, RIND, and strokes with minimum residuum. *Stroke* 1984;15:663-5.
 - 38 Wall SD, Brant-Zawadzki M, Jeffrey RB, Barnes B. High frequency CT findings within 24 hours after cerebral infarction. *AJR* 1982;138:307-11.