

Dysarthria in bilateral thalamic infarction

A case study

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Abstract. A patient suffering from bilateral thalamic infarction in the region supplied by the paramedian arteries sparing the internal capsules underwent acoustic analysis of sentence utterances. The results were compared with the findings obtained in parkinsonian subjects, in patients with upper motor neuron lesions, and in normal subjects. Acoustic measurements revealed increased pitch, monotonous speech, rough voice quality, and normal speech tempo concomitant with articulatory impreciseness in terms of incomplete closure productions. This constellation resembled parkinsonian dysarthria. Damage to the thalamic projection area of the pallidal efferents, therefore, seems to be the most probable cause of the patient's speech disorders. In parkinsonian subjects stereotactical lesions of this structure ameliorate rigor, but not akinesia. Thus, our patient's speech deficits, and by analogy the corresponding parkinsonian dysarthric disturbances, may be considered akinetic signs.

Key words: Thalamic dysarthria – Thalamic ischaemia – Acoustic analysis

Introduction

Thalamic disorders may give rise to speech and language deficits, as indicated by the verbal impairment following natural or surgical lesions and by the effects of electrical stimulation on linguistic abilities [10]. Whereas aphasic disturbances have been studied in some detail, few data are available so far concerning speech abnormalities due to thalamic dysfunction [26].

With respect to electrical stimulation two findings show some relevance of thalamic structures for speech motor control. First, Mateer [43] noted articulatory distortions resulting in prolongation of speech utterances during this procedure. Second, Guiot and coworkers [29] observed decreased voice volume and articulatory

inaccuracy concomitant with speech acceleration. So far no more detailed analyses are available.

The sequelae of stereotactic thalamic surgery, most often performed in parkinsonian patients, include slurred speech, decreased voice volume, whispering voice, rapid speech rate, and dysfluencies [11, 53, 55]. Some authors [41, 42] have maintained that the speech disturbances encountered as side effects of thalamotomy resemble dysarthria of Parkinson's disease (PD). However, Bell [6] could not find any relationship between pre- and postoperative speech deficits in PD subjects who underwent thalamic surgery and concluded that speech deterioration following surgical intervention results from lesions of the internal capsules. The observation of suprabulbar signs in postoperative dysarthrias and pathologic-anatomical data obtained in patients with postoperative speech deficits are in agreement with this suggestion [15, 32]. Indeed, Samra and coworkers [54] have reported persistent speech deviations following lesions restricted to the ventrolateral nuclei. However, all these subjects had suffered from speech disorders prior to surgery. Thus, natural progression of PD might have contributed to the aggravation of dysarthria and dysphonia months to years after surgical intervention.

The following speech deficits have been observed in natural thalamic lesions: slurred articulation [e.g., 12, 14, 22, 37, 39, 40, 57], characterized in one instance as undershooting of articulatory gestures [47], hypernasality [14, 47], decreased and fading voice volume [e.g., 8, 9, 25, 27, 39, 48], whispering [24], low- or high-pitched voice [37, 44, 45], hoarse voice quality [47], quivering voice [45], and monotonous, aprosodic speech [e.g., 7, 27, 28, 44]. Moreover, reduced verbal spontaneity and akinetic mutism, respectively [4, 13, 17, 19, 28, 33, 58], modification of speech tempo [19, 20, 38, 44, 47], and iterative phenomena like palilalia [46] were noted. However, no formal speech examinations and no parametric studies have been performed so far.

The patients reported with speech deficits due to natural thalamic lesions suffered – with a few exceptions – from a contra- or bilateral pyramidal syndrome as far

as one can judge from the clinical data provided. Since lacunar capsular infarcts can give rise to dysarthria [5, 21, 35], a contribution of capsular involvement to the observed speech motor dysfunctions must be considered in these subjects.

Further elucidation of thalamic speech deficits thus requires investigation of patients with lesions restricted to the thalamus. The present study was of a dysarthric patient suffering from bilateral thalamic infarction sparing the internal capsules. The patient's speech was submitted to acoustic analysis and, on the basis of these data, compared with spastic dysarthrics and parkinsonian subjects as well as normals.

Patients and methods

Case report

Three days after surgical intervention for total hip replacement the 64-year-old woman became somnolent. Apart from coxarthrosis, the past medical history was unremarkable. After she had regained alertness the neurological examination revealed complete gaze paresis upwards, incomplete gaze paresis downwards, impaired convergence, decreased voice volume concomitant with monotonous speech, dysphagia, and hypomimia. The patient presented with incomplete orientation in time and space and appeared to lack spontaneity. Voluntary innervation of the orofacial as well as the limb muscles was intact, tendon reflexes, plantar reflexes, sensory functions and coordination being normal. Otolaryngological investigation gave no indication of vocal fold paresis.

At the time of detailed neuropsychological assessment and speech evaluation, 7 months after disease onset, both vertical gaze paresis and the lack of spontaneity had improved. The remaining clinical findings were unchanged. Magnetic resonance imaging (MRI) showed bilateral postschaemic lesions in the anterior half of the thalamus (Fig. 1). On both sides the infarction extended from the medial to the ventrolateral and ventroanterior nuclei, sparing the internal capsules.

Recordings of somatosensory evoked potentials after electrical stimulation of the median and tibial nerves as well as brain-stem auditory-evoked potentials yielded normal latency and amplitude values. Electro-oculography showed no pathological nystagmus.

In spontaneous speech the patient's voice showed decreased volume, high pitch, and rough and breathy voice quality. Voice fading could frequently be observed. Her utterances sounded monotonous. Production of consonants was characterized by incomplete closures. The patient spoke at a normal rate.

Neuropsychological assessment included measurements of IQ, attentional abilities, so-called frontal lobe functions, and various declarative and procedural memory functions. In short, the patient displayed normal IQ and attention, but severe deficits in delayed recall of both verbal and visuospatial material. Procedural learning was preserved.

Formal language testing (Aachener Aphasic Test [34]) disclosed no aphasic deficits: The results of the Token Test were within the normal range. Spontaneous speech showed correct syntax and included no phonemic paraphasias, semantic paraphasias or word-finding difficulties. Repetition, confrontation naming, writing and comprehension of both auditory and written stimuli were intact.

Control groups

Twenty patients with idiopathic PD (14 men, 6 women; age 47–81 years), 12 patients with spastic dysarthria following uni- or bilateral lesions of the upper motor neuron due to closed head trauma or cerebrovascular accident (11 men, 1 woman; age 21–62 years), and 30 normal speakers (16 men, 14 women; age 19–64 years)

were recruited as controls. The acoustic data from these groups have been published in part elsewhere [2, 59].

Acoustic speech evaluation

Sentences of the form "Ich habe /geCVCe/ gehört" ("I have heard...") were used as speech material. By substituting the consonants /p/, /t/, /k/ for C and the long vowels /i/, /y/, /u/, /a/ for V the formula /geCVCe/ allowed the derivation of 12 target words: "gepipe", "gepype", "gepupe", "gepape", "getite", "getyte", "getute" etc. The resulting 12 test sentences, e.g. *Ich habe gepipe gehört*", with the syllable CV bearing the sentence accent, were presented twice by the examiner in randomized order and had to be repeated by the subject. Thus, 24 sentence utterances could be obtained. Since the target words represent regular phonological forms of standard German, the test sentences approximate natural speech demands.

Recordings were made in a sound-attenuated booth using high-quality equipment. Speech signals were digitized at a sampling rate of 20 kHz after low-pass filtering at 9 kHz and were fed into a LSI 11/73 for further processing (see [2, 59, 60]).

For each sentence utterance the speech wave envelope (sound pressure level (SPL) contour) was calculated in order to obtain parameters of articulatory accuracy (articulatory precision) and speech tempo. The rise-fall patterns of the SPL contours reflect the sequence of opening and closing gestures required for the production of the four syllables /be/, /ge/, /CV/, and /Ce/ of the test sentences. As an example, Fig. 2 shows the SPL contour of the test sentence "Ich habe gepipe gehört". The period between two successive intensity troughs was considered an index of syllable length. Fig. 2 indicates the so-defined durations of the syllables /be/, /ge/, /pi/, and /pe/ by vertical bars. As a measure of speech tempo the mean duration of these four syllables was determined for each test sentence and the median value across the 24 productions of a subject was computed (*mean syllable duration* = MSD).

Complete closure of the vocal tract as required for the production of voiceless stop consonants results in an interruption of sound emission from the mouth with a concomitant decrease of sound intensity to the level of tape noise. During incomplete closure due to "undershooting" of mandibular, labial, or lingual movements or due to weakness of the corresponding muscles, respectively, acoustic energy is still emitted from the mouth at the time of stop consonant production giving rise to increased SPL. Thus, the minimum amplitude of the intensity contour between two syllabic peaks, i.e. at the time points indicated by vertical bars in Fig. 2, was calculated (*sound intensity during closure* = IDC). This measure may be considered to reflect the degree of articulatory undershooting or of muscle weakness, respectively.

In addition, the fundamental frequency (F_0) contour of the vocalic part of each of the five syllables /ha/, /ge/, /CV/, /Ce/, and /hört/ of the sentence utterances were calculated. F_0 represents the frequency of vocal fold vibrations and provides, thus, an objective measure of pitch. For each subject the *mean fundamental frequency* of the 24 test sentence utterances and the *vocal jitter*, i.e. the cycle-to-cycle variation of F_0 , were computed, the latter being an index of vocal roughness [30]. Moreover, the *F₀ difference between the stressed (CV) and the unstressed syllable (Ce)* of the target words each provides a measure of speech monotony. In accordance with psychoacoustic standards F_0 was transferred into a logarithmic measure, i.e. semitones relative to an arbitrary value of 50 Hz, to account for inter-individual pitch differences. Finally, the *energy spectrum* of the vowel of the target word was computed. An increased drop in harmonic energy from the first to the second harmonic in the voice spectrum can be considered an acoustic correlate of perceived weak and breathy voice.

Results

The acoustic data substantiated the perceptual features of our patient's speech deficits. Her mean F_0 amounted

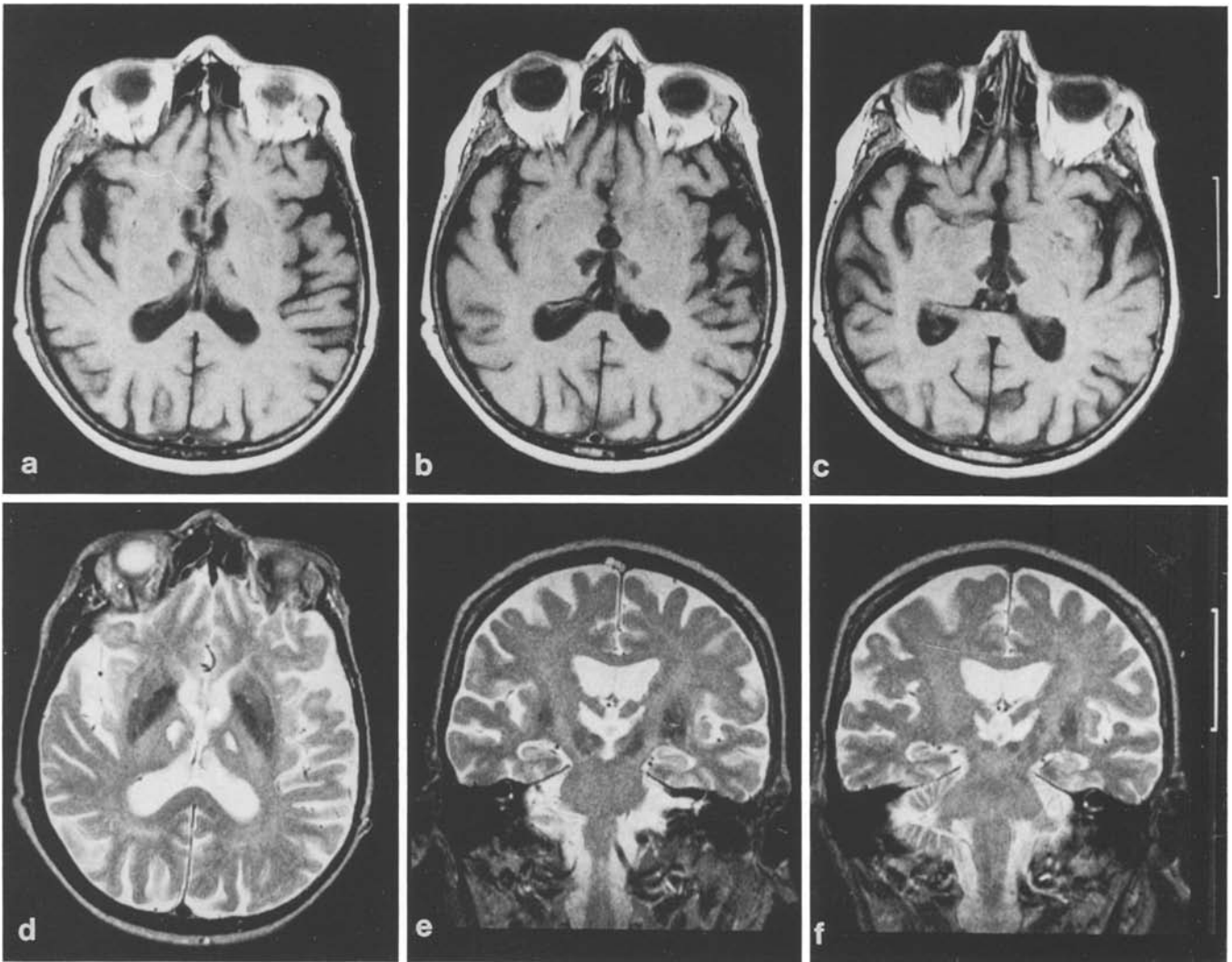


Fig. 1a-f. Magnetic resonance images showing bilateral ischaemic lesions extending from the medial parts of the thalamus to the ventrolateral and ventroanterior nuclei: **a-c** T1-weighted (TR/TE: 600/15 ms) axial, contiguous 4 mm slices; **d-f** T2-weighted axial (**d**) and coronal (**e-f**) scans (TR/TE: 2100/90 ms)

to 270 Hz, a value considerably above the normal range as reported in the literature [51] or as obtained in our group of healthy female controls (median: 197 Hz, range: 171–227 Hz). In addition, vocal jitter was markedly increased relative to the group of normals (0.42; normal median value: 0.19, normal range: 0.13–0.26), confirming rough voice quality in our patient. Furthermore, an overly large drop of harmonic energy from the first to the second harmonic in the voice spectrum could be noted (20 dB; normal median value: 11 dB, normal range: 6–16 dB), which may be considered a correlate of the patient's weak and breathy voice. The F_0 contours calculated over whole-sentence frames showed a reduction in stress-related F_0 variation, reflecting the monotonous quality of speech (0.6 semitones on average; normal me-

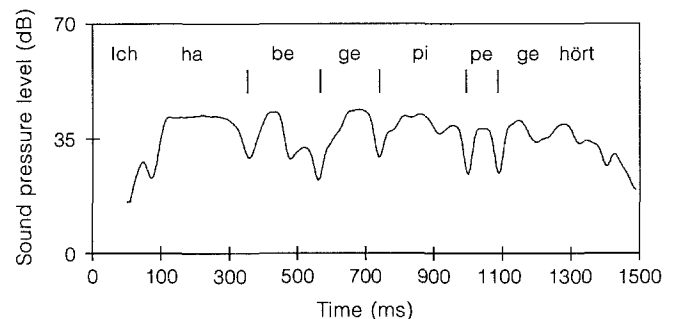


Fig. 2. Sound pressure level (SPL) contour of the test sentence “*Ich habe gepipe gehört*” spoken by the thalamic patient. The syllabic boundaries are indicated by vertical lines. Intensity during closure was determined as the sound intensity emitted during stop consonant production, i.e. as the SPL at the indicated syllable boundaries

dian value: 3.2 semitones, normal range: 1.8–4.8 semitones).

A fairly consistent increase of the radiated acoustic energy during stop closure could be noted across the 24 sentence productions. These findings indicate undershoot-

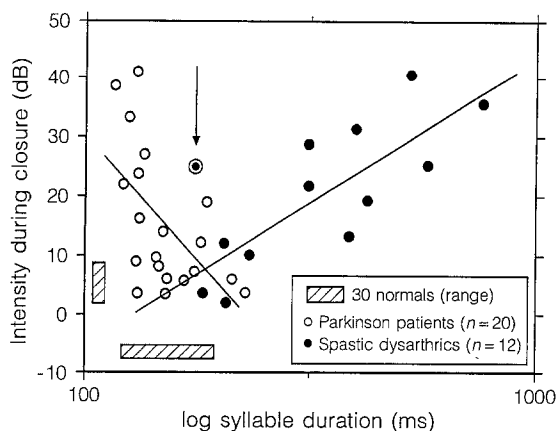


Fig. 3. Closure intensity (dB) and mean syllable duration (log ms) of 20 parkinsonian subjects (*open circles*), 12 spastic dysarthrics (*filled circles*), and the patient with bilateral thalamic infarction (*arrow*). Bars indicate the normal range

ing of articulatory gestures or weakness of the corresponding muscles, respectively. Despite her severe dysarthric impairments the patient showed normal speech rate. MSD amounted to 180ms, which is in the upper half of the normal range. Fig. 3 demonstrates to what extent the patient's articulatory performance resembled parkinsonian dysarthria rather than the speech of the upper motor neuron group. In the spastic dysarthrics there was a clear correlation between slowness and articulatory imprecision. In contrast, the parkinsonian patients as well as the thalamic subject had incomplete closures in the presence of normal or even reduced syllable durations.

Discussion

In a patient suffering from bilateral thalamic infarction acoustic measurements documented normal speech rate concomitant with articulatory impreciseness in terms of incomplete stop consonant closures, monotonous speech, high-pitched, breathy and weak voice, and rough voice quality.

Principally, speech deficits associated with thalamic lesions may be attributed to a dysfunction of the adjacent corticobulbar pathways. However, clinical, electrophysiological, and neuroradiological data gave no indications of an involvement of the internal capsules. Moreover, in patients with lesions of the upper motor neurons articulatory inaccuracy was associated with slowed speech whereas our patient showed normal speech tempo. Thus, a significant contribution of corticobulbar disorders to our patient's dysarthria can be ruled out.

Both the striato- and cerebellocortical projections incorporate thalamic structures. Conceivably, interruption of these motor loops at the thalamic level might impair speech functions. First, Schuster [56] has observed speech disorders reminiscent of cerebellar dysarthria in a patient with thalamic softening. Furthermore, von Cramon [16] has reported ataxic dysarthria in a subject with bilateral disruption of the cerebellothalamic pathways at the

mesodiencephalic level. Second, parkinsonian speech signs have been noted after thalamic surgery [41, 42].

Normal speech rate concomitant with incomplete stop consonant closures has been documented in parkinsonian dysarthria [2]. In contrast, acoustic investigations of dysarthric deficits in cerebellar atrophy and Friedreich's ataxia have revealed slowed speech in terms of prolonged syllable and sentence durations [23, 36]. Thus, the normal speech rate of our patient argues against a dysarthria of predominantly cerebellar origin. Moreover, decreased voice volume and high-pitched voice are more typical for PD than for cerebellar dysarthria [1, 3]. Thus, the parkinsonian-like perceptual and acoustic features of our patient's dysarthria indicate damage to the thalamic region of the striatocortical loop. In parkinsonian patients stereotactic lesions of the anterior ventrolateral thalamic region abolish rigidity, but not akinesia of the speech motor system [31, 32, 49, 50]. The speech disturbances of our patient, therefore, should reflect akinetic dysarthric signs rather than deficits due to rigidity.

Parkinsonian bradykinesia, i.e. slowing of movement, may affect the speech motor system as well as the limbs (see [2]). Conceivably, PD subjects manage normal syllabic rate in the presence of bradykinesia by undershooting of articulatory gestures, i.e. by reduced movement amplitudes. Assuming a striatocortical dysfunction, the articulatory disturbances of our patient might represent, as suggested for PD dysarthria, a tradeoff between speech tempo and movement excursion.

The neuroradiological data of the present study are compatible with the suggestion of a striatocortical dysfunction. At both sides the lesions obviously involved the ventrolateral and parts of the ventroanterior nuclei representing the major projection area of the pallidal efferents at the thalamic level. Moreover, the distribution of the ischaemic lesions indicates infarction in the region supplied by the paramedian thalamic arteries. In the anterior direction this area may vary considerably, depending upon the size of the polar thalamic artery [18]. Principally, however, ischaemia within the region of blood supply of the paramedian thalamic arteries can extend to the anterior ventrolateral and ventroanterior nuclei. Often the paramedian arteries of both sides arise from a common branch of the basilar communicating artery, which may explain the bilateral distribution of the infarction [52].

Speech deficits similar to those reported here have also been observed in subjects with unilateral left-sided thalamic haemorrhage [4]. Thus, bilateral damage does not seem to be a prerequisite for the speech impairments observed in our patient. Conceivably, bilateral lesions result in more severe and more enduring deficits. It is well known that the risks of persistent speech impairments are higher following bilateral than after unilateral thalamotomy [53]. Moreover, the transient character of the speech abnormalities after unilateral thalamic haemorrhages [4] supports this suggestion.

In summary, our results show that thalamic lesions may result in a parkinsonian-like dysarthria probably representing akinetic signs due to damage to the projection area of the pallidal efferents.

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References

- Ackermann H, Ziegler W (1989) Die Dysarthrophonie des Parkinson-Syndroms. *Fortschr Neurol Psychiatr* 57:149–160
- Ackermann H, Ziegler W (1991) Articulatory deficits in Parkinsonian dysarthria. An acoustic analysis. *J Neurol Neurosurg Psychiatry* 54:1093–1098
- Ackermann H, Ziegler W (1992) Die zerebelläre Dysarthrie. Eine Literaturübersicht. *Fortschr Neurol Psychiatr* 60:28–40
- Alexander MP, LoVerme SR (1980) Aphasia after left hemispheric intracerebral hemorrhage. *Neurology* 30:1193–1202
- Alexander MP, Naeser MA, Palumbo CL (1987) Correlations of subcortical CT lesion sites and aphasia profiles. *Brain* 110:961–991
- Bell DS (1968) Speech functions of the thalamus inferred from the effects of thalamotomy. *Brain* 91:619–638
- Biller J, Mercht M, Emanuele MA (1984) Nonhemorrhagic infarction of the thalamus. *Neurology* 34:1269–1270
- Bogousslavsky J, Miklossy J, Deruaz JP, Regli R, Assal G (1986) Unilateral left paramedian infarction of thalamus and midbrain. A clinico-pathological study. *J Neurol Neurosurg Psychiatry* 49:686–694
- Bruyn RPM (1989) Thalamic aphasia. A conceptual critique. *J Neurol* 236:21–25
- Buren JM van (1975) The question of thalamic participation in speech mechanisms. *Brain Lang* 2:31–44
- Canter GJ, Lancker DR van (1985) Disturbances of the temporal organization of speech following bilateral thalamic surgery in a patient with Parkinson's disease. *J Commun Disord* 18:329–349
- Cappa SF, Vignolo LA (1979) "Transcortical" features of aphasia following left thalamic hemorrhage. *Cortex* 15:121–129
- Castaigne P, Lhermitte F, Buge A, Escourolle R, Hauw JJ, Lyon-Caen O (1981) Paramedian thalamic and midbrain infarcts. Clinical and neuropathological study. *Ann Neurol* 10:127–148
- Ciemins VA (1970) Localized thalamic hemorrhage. A cause of aphasia. *Neurology* 20:776–782
- Cooper IS (1961) Parkinsonism. Its medical and surgical therapy. Thomas, Springfield, Ill
- Cramon D von (1981) Bilateral cerebellar dysfunctions in a unilateral meso-diencephalic lesion. *J Neurol Neurosurg Psychiatry* 44:361–363
- Cramon D von, Kühnlein J, Wolfram A (1981) Die thalamische Demenz. *Fortschr Neurol Psychiatr* 49:129–135
- Cramon DY von, Hebel N, Schuri U (1985) A contribution to the anatomical basis of thalamic amnesia. *Brain* 108:993–1008
- Davous P, Bianco C, Duval-Lota AM, De Recondo J, Vedrenne C, Rondot P (1984) Aphasie par infarctus thalamique paramédian gauche. Observation anatomo-clinique. *Rev Neurol (Paris)* 140:711–719
- Elghozi D, Strube E, Signoret JL, Cambier J, Lhermitte F (1978) Quasi-aphasie lors de lésions du thalamus. Relations du trouble du langage et de l'activation électrique de l'hémisphère gauche. *Rev Neurol (Paris)* 134:557–573
- Fisher CM (1979) Capsular infarcts. The underlying vascular lesions. *Arch Neurol* 36:65–73
- Friedman JH (1985) Syndrome of diffuse encephalopathy due to nondominant thalamic infarction. *Neurology* 35:1524–1526
- Gentil M (1990) Dysarthria in Friedreich disease. *Brain Lang* 38:438–448
- Gentilini M, De Renzi E, Crisi G (1987) Bilateral paramedian thalamic artery infarcts. Report of eight cases. *J Neurol Neurosurg Psychiatry* 50:900–909
- Gorelick PB, Hier DB, Benevento L, Levitt S, Tan W (1984) Aphasia after left thalamic infarction. *Arch Neurol* 41:1296–1298
- Graff-Radford NR, Damasio AR (1984) Disturbances of speech and language associated with thalamic dysfunction. *Semin Neurol* 4:162–168
- Graff-Radford NR, Eslinger PJ, Damasio AR, Yamada T (1984) Nonhemorrhagic infarction of the thalamus. Behavioral, anatomic, and physiologic correlates. *Neurology* 34:14–23
- Guberman A, Stuss D (1983) The syndrome of bilateral paramedian thalamic infarction. *Neurology* 33:540–546
- Guiot G, Hertzog E, Rondot P, Molina P (1961) Arrest or acceleration of speech evoked by thalamic stimulation in the course of stereotaxic procedures for Parkinsonism. *Brain* 84:363–379
- Hartmann E, Cramon D von (1984) Acoustic measurement of voice quality in central dysphonia. *J Commun Disord* 17:425–440
- Hassler R (1966) Thalamic regulation of muscle tone and the speed of movements. In: Purpura DP, Yahr MD (eds) *The thalamus*. Columbia University Press, New York London, pp 419–436
- Hassler R, Mundinger F, Riechert T (1979) Stereotaxis in Parkinson syndrome. Clinical-anatomic contributions to its pathophysiology. Springer, Berlin Heidelberg New York
- Horenstein S, Chung H, Brenner S (1978) Aphasia in two verified cases of left thalamic hemorrhage. *Trans Am Neurol Assoc* 102:193–198
- Huber W, Poeck K, Weniger D, Willmes K (1983) Aachener Aphasia Test. Hogrefe, Göttingen
- Ichikawa K, Kageyama Y (1991) Clinical anatomic study of pure dysarthria. *Stroke* 22:809–812
- Kent RD, Netsell R, Abbs JH (1979) Acoustic characteristics of dysarthria associated with cerebellar disease. *J Speech Hear Res* 22:627–648
- Kessler C, Kummer R von, Herold S (1982) Zur Ätiologie der doppelseitigen, symmetrischen Thalamusläsion. *Nervenarzt* 53:406–410
- Kobari M, Ishihara N, Yunoki K (1987) Bilateral thalamic infarction associated with selective downward gaze paralysis. *Eur Neurol* 26:246–251
- Kosten TR, Rapoport AM (1982) Left thalamic hemorrhage and aphasia. *Conn Med* 46:633–635
- Kritchevsky M, Graff-Radford NR, Damasio AR (1987) Normal memory after damage to medial thalamus. *Arch Neurol* 44:959–962
- Laitinen L (1966) Thalamic targets in the stereotaxic treatment of Parkinson's disease. *J Neurosurg* 24:82–85
- Markham CH, Rand RW (1963) Stereotactic surgery in Parkinson's disease. *Arch Neurol* 8:621–631
- Mateer C (1978) Asymmetric effects of thalamic stimulation on rate of speech. *Neuropsychologia* 16:497–499
- Mazaux JM, Orgogozo JM (1982) Etude analytique et quantitative des troubles du langage par lésion du thalamus gauche. L'aphasie thalamique. *Cortex* 18:403–416
- Mazaux JM, Orgogozo JM, Henry P, Loiseau P (1979) Troubles du langage au cours des lésions thalamiques. *Rev Neurol (Paris)* 135:59–64
- McFarling D, Rothi LJ, Heilman KM (1982) Transcortical aphasia from ischaemic infarcts of the thalamus. A report of two cases. *J Neurol Neurosurg Psychiatry* 45:107–112
- Meissner I, Sapir S, Kokmen E, Stein SD (1987) The paramedian diencephalic syndrome. A dynamic phenomenon. *Stroke* 18:380–385
- Mohr JP, Watters WC, Duncan GW (1975) Thalamic hemorrhage and aphasia. *Brain Lang* 2:3–17
- Narabayashi H (1990) Surgical treatment in the levodopa era. In: Stern GM (ed) *Parkinson's disease*. Chapman & Hall, London, pp 597–646
- Narabayashi H (1991) Role of stereotaxic surgery in treatment of Parkinson's disease. In: Nagatsu T, Narabayashi H, Yos-

- hida M (eds) Parkinson's disease. From clinical aspects to molecular basis. Springer, Wien New York, pp 187–199
51. Pegoraro-Krook MI (1988) Speaking fundamental frequency characteristics of normal Swedish subjects obtained by glottal frequency analysis. *Folia Phoniatr* 40:82–90
 52. Percheron G (1976) Les artères du thalamus humain. *Rev Neurol (Paris)* 132:297–307, 309–324
 53. Riklan M, Levita E (1969) Subcortical correlates of human behavior. A psychological study of thalamic and basal ganglia surgery. Williams & Wilkins, Baltimore, pp 125–159
 54. Samra K, Riklan M, Levita E, Zimmerman J, Waltz JM, Bergmann L, Cooper IS (1969) Language and speech correlates of anatomically verified lesions in thalamic surgery for Parkinsonism. *J Speech Hear Res* 12:510–540
 55. Schaltenbrand G (1975) The effects on speech and language of stereotactical stimulation in thalamus and corpus callosum. *Brain Lang* 2:70–77
 56. Schuster P (1937) Beiträge zur Pathologie des Thalamus opticus. IV. Mitteilung. *Arch Psychiatr Nervenkr* 106:201–233
 57. Walshe TM, Davis KR, Miller Fisher C (1977) Thalamic hemorrhage. A computed tomographic-clinical correlation. *Neurology* 27:217–222
 58. Watson RT, Valenstein E, Heilman KM (1981) Thalamic neglect. Possible role of the medial thalamus and nucleus reticularis in behavior. *Arch Neurol* 38:501–506
 59. Ziegler W, Cramon D von (1986) Spastic dysarthria after acquired brain injury. An acoustic study. *Br J Disord Commun* 21:173–187
 60. Ziegler W, Hoole P, Hartmann E, Cramon D von (1988) Accelerated speech in dysarthria after acquired brain injury. Acoustic correlates. *Br J Disord Commun* 23:215–228