

**HAM-TMC LIBRARY INTERLIBRARY LENDING  
ARTICLE/PHOTOCOPY**

Wednesday, November 04, 2009

**ILLiad TN: 203311**



**ILL Number: 59473443**



**Shipping Address:**

14-DAL VIA TEXPRESS  
Texas Women's University  
P.O. Box 425528  
304 Administration Drive  
Denton, TX 76204-5528

**Borrower: IWU System: OCLC**

**Maxcost: \$0**

**Lending String:** \*TMC,TMC,AHX,AHX,TBM

**Patron:** Souris, Dr. Stephen

**Journal Title:** Clinical electroencephalography.

**Volume:** 18 **Issue:** 4

**Month/Year:** 10 1987**Pages:** 201-10

**Article Author:**

**Article Title:** Asokan, G; Temporal minor slow and sharp EEG activity and cerebrovascular disorder

**NOTICE:**

**THIS MATERIAL MAY BE PROTECTED BY  
UNITED STATES COPYRIGHT LAW  
(TITLE 17, U.S.C.)**

(S) Batch:

(S) Invoice #:

Solomon-ID:

Billing Category: Exempt

EFTS: No

Document Charges:	
Additional Charges:	
<b>Total Due:</b>	

**Call #/Location:**

Reasons-for-Non-Supply

- BDY (At Bindery)
- CST (Cost exceeds stated maxcost)
- INC (Not as Cited)
- LAC (Lacking volume/issue)
- LOS (Lost)
- NYR (Not Yet Received)
- NOS (Not on Shelf)
- NOT (Title Not Owned)
- OTH (Other) Specify: \_\_\_\_\_

**DELIVERY: Ariel**

**Fax:** 940/898-3726

**Ariel:** 70.129.12.30

**Phone:**

*This document has been supplied to you  
from:*

OCLC: TMC  
DOCLINE: TXUTEX

**44-HOU via TExpress  
HAM-TMC Library  
1133 JOHN FREEMAN BLVD  
HOUSTON, TX 77030**

**Hours: Monday - Friday, 8am - 5pm**

**Phone: 713-799-7105**

**Fax: 713-790-7056**

**ARIEL: 192.68.30.164**

*Email:*

**TMCARIEL@EXCH.LIBRARY.TMC.EDU**

*Thank You for Using Our Services!*

**THIS IS NOT AN INVOICE!  
PLEASE WAIT TO BE BILLED.**

# Temporal Minor Slow and Sharp EEG Activity and Cerebrovascular Disorder

G. Asokan, J. Pareja and E. Niedermeyer

## Key Words

Cerebrovascular disorders  
Electroencephalography  
Hippocampic Ischemia  
Temporal Minor Slow and Sharp Activity  
Vertebrobasilar Artery Insufficiency  
Wicket Spikes

## Introduction

Focal EEG changes located over the anterior temporal and midtemporal regions represent a fairly common pattern which is most often found in old age. This pattern consists of runs of medium voltage 2-7/sec waves mixed with lower amplitude 8-14/sec waves and some intermingled minor sharp transients, which occasionally appear to be more prominent (even assuming the character of frank spikes). This pattern is - in accordance with almost all observers - most often lateralized to the left. It is usually most prominent in light drowsiness and may linger on into light NREM sleep with mild changes in its wave morphology.

Temporal minor slow wave and sharp activity (TMSSA) is a well known pattern in hospitalized individuals<sup>1,2</sup> and aged community volunteers.<sup>3,4</sup> Gibbs and Gibbs<sup>5</sup> have used the term "minimal focal (temporal) slow activity," but there is no doubt that the slowing is quite often mixed with more or less sharp wave forms. For this reason, a term like TMSSA appears to be preferable. There are also two variants of TMSSA. One variant consists of rhythmical activity over the anterior temporal and midtemporal regions. This rhythmical activity may occur in long trains and must be differentiated from the posterior alpha rhythm. In general, the temporal rhythmical activity lies in the 6-9/sec range and is somewhat slower than the posterior alpha rhythm. Like TMSSA, the rhythmical variant is also quite often lateralized to the left.<sup>6</sup> Maynard and Hughes<sup>7</sup> have introduced the term "bursts of rhythmical temporal theta," but it must be said that the

rhythmical variant is not necessarily of burst-like character. A second variant of TMSSA occurs in sleep and consists of brief trains of rhythmical spiking over the anterior and midtemporal areas. This pattern had been termed "wicket spikes".<sup>8</sup>

TMSSA has been ascribed to old age as well as to cerebrovascular disorder. Naturally, these two conditions very often coincide. There are, however, reasons to presume that cerebrovascular disorder is the usual cause of TMSSA. This view has been supported by the recent work of Visser et al.<sup>9</sup>

The clinical value of TMSSA is not widely enough known. For this reason, another study of this pattern appears to be worthwhile.

## Material and Methods

This study is based upon the two-year intake of EEG recordings of our laboratory (serving the Johns Hopkins as a central laboratory).

EEG records were obtained on 16- or 20-channel instruments. The International (10-20) Electrode System was employed with bipolar and referential (unipolar) montages. Sleep records (mostly unседated) were obtained when feasible. Intermittent photic stimulation was used in all patients; hyperventilation was also obtained except in patients with stroke problems.

The records were visually analyzed; all of the tracings were seen by one of us (E.N.). Examples of typical TMSSA are shown in Figures 1-3. Rhythmical activity in the theta-alpha range over the anterior temporal-midtemporal region was regarded as a variant

From the Department of Neurology, The Johns Hopkins University School of Medicine and Hospital, Baltimore, Maryland. G. Asokan is a Fellow of WHO (from Barbados), J. Pareja is a Former Fellow (from Cartagena, Colombia), and E. Niedermeyer is Director of EEG, Johns Hopkins Hospital.

Requests for reprints should be addressed to Ernst Niedermeyer, M.D., The Johns Hopkins Hospital, 600 North Wolfe Street, Baltimore, Maryland 21205.

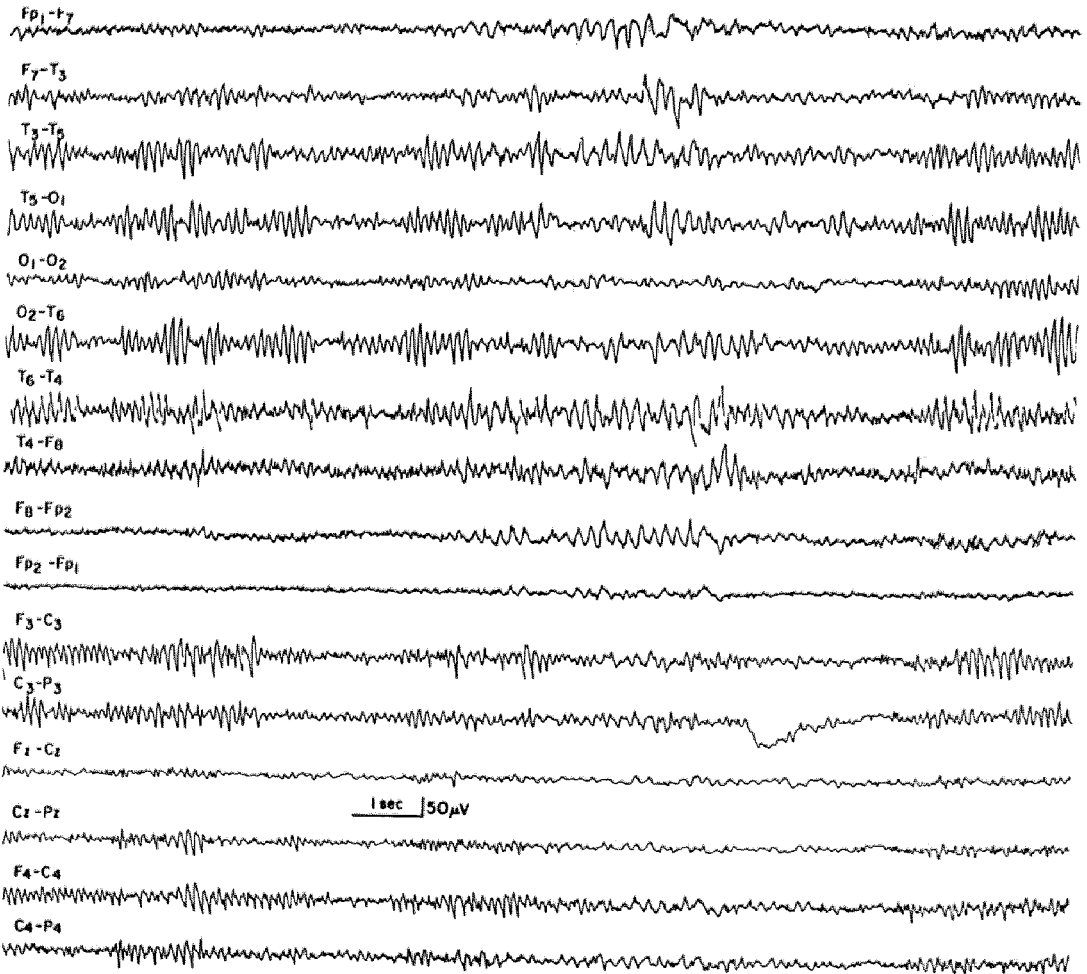
of TMSSA (Figure 4). The clinical charts of the patients were extensively reviewed with regard to clinical diagnostic impression and prominent symptoms.

**Results**

From a total of 6,392 records obtained in our EEG laboratory during the calendar years of 1983 and 1984, 227 tracings (from 209 patients) were retained because of the presence of TMSSA. This finding indicates a prevalence of 3.5% (out of a patient population which also comprises children, infants and newborns). The consistency of the prevalence is demonstrated by the incidence of 3.7% for 1983 and

3.3% for 1984.

Table 1 shows the age and sex distribution of TMSSA with the expected peak in the 7th decade of life. Interestingly, there was a small (but not negligible) number of patients in the age range of 21 to 40 years. A 2:1 predominance of female patients with TMSSA came as a surprise. Table 2 demonstrates the lateralization of TMSSA and the expected preponderance of the left side (84.14% predominantly on the left). Table 3 indicates the occurrence of special variants of the TMSSA pattern with a considerable number of patients (95) with rhythmical stretches and 14 patients with wicket spikes in sleep (Figure 5). The occurrence of frank spikes



**Figure 1.** Age 56 years. Depression with chronic pain. Note a run of 5.5-7/sec activity mixed with lower amplitude 8-11/sec waves over the left anterior temporal - midtemporal area. There is also a run of 6-8/sec activity over the right anterior temporal-midtemporal area, slightly less prominent. The posterior alpha rhythm is in the 8-10/sec range.

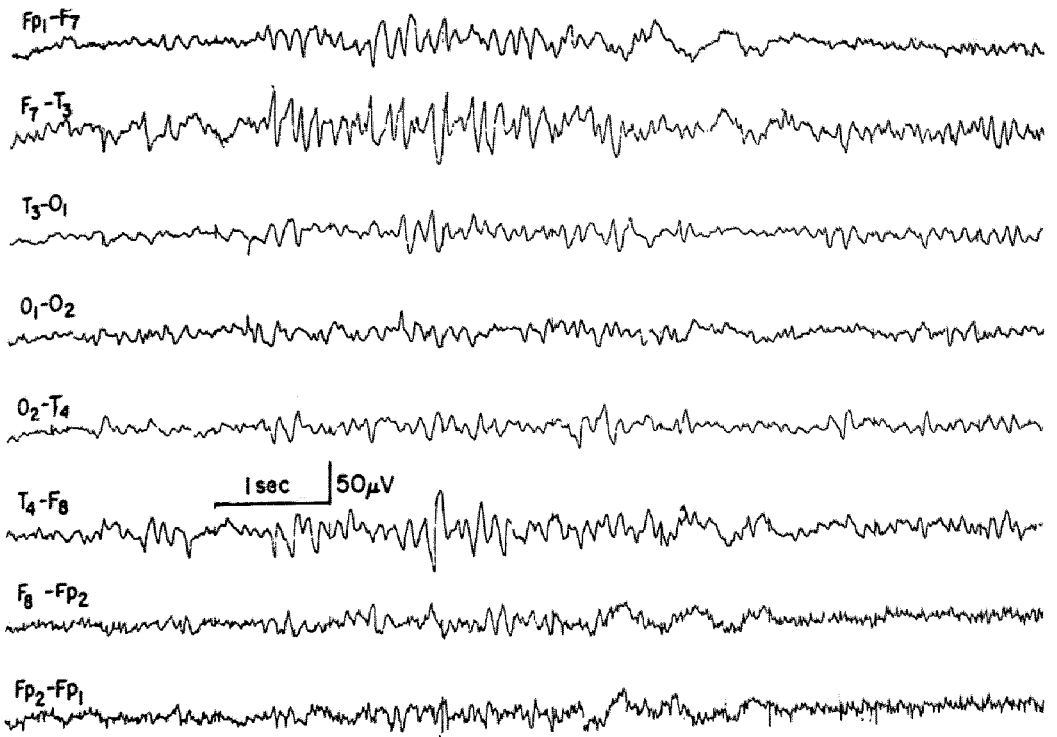


Figure 2. Age 54 years. Incipient cerebrovascular disorder with occasional episodes of dimmed vision and slurred speech. A run of anterior temporal-midtemporal 8-9/sec activity is shown, more prominent on the left, somewhat sharp. This is followed by a brief train of anterior delta waves. (Courtesy of Urban and Schwarzenberg Medical Publishers, Baltimore, MD)

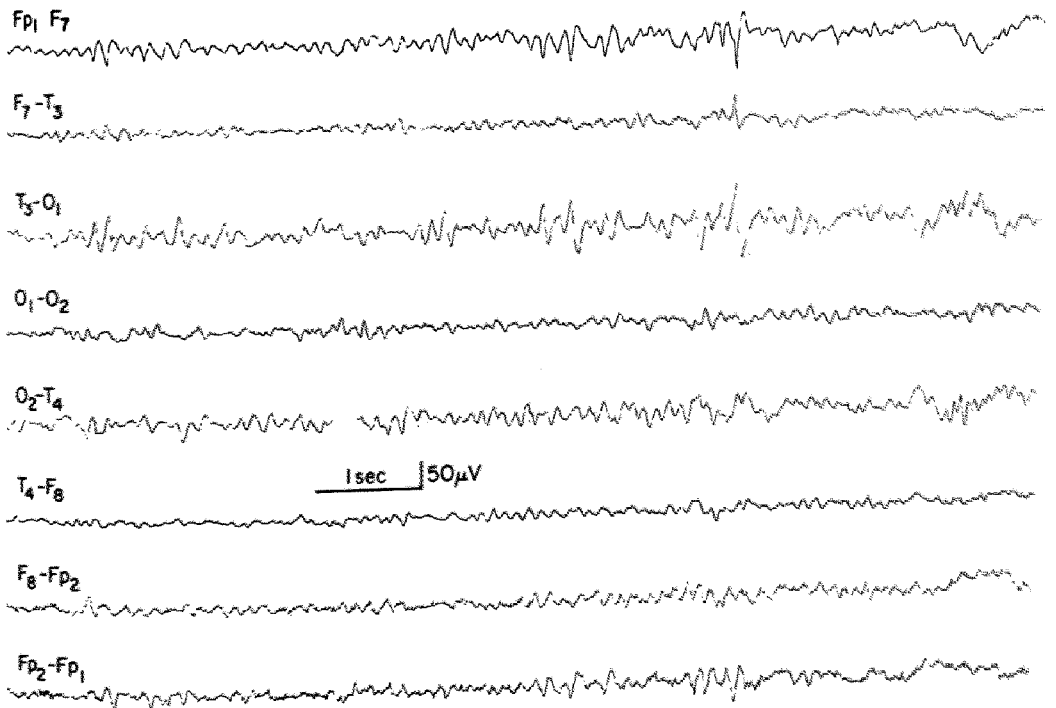
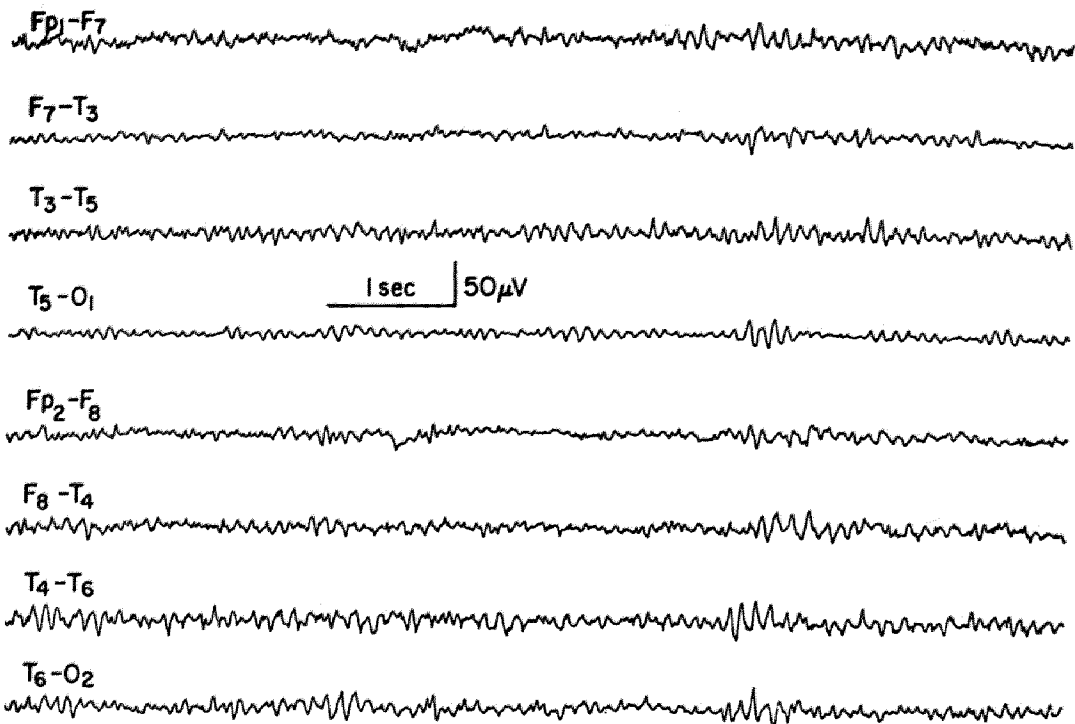


Figure 3. Same patient as in Figure 2. Note distinct spike discharge over the left anterior temporal area, spreading into the left midtemporal region. (Courtesy of Urban and Schwarzenberg Medical Publishers, Baltimore, MD)



**Figure 4.** Age 61 years. Early cerebrovascular disorder and evidence of coronary disease. Note posterior 9-11/sec alpha rhythm in channels 3, 4, 7 and 8. Also noted independent rhythmical 8-10/sec activity over the anterior temporal-midtemporal regions, slightly more prominent on the left. (Courtesy of Urban and Schwarzenberg Medical Publishers, Baltimore, MD)

**TABLE 1**

AGE AND SEX DISTRIBUTION OF PATIENTS

SEX	AGE								Total
	21-30	31-40	41-50	51-60	61-70	71-80	81-90	91-100	
MALE	4	8	8	14	19	12	4	0	69
FEMALE	4	9	19	31	39	26	11	1	140
TOTAL	8	17	27	45	58	38	15	1	209

Males with TMSSA 33.01%

Females with TMSSA 66.99%

**TABLE 2**

LATERALIZATION OF TEMPORAL MINOR SLOW AND SHARP ACTIVITY

SEX	ONLY ON LEFT	MORE ON LEFT	ONLY ON RIGHT	MORE ON RIGHT	BOTH SIDES	TOTAL
	MALE	29	37	1		
FEMALE	62	63	5	10	14	154
TOTAL	91	100	6	11	19	227

Predominantly left - 191

84.14%

Predominantly right - 17

7.49%

Both sides equally involved - 19

8.3%

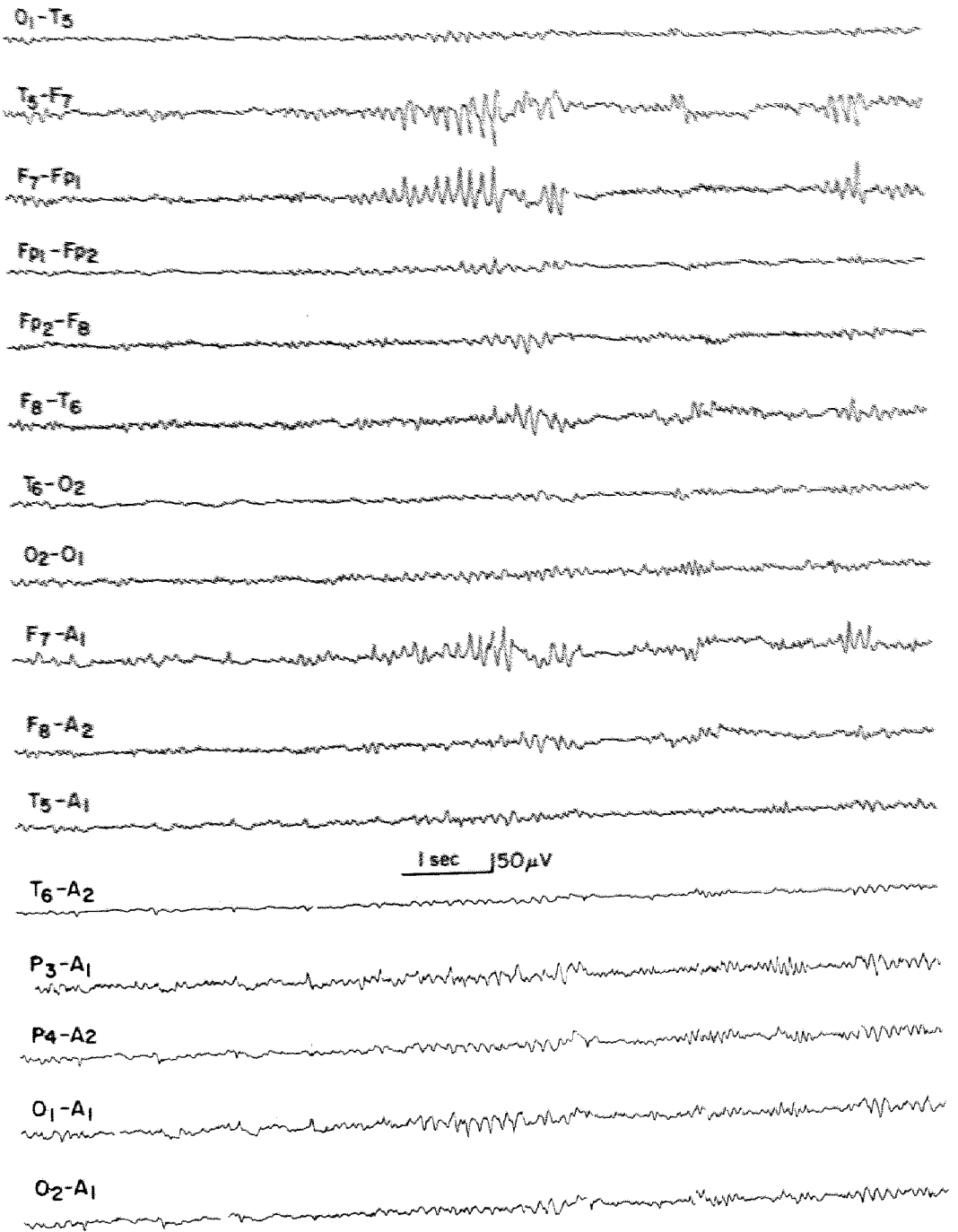


Figure 5. Age 62 years. Cerebrovascular disorder. Two trains of "wicket spikes" are noted over the left anterior temporal area during light NREM sleep.

(i.e. spikes undistinguishable from classical spikes found in epileptic patients) (Figure 3) in 6 patients will require special discussion. The following other abnormalities were found in

patients with TMSSA: diffuse slowing in 30, and focal slowing in 6 patients.

Table 4 lists the most prominent clinical symptoms or symptom-complexes, whereas

the leading clinical diagnostic impressions are demonstrated in Table 5.

**Discussion**

1). *The pattern*

The pattern of TMSSA poses some interesting questions. Several authors have placed special emphasis on the term "temporal slowing." As a matter of fact, recurrent runs of prominent activity in the delta range and especially continuous temporal delta activity should be

separated from TMSSA. Consistent temporal slowing has a different meaning and may denote a distinct unilateral or bilateral temporal lesion or dysfunction of more serious implications. In such cases, states of middle cerebral artery insufficiency (precursors of classical strokes in this territory) may be the cause<sup>10,11</sup> and, of course, other causes including neoplastic etiology must also be considered.

Unlike recurrent delta runs, TMSSA is usually a mixed pattern. If it does show predominantly rhythmical features, then the rhythmical activity tends to lie in the 6-9/sec. range. Most commonly, however, there are 2-7/sec waves with intermingled 8-14/sec activity. There may be short runs of a few delta (2-3/sec) waves but, in general, the delta frequency range does not stand in the foreground. For this reason, methods of visual or automatic frequency analysis may be misleading since the "gestalt" of a complex pattern is crucial for the detection of TMSSA. This does not invalidate approaches utilizing frequency analysis<sup>9</sup> or placing special stress on the distinction of delta and theta frequency bands.<sup>12,13</sup> The analysis of only short stretches of wakefulness in the evaluation of the records is conducive to further problems. Quite often, faintly suggestive patterns of TMSSA are present in the waking state whereas in earliest drowsiness this pattern becomes quite impressive.

The sharp character of TMSSA must not be ignored. Some runs of this pattern may show no sharp features whatsoever, while others are characterized by a gamut of sharp elements which may reach from minimally sharp

**TABLE 3**

SPECIAL VARIANTS OF TEMPORAL MINOR SLOW AND SHARP ACTIVITY	
	N
Rhythmical activity over anterior temporal-midtemporal area	95
Frank spikes	6
Wicket spikes (in sleep)	14

**TABLE 4**

LEADING SYMPTOMS AND SIGNS (In order of significance)	
Dizziness and vertigo	
Memory loss and intellectual decline	
Syncope	
Headache and migrainous symptoms	
Confusion	
Blurred vision	
Delirious	

**TABLE 5**

DIAGNOSTIC IMPRESSION	
	N*
Cerebrovascular/Cardiovascular disease (without stroke)	48
Hypertension	41
Cerebrovascular accident	30
Carotid artery disease	27
Transient ischemic attacks	26
Endogenous depression	21
Migraine	15
Diabetes mellitus	14
Chronic alcoholism	14
Epileptic seizure disorder	12
Status post-cardiac surgery	9

\*There were more than one diagnostic labels in several patients.

transients to frank spikes. All this must be kept in mind in order to do justice to the complex nature of TMSSA. While all these variations of TMSSA still form a distinct pattern, it might be worthwhile to single out the rhythmical variant (usually 6-9/sec) and the cases characterized by intermixed frank sharp waves or spikes. The former must be distinguished from a posterior alpha (or sub-alpha) basic rhythm by virtue of the spatial distribution, while the latter needs special discussion because of possible epileptological consequences. This is also true for the "wicket spikes" which are almost always confined to light NREM sleep. There is little doubt that wicket spikes are just a variant of TMSSA which evolves in early sleep.

## 2). *The lateralization*

Practically all investigators of TMSSA agree on the impressive lateralization of this pattern to the left side. Earlier work of Busse et al.<sup>14</sup> placed stress on left-sided predominance of TMSSA in 78%. Subsequent studies confirmed these findings (75% to 90%). Thus, our own figure of 84% is congruent with those of other studies.

According to Visser et al.,<sup>9</sup> verbal fluency and word association productivity tests are impaired in individuals with TMSSA while, on the other hand, the presence of this pattern is usually regarded as being unrelated to neurological deficits.<sup>15</sup> The cause for the consistent lateralization of TMSSA remains unclear, but one cannot simply ignore the role of hemispherical dominance. One is even tempted to speculate that the dominant hemisphere could be subject to greater stress and more prone to vascular pathology. There is, however, no convincing lateralization of the typical hemiplegic (capsular) cerebrovascular accidents to the left side. One could further speculate that greater life-long stress is endured by the mechanisms of verbal memory and their hippocampic substratum. Although no significant differences have been found thus far between the memory functions of elderly persons with and without TMSSA,<sup>16,17</sup> the role of verbal memory and hippocampic ischemia in the genesis of TMSSA ought to be further investigated.

## 3). *The level of vigilance*

Light drowsiness is, in general, most conducive to the demonstration of TMSSA. In patients with weak development of this pattern, its occurrence may be limited to light drowsiness. Whenever TMSSA is more strongly

developed, the pattern is found in the waking state, appears to be enhanced in light drowsiness and shows some decline in deeper drowsiness and light NREM sleep.

TMSSA may change its wave morphology in the course of changes of vigilance. In sleep, it may turn into a rhythmical repetition of (rather unimpressive) spikes over the anterior temporal-midtemporal region. This pattern has been termed "wicket spikes",<sup>8</sup> and the first describers were aware of the rather benign and non-epileptic nature of this type of discharge.

## 4). *TMSSA: an abnormal or normal pattern?*

It is interesting to note that TMSSA was the only abnormality in 173 out of 209 patients (82.8%). This underscores the importance of the interpretation. If the electroencephalographer feels that TMSSA is an essentially normal pattern or a variant of normalcy, all of these records will be read as within normal limits of variability.

We feel that such a philosophy would be too restrictive and deprive the referring clinician of worthwhile information. A quantifying approach is most advisable, i.e. an assessment of prominence, frequency, rarity and inconspicuousness of the pattern in a given record. Therefore, we read records with infrequent and inconspicuous examples of TMSSA as "within broad normal limits of variability," whereas the majority of the tracings were read as "minimally abnormal" or "slightly abnormal" according to quantity and impressiveness of the pattern. TMSSA alone never constitutes a marked abnormality.

## 5). *The role of age and sex*

There is no doubt that TMSSA represents a pattern of old age but there are exceptions. In our material, 25 out of 209 patients (11.9%) were below age 40, of whom 8 (3.8%) were between 21 and 30 years. These figures alone support the view that TMSSA is not simply a pattern of old age. In full agreement with Visser et al.,<sup>9</sup> we feel that TMSSA is an EEG expression of cerebrovascular disorder which, under unusual circumstances, may also occur in younger adults. For this reason, we reviewed the records of patients below age 40 with particular interest. Unusually early occurrence of cerebrovascular disorder such as lupus erythematosus and other connective tissue diseases may be conducive to TMSSA. Even dysfunctional disturbances such as migraine (15 cases in our material, including cases of basilar artery

migraine) may be associated with this pattern.

In general, however, TMSSA is a sign of structural cerebrovascular disorder, and there is good reason to presume that this pattern is more likely to indicate early or moderately advanced stages of the disease.

In our material, there is also an unusual 2:1 predominance of the female gender, with 140 females and 69 males. Sex preferences of EEG patterns are very uncommon (such as the predominance of females in 6/sec spike-waves of the posterior type, or frontocentral beta activity of old age). It is a reasonable conjecture however, that cerebrovascular conditions leading to TMSSA are more likely to be found in females than in males.

#### 6). *The clinical correlates*

A listing of the symptomatology found in patients with TMSSA (Table 4) clearly shows a predominance of disturbance attributable to cerebrovascular disorder. The vertebrobasilar system might be chiefly involved when one considers the nature of the leading symptoms. Decline of memory is very frequently encountered (even though it may not be one of the principal symptoms). One has to consider that the hippocampic region - a crucial structure for processing and recall of memory material - depends chiefly on the vertebrobasilar and posterior cerebral artery system. The role of TMSSA in cases of vertebrobasilar artery insufficiency has been pointed out in the past.<sup>18,19</sup> Anderson et al.<sup>20</sup> have shown fine examples of TMSSA in well documented cases of vertebrobasilar artery insufficiency.

On the other hand, TMSSA has also been observed in patients with carotid artery disease and typical cerebrovascular accidents. In such cases, serious involvement of the internal carotid-middle cerebral artery system co-exists with clinically less significant involvement of the vertebrobasilar system. The isolated appearance of TMSSA is more likely to indicate vertebrobasilar circulatory problems of rather mild nature, rather than impending capsular strokes caused by insufficiency of the internal carotid - middle cerebral artery system.

Not seldom, patients with TMSSA suffer from depression of endogenous character. In such cases, the EEG findings can serve as an important guideline for the psychiatrist by the demonstration of suggestive cerebrovascular problems. In another study, a rather high prevalence of TMSSA was found in older

patients with severe chronic pain.<sup>21</sup>

A more difficult problem is posed in patients with epileptic seizure disorder of old age onset and TMSSA (with no other abnormalities). In such patients, TMSSA is more likely to show sharp elements or even frank spikes, but on this basis alone one should not construe a case of epileptic seizure disorder since most of these cases are unassociated with seizures. The anterior temporal spike focus shows a natural tendency towards greater prominence in older age<sup>22</sup> and plays a major role in epilepsies of old age onset.<sup>23</sup> While TMSSA often shows a varying degree of paroxysmal features, this pattern may also be an EEG correlate of overt old age epilepsy. The pattern alone, however, cannot reveal its significance as a potentially or overtly paroxysmal abnormality. These uncertainties may pose an unpleasant problem for the electroencephalographer when TMSSA of spiky appearance is found in a patient referred with the question of "epileptic seizures vs. syncopal fainting." Unfortunately, the EEG reader will be unable to give a satisfactory answer to the specific question. In general, syncope is more commonly found in such a context. The electroencephalographer faces a similar dilemma when the same question is asked along with the referral of a patient featuring "small sharp spikes" and no other abnormalities.<sup>24</sup>

TMSSA is not uncommon in patients with chronic alcoholism. One should keep in mind that alcoholics tend to develop early cardiovascular and cerebrovascular disorders. Accompanying heavy cigarette smoking contributes further to the vascular problems.

In the common differential diagnosis between senile dementia Alzheimer-type and cerebrovascular (multi-infarct) dementia, the occurrence of TMSSA strongly suggests a vascular basis. An overlap of these two major forms of dementia is quite uncommon. Elderly persons of remarkable cardiovascular health may fall victim to Alzheimer-type dementia

#### **Concluding Remarks**

TMSSA represents a rather minor abnormality (provided that it reaches abnormal proportions) but it is a diagnostically very helpful pattern. It is presumably a very important indicator of cerebrovascular disorder in its early stage. More seldom, it is the transient expression of merely dysfunctional cerebrovascular problems.

Why is the antero-midtemporal region the site of the pattern (which, incidentally, is not found outside the temporal lobe)? Its relationship to vertebrobasilar artery insufficiency and its lack of relationship to capsular strokes (unless vertebrobasilar and middle cerebral artery disorder co-exist) point to the hippocampic region which is well known for its relatively poor blood supply.<sup>25,26</sup> As noted before, the very frequently observed lateralization to the left hemisphere cannot be readily explained. One is tempted to speculate that the left hippocampus (subserving processing and recall of verbal memory) is more vulnerable to mild ischemic conditions.

The interpretation of TMSSA represents a challenge for the electroencephalographer who must refrain from over- and underrating the significance of this pattern. Modern views in clinical electroencephalography tend to minimize or even ignore such minor deviations. Such trends can be detrimental to EEG by depriving the electroencephalographer of important clinical-electrical correlations and withholding valuable information from the referring clinician.

EEG is indeed a much better diagnostic tool in the field of cerebrovascular disorder than widely assumed - this is true for acute massive

stroke<sup>27</sup> as well as for chronic mild states of cerebrovascular insufficiency found in conjunction with TMSSA.

### Summary

The clinical significance of temporal minor slow and sharp activity (TMSSA) is discussed on the basis of earlier literature and personal observations (209 patients, 227 EEG records).

This pattern consists of mixed 2-7/sec and 8-14/sec activity with intermingled minor sharp transients (occasionally even frank spikes) over the anterior temporal-midtemporal region and, in the vast majority (84% in our material) predominantly on the left side. This pattern is most prominent in early drowsiness, and may change to rhythmical spiky discharges in light NREM sleep ("wicket spikes").

The origin of TMSSA is unclear. There is reason to believe that hippocampic ischemia might be the underlying substratum but the evidence remains tenuous. Vertebrobasilar artery insufficiency states may result in TMSSA since the hippocampus largely depends on this vascular system (via posterior cerebral artery).

The occurrence of TMSSA usually represents a mild abnormality of potentially considerable clinical significance.

### REFERENCES

1. STRAUSS, H., OSTOW, M., and GREENSTEIN, L., *Diagnostic Electroencephalography*, New York, Grune and Stratton, 1952.
2. STRAUSS, H., OSTOW, M., GREENSTEIN, L., and LEWYN, S., Temporal slowing as a source of error on electroencephalographic localization, *J. Mt. Sinai Hosp.*, 22:306-315, 1955.
3. BUSSE, E.W., BARNES, R.H., SILVERMAN, A.J., SKY, G.M., THALER, M., and FROST, L.L., Studies of the process of aging: Factors that influence the psyche of elderly persons, *Am. J. Psychiat.*, 110:897-903, 1954.
4. SILVERMAN, A.J., BUSSE, E.W., and BARNES, R.H., Studies in the process of aging: Electroencephalographic findings in 400 elderly subjects, *Electroenceph. clin. Neurophysiol.*, 7:67-74, 1955.
5. GIBBS, F.A., and GIBBS, E.L., *Atlas of Electroencephalography*, 2nd. ed., Vol. 3, Reading, Mass., Addison-Wesley Press, 1964.
6. KENDEL, K., and KOUFEN, H., EEG-Veränderungen bei zerebralen Gefäßinsulten des Hirnstamms, *Dtsch. Z. Nervenheilk.*, 197:42-55, 1970.
7. MAYNARD, S.D., and HUGHES, J.R., A distinctive electrographic entity: Bursts of rhythmical temporal theta, *Clin. Electroenceph.*, 15:145-150, 1984.
8. LEBEL, M., and REIHER, J., Wicket spikes: A previously undescribed EEG pattern, *Electroenceph. clin. Neurophysiol.*, 41:548, 1976 (abstract).
9. VISSER, S.L., HOOIJER, C., JONKER, C., VAN TILBURG, W., and DERIJKE, W., Anterior temporal focal abnormalities in EEG in normal aged subjects, correlations with psychological and CT brain scan findings, *Electroenceph. clin. Neurophysiol.*, 66:1-7, 1987.
10. BRUENS, J.H., GASTAUT, H., and GROVE, G., Electroencephalographic study of the signs of chronic vascular insufficiency of the Sylvian

- region in aged people, *Electroenceph. clin. Neurophysiol.*, 12:283-295, 1960.
11. VAN DER DRIFT, J.H.A., and KOK, N.K.D., The EEG in cerebrovascular disorders in relation to pathology, In Remond, A., (editor-in-chief), *Handbook of Electroencephalography and Clinical Neurophysiology*, Vol. 14A, pp. 12-30, 47-64, Amsterdam, Elsevier, 1972.
  12. TORRES, F., FAORO, A., LOEWENSON, R., and JOHNSON, E., The electroencephalogram of elderly subjects revisited, *Electroenceph. clin. Neurophysiol.*, 56:391-398, 1983.
  13. ARENAS, A.M., BRENNER, R.P., and REYNOLDS, IIIrd, C.F., Temporal slowing in the elderly revisited, *Am. J. EEG Technol.*, 26:105-114, 1986.
  14. BUSSE, E.W., BARNES, R.H., FRIEDMANN, E.L., and KELTY, E.J., Psychological functioning of aged individuals with normal and abnormal electroencephalograms, I. A study of non-hospitalized community volunteers, *J. Nerv. Ment. Dis.*, 124:135-141, 1956.
  15. KOOI, K.A., GUVENER, A.M., TUPPER, C.J., and BAGCHI, B.K., Electroencephalographic patterns of the temporal region in normal adults, *Neurology*, 14:1029-1034, 1964.
  16. DRACHMAN, D.A., and HUGHES, J.R., Memory and the hippocampal complexes, III. Aging and temporal EEG abnormalities, *Neurology*, 21:1-14, 1971.
  17. ÖBRIST, W.D., EEG and intellectual function in the aged, *Proceed. Am. EEG Soc.*, Minneapolis, Sept., 1971.
  18. TUCKER, J.S., The electroencephalogram in brain stem vascular diseases, *Electroenceph. clin. Neurophysiol.*, 10:405-416, 1958.
  19. NIEDERMEYER, E., The electroencephalogram and vertebrobasilar artery insufficiency, *Neurology*, 13:412-422, 1963.
  20. ANDERSON, E.M., CARNEY, A.L., and PAGE, L., EEG in the vascular laboratory, In Carney, A.L., and Anderson, E.M., (eds.): *Diagnosis and Treatment of Brain Ischemia*, pp. 325-334, New York, Raven Press, 1981.
  21. NIEDERMEYER, E., LONG, D.M., HENDLER, N.H., CLARK, D.L., and DANIECKI, K., Chronic pain and mu rhythm, *Electroenceph. clin. Neurophysiol.*, 53:30P, 1982 (abstract).
  22. GIBBS, F.A., and GIBBS, E.L., Age factor in epilepsy: A summary and synthesis, *New Engl. J. Med.*, 269:1230-1236, 1963.
  23. TAKAHASHI, T., NIEDERMEYER, E., and KNOTT, J.R., The EEG in older and younger adult groups with convulsive disorder, *Epilepsia*, Amsterdam, 6:24-32, 1965.
  24. KOSHINO, Y., and NIEDERMEYER, E., The clinical significance of small sharp spikes in the electroencephalograms, *Clin. Electroenceph.*, 6:131-140, 1975.
  25. UCHIMURA, I., Über die Gefäßversorgung des Ammonshorns, *Z. ges. Neurol. Psychiat.*, 112:1-19, 1928.
  26. SCHROEDER, M., Die Lokalisation der Ammonshornschädigung im arteriellen Grenzgebiet, *Inaugural Dissert.*, Munich, 1962.
  27. VELHO-GRONEBERG, P., The EEG: An important (and often underrated) tool in the diagnosis of strokes, *Am J. EEG Technol.*, 26:213-224, 1986.