

Evaluation of Dementia in Subcortical Arteriosclerotic Encephalopathy (Binswanger's Disease)

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Summary. Sixteen patients with a clinical diagnosis of subcortical arteriosclerotic encephalopathy (Binswanger's disease) were studied. Mild or moderate dementia was found in 14 cases, with characteristic disturbances of concentration, and psychic retardation with apathy. Neuropsychological studies revealed that signs of diffuse lesions in the frontal lobes dominated. Impairment of memory was significant in all cases. Although intellectual impairment in subcortical arteriosclerotic encephalopathy resembled subcortical dementia, some significant differences were found.

Key words: Subcortical arteriosclerotic encephalopathy – Dementia – Subcortical dementia – Neuropsychological evaluation

Introduction

Subcortical arteriosclerotic encephalopathy (SAE), or Binswanger's disease, is a clinicopathological entity with quite well defined neuropathological features. Its pathogenesis remains unclear [14] and the diagnostic clinical criteria are still not clearly established [6, 14].

The neuropathology of SAE consists of symmetrical areas of demyelination in cerebral white matter (with subcortical U fibres spared), loss of oligodendroglia and astrocytic gliosis. Lacunar infarcts are usually present (in subcortical grey matter as well) and cortical infarcts frequently co-exist. Thickened subcortical arteries and arterioles show lipohyalinosis and fibrosis [7, 12, 16, 21, 22]. Arteriosclerosis is considered to be the cause of ischaemia and hypoxia, which are responsible for incomplete infarctions in the white matter. However, the exact pathomechanism of SAE remains unclear [6, 14].

In the past, the diagnosis of SAE was based on neuropathological data, and the disease appeared to be uncommon. When in the late 1970s computed tomography (CT) became widely available, the clinical diagnosis of

SAE became possible. CT in SAE shows diffuse areas of white matter low attenuation (lucency) [16, 19–21, 23]. Both CT and magnetic imaging (MRI) studies have revealed that white matter pathology is relatively common among the elderly [19, 24, 25, 27]. Radiological features of white matter pathology have been found also in patients without dementia or other signs of SAE [24, 25]. The morphological basis of these changes and their relation to SAE remains unknown. On the other hand, during the last 10 years, there have been several case reports [4, 16, 19, 21, 27] in which clinical, radiological and neuropathological correlations were established. Analysis of these reports shows that the clinical diagnosis of SAE is possible after careful evaluation of clinical and radiological data [21].

Dementia is the most prominent clinical feature of SAE [6]. Abulia and mental slowing are well pronounced. There have been few reports describing the pattern of the dementia. According to Loizou et al. [20] "no specific features could be ascertained, the dementia being global and affecting verbal, visual and cognitive functions". Kinkel et al. [19] stated that "the dementia associated with SAE is predominantly of the subcortical type". A similar opinion was expressed by Gupta et al. [17]. The neuropsychological studies of Derix et al. [13] showed the resemblance of intellectual impairment in SAE to the dementia of subcortical type.

The term "subcortical dementia" was proposed in 1974 by Albert et al. [1] to denote the picture of intellectual impairment in patients with progressive supranuclear palsy. The characteristic features of subcortical dementia were forgetfulness, slowness of thought process or bradyphrenia, disturbance of mood and personality, reduced ability to manipulate acquired knowledge [1]. The same symptoms were subsequently attributed to other disorders associated with predominantly subcortical pathology, such as Parkinson's disease and Huntington's disease [5, 9, 10, 15].

Views on the nature of subcortical dementia are still debated after more than 10 years of study and discussion. While some authors, e.g. Cummings [9], consider that there exist sufficient data to sustain the concept that

subcortical dementia is different from cortical dementia, others, e.g. Whitehouse [26] and Brown and Marsden [5], consider that the concept is not well founded. Freeman and Albert [15] pointed out the significance of anatomical and functional connections between subcortical structures and the cortex of the frontal lobes and proposed the terms “fronto-temporal or frontal system dementia”, since “they more accurately reflect the concept previously labelled subcortical dementia”. The aim of this study was the evaluation of dementia in SAE and the analysis of its relation to subcortical dementia.

Patients and Methods

Patient Selection

The group studied consisted of patients who, after referral from several neurological departments and outpatient clinics, underwent CT examination in the Department of Diagnostic Imaging. Patients with diffuse white matter low attenuation on CT scans were selected. Subjects with multiple sclerosis, brain tumour, previous brain radiotherapy and/or chemotherapy (methotrexate), or a history of severe head trauma, were excluded. If other clinical data obtained from the referring unit suggested the clinical diagnosis of SAE, the patient was admitted to the Department of Neurology, Medical Academy of Łódź, for at least 2 weeks. Informed consent of the patient and his/her family was obtained.

Clinical Diagnostic Criteria for SAE

The following criteria for diagnosis of SAE were used:

1. Presence of white matter low attenuation on CT scans (obligatory criterion)
- At least three of the following:
 2. Hypertension
 3. History of stroke (or transient ischaemic attack)
 4. Focal neurological signs
 5. Dementia or marked impairment of intellectual function which did not achieve the level of dementia.

Clinical Evaluation

Clinical evaluation included medical history using available records and interview of a close informant, detailed general and neu-

rological examination. The clinical laboratory tests included: erythrocyte sedimentation rate, red and white blood counts, haemoglobin, serum electrolytes, serum glucose, proteins, lipids and lipoproteins, kidney, liver and thyroid tests, VDRL. Chest radiography, electrocardiography (ECG) and electroencephalography (EEG) were performed.

Analysis of CT Scans

A Siemens DR2 scanner was used. Intensity of white matter low attenuation, cortical atrophy, ventricular dilatation (expressed as Evan's ratio) and subcortical and cortical focal ischaemic lesions were evaluated on CT scans.

The intensity of white matter lucency was expressed as the difference (in Hounsfield Units (HU) between densities of grey and white matter (according to Zeumer et al. [27] with modifications): I, 8–9 HU; II, 10–12 HU; III, difference > 13 HU.

The following grading of cortical atrophy was used (according to Zeumer et al. [27]): I, prominent Sylvian fissure and slightly visible cortical sulci; II, Sylvian fissure dilated and cortical sulci < 3 mm in width; III, numerous cortical sulci > 3 mm.

Diagnosis and Evaluation of Dementia

All patients underwent detailed psychiatric and neuropsychological examination. Dementia was diagnosed according to DSM III-R [2]. The diagnosis was based on clinical examination and information obtained from a person well acquainted with the patient. The severity of dementia was evaluated as mild (9 patients) or moderate (5 patients) according to DSM III-R [2]. There were no cases of severe dementia in the group. In 2 patients intellectual impairment did not achieve the level of dementia. In all cases the Wechsler-Bellevue Intelligence Scale, Benton Visual Retention Test and Luria's tests for detection of neuropsychological deficits [8] were performed.

Results

Patients

The group studied consisted of 11 male and 5 female patients. Their age ranged from 56 to 85 years (mean 67). The duration of illness varied from 6 to 24 months in most cases, being notably longer in 4 cases. Three patients died and in 2 cases autopsy was performed. Neuropathological examination confirmed the clinical diag-

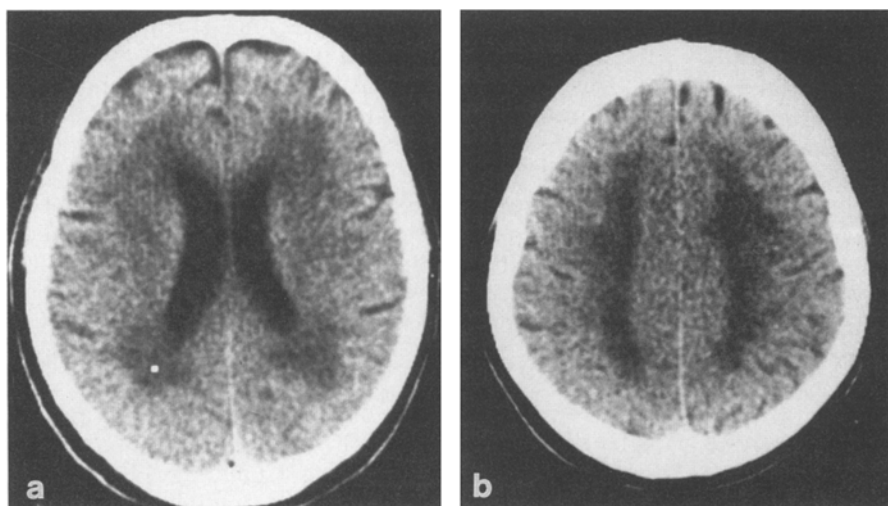
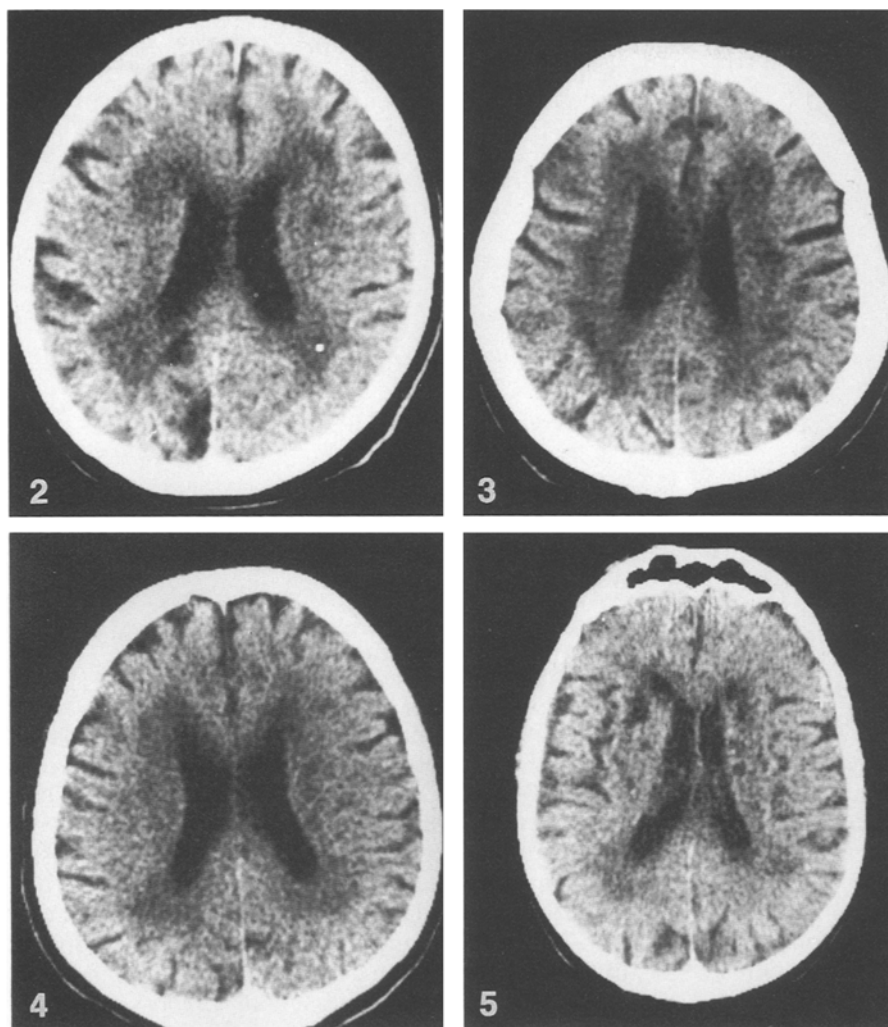


Fig. 1a, b. Case 6. CT scan: moderately diminished attenuation of white matter, moderate cortical atrophy, dilated ventricles



Figs. 2–5. **2** Case 2. CT scan: moderate hypodensities of white matter adjacent to horns of lateral ventricles, cortical infarct in left occipital lobe, subcortical lesion in the right frontal lobe, moderate cortical atrophy and ventricular dilatation. **3** Case 3. CT scan: moderate to severe white matter low attenuation, severe cortical atrophy, dilated ventricles. **4** Case 4. CT scan: moderate white matter low attenuation, severe cortical atrophy, dilated lateral ventricles. **5** Case 5. CT scan: mild periventricular white matter low attenuation, bilateral subcortical focal ischaemic lesions, severe cortical atrophy and ventricular dilatation

Table 1. Characteristics of patients

Patient no.	Sex	Age (years)	Duration of illness	Blood pressure (mm Hg)	Diabetes	Number of strokes	Focal signs (side)	Pseudobulbar palsy	Pattern of the disease	Incontinence	Severity of dementia (DSM III-R)
1	f	56	4	140/80		2	Right	+	S, P, I		Moderate
2	m	62	12	185/115	+	8	Bilateral	+	S, P	+	Moderate
3	m	61	1.5	170/115				Slight dysarthria	S, P		Mild
4	m	62	0.5	180/110		1 (TIA) ^a	Right		S, P		Non-demented
5	f	72	1.5	170/110		1	Right		S, P, I		Non-demented
6	m	73	1.5	130/90			Left		S, P, I		Moderate
7	f	79	0.5	120/80	+		Bilateral		S, P		Moderate
8	m	64	1.5	210/140		1	Left	Dysarthria	S, P, I	+	Mild
9	m	70	1.5	170/110	+	1	Bilateral	+	S, P, I		Mild
10	m	60	1	200/105		1	Right		S, P, I		Mild
11	m	67	5	160/120	+	4	Bilateral	Dysarthria	S, P, I	+	Mild
12	m	64	2	200/130	+	2	Bilateral	+	S, P, I	+	Mild
13	m	62	12	190/120		2	Right	+	S, P, I	+	Mild
14	f	85	2	170/110			Right		S, P, I		Mild
15	m	74	1.5	125/90			Left		S, P		Mild
16	f	63	13	170/100		1	Bilateral		S, P, I		Moderate

S, Periods of stepwise deterioration; P, plateau periods; I, periods of improvement

^a Transient ischaemic attack

Table 2. Computed tomography findings

Patient	White matter low attenuation		Cortical atrophy	Focal lesions		Evan's ratio
	intensity	localization		subcortical	cortical	
1	III	F, P	I	-	-	0.29
2	III	F, P	II	lF, rF	-	0.35
3	II	P, F	III	lF	-	0.31
4	II	P	II	-	lT	0.27
5	II	F, P	II	-	rO	0.33
6	II	F, P	II	-	-	0.36
7	I	P	III	lT	lT	0.31
8	I	P	III	lF, rF, lP, rP	-	0.34
9	II/III	F, P	III	lP, rP	-	0.35
10	II	F, P	II/III	lP, rP, rF	lP, lO, rF	0.33
11	II	P > F	II	lT, lP	-	0.31
12	II/III	F, P	II	lP	lT, rO	0.35
13	III	F, P	III	rF, rP-O	lP	0.33
14	II	F, P	III	-	lP	0.35
15	III	P > F	III	lT, lO, rT, rP	-	0.31
16	II/III	F, P, O	III	-	-	0.37

F, Frontal lobe; O, occipital lobe; P, parietal lobe; T, temporal lobe; l, left; r, right. For other explanations see text

nosis. In 1 of the autopsied cases, cerebral amyloid angiopathy was partly responsible for the development of SAE [4]. The patients' data are summarized in Table 1. A detailed description of 3 cases is presented below.

CT Scans

Mild low attenuation of the white matter was found in 2 cases and moderate and/or severe in the other patients. The white matter of the frontal and parietal lobes was affected predominantly. Cortical atrophy and enlargement of the lateral ventricles were present in almost all cases. In 3 cases single and in 7 cases numerous subcortical infarcts were present. In 5 patients they were bilateral. Cortical infarcts were found in 7 cases and they were mostly single. Details of CT findings are presented in Table 2.

Neurological status

Weakness of limbs and gait disturbances were not severe enough to reduce significantly patients' ability to move around and undertake self-care. Patients with pseudobulbar palsy were able to swallow food (except patient 1 in the terminal stage). Dysarthria occurred often as an isolated symptom and, if it co-existed with dysphagia, the latter was less pronounced.

Psychiatric and Neuropsychological Evaluation

Memory disturbance was the most prominent feature in non-demented and mildly demented patients with SAE. Recognition of the intellectual decline was preserved for a relatively long period of time. This is consistent with the opinion that preserved insight concerning mental deficiency is a differentiating feature between vascular dementia and the dementia of Alzheimer type.

Moderately demented patients showed marked apathy, withdrawal from former activities and loss of interest. The loss of spontaneity was remarkable, but under stimulation they were able to perform basic activities connected with self-care or even more complicated tasks, such as simple car repair. Families often complained that the patient became lazy and would not do things he or she was capable of doing. The patients' mood was that of indifference; recognition of illness was not accompanied by the adequate emotional reaction. The loss of activity and emotional blunting were the features which were responsible for the change of personality of the patient. Thus, the report of "well-preserved personality" in vascular dementia [18] did not apply to our patients with SAE.

One of the patients (no. 6), besides being moderately demented, showed marked character changes. He had frequent outbursts of temper; occasionally he refused steadfastly to cooperate during psychological examination. Early in the course of his illness temporal lobe epilepsy developed.

The comparison of the behaviour of demented SAE patients and patients with Alzheimer's disease during their stay in the hospital showed a remarkable difference. While the latter often wandered around losing their way to bed, the former tended to spend the whole time in their room, mostly in bed.

Difficulties in solving both concrete and abstract problems were found in mildly and, more pronounced, in moderately demented patients. The ability to shift from one strategy to another was impaired. The patients perseverated in their mistakes; their thinking was stereotyped. They were often unable to overcome an established response set. Speed of responding was low or fluctuating. Verbal behaviour of the patients was related to their disturbances of thought processes: impoverishment of vocabulary, incoherent statements, perseverations,

Table 3. The results of neuropsychological evaluation

Patient	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16
Verbal IQ	-	91	113	96	90	-	-	100	-	102	120	106	98	74	75	89
Performance IQ	-	92	112	103	97	-	-	97	-	82	110	94	93	89	89	89
Attention and concentration	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+	+
Memory	+	+	+	+	+	0	+	+	+	+	+	+	+	+	+	+
Speech				+	+	0	+	+	+	+	+					
Reading						0	+			+						+
Writing						0	+		+	+						+
Arithmetic	+	+			+	0	+	+	+	+						+
Praxis	+	+		+	+	+	+	+	+	+	+	+	+		+	+
Visuoconstruction		+		+	+	0	+		+	+	+				+	
Visuoperception						0										
Thinking	+	+	+				+		+	+	+	+	+	+	+	+

+, Impaired function; 0, patient refused to cooperate

bizarre speech were noted. The observed visuoconstructional deficits were mainly the result of impaired concentration. Mental and motor retardation were most prominent in all cases. Besides the disturbances of mental functions, apractic disturbances were also observed. This particularly concerned alternating movements and repetitive actions (Table 3).

Case Reports

Case 8

An industry manager came to medical attention aged 55 years because of severe hypertension (210/140 mmHg). He retired at the age of 60 years. Discrete gait disturbances were noticed by his wife 2 years later. He began to have difficulty in recognizing characters from TV serials. At age 63 chest pains developed. The antihypertensive treatment was modified and nitrates were administered. It is probable that he took an overdose of the latter. Three days later, he gradually became apathetic and confused. Blood pressure was found to be 120/80 mmHg. Slight left hemiparesis was present. After the next 3 days he improved; apathy and lack of spontaneity were striking, however. Slowing of performance and memory was prominent. He was afraid to go out of the house. A few episodes of incontinence occurred.

One year after the stroke the patient was admitted to hospitals. The blood pressure was 150/105 mmHg. There was bilateral palatal weakness, more pronounced on the left side. He was dysarthric but had no difficulty swallowing. Palmomental reflexes were present bilaterally. Mild weakness of both lower limbs with exaggerated left knee and ankle reflexes were found. The left plantar response was extensor. The EEG showed bilateral slight theta activity.

Apathy and psychomotor retardation were marked. He spoke with a soft voice. Speed of performance during examination fluctuated. Verbal and visual memory were markedly impaired, he perseverated with his errors frequently during memory testing. There was slight dys-

praxia and difficulty in word-finding occurred occasionally. His IQ was 20% reduced when compared with his premorbid intelligence.

Case 10

A 59-year-old engineer was admitted to the regional hospital because of sudden development of mild aphasia and confusion. He was unable to recognize his wife, daughter and attending physician. He was disorientated in time and place. He could not feed and dress himself. Slight right limb weakness with hyper-reflexia was found. Blood pressure was 200/105 mmHg. He improved gradually after some days, but, as his wife stated, he was a completely different man. He was very inert. The speed of response was low and there was a lack of facial expression. At home he spent time in bed or staring at the TV. He was afraid to go out of the house without his wife.

CT 3 weeks later showed white matter low attenuation around the bodies, anterior and posterior horns of the lateral ventricles and in the centrum semiovale. There was a small infarct in the white matter of the right frontal lobe and a larger one in the cortex of the left occipital lobe. There was marked cortical atrophy and moderate ventricular dilatation.

Eight weeks from the onset of his illness the patient was admitted to the Department of Neurology, Medical Academy of Łódź. He gave his name, date of birth, but not his age. He was orientated in place but not in time. He could find his way to the toilet and TV room; however, he seldom left his room spontaneously. He was able to wash, shave and dress himself, but he had to be reminded about it many times.

Partial right homonymous hemianopia limited to the periphery of the visual fields, bilateral palmomental reflex and extensor right plantar response were present. He was not incontinent. His blood pressure was 150/100 mmHg.

He was very apathetic but cooperative during neuropsychological examination. The speed of responses fluctuated markedly when observed on subsequent days. His ability to sustain attention was significantly reduced.

Visual and verbal memory was impaired. His conversational speech was reduced to short, adequate answers; occasionally there was word-finding difficulty. He had mild dyslexia and dysgraphia. On visuoperceptual tasks he performed well in contrast to his performance on visuocstructional tasks. His level of intellectual performance was reduced (14%) when compared with the estimated premorbid level.

EEG showed symmetrical theta activity. Digital subtraction angiography (DSA) did not show any abnormality in the cerebral arteries.

During the next 3 years dementia progressed, although transitory priods of improvement were observed. Neurological status was stable and no strokes were noted. Prominent apathy and lack of initiative were interpreted by the psychiatrist as signs of depression, and mianserin was administered without any beneficial effect.

Case 16

A female schoolteacher came to medical attention at the age of 41 years when she suffered an acute episode of left limb weakness from which she fully recovered. She retired prematurely at the age of 51 years because of her reduced efficiency. In the same year she was admitted to a psychiatric hospital because of depression. She was treated with a tricyclic antidepressant with some initial effect only. Mild dementia was diagnosed. She was followed up by a psychiatrist as an outpatient. Her mood was often depressed, but she never improved significantly on antidepressants. The intellectual deterioration progressed gradually over the course of the next 12 years.

At the age of 63 she was housebound. She was unable to feed, wash or dress herself, but she was continent. Transitory episodes of disorientation in time and place were observed. Slight dysphasia and gait disturbances developed. CT showed severe symmetrical white matter hypodensities in the frontal, parietal and occipital lobes, moderate cortical atrophy and ventricular dilatation but no focal ischaemic changes.

During her stay in the Department of Neurology, she left her room sporadically. Her appearance was untidy. Blood pressure was 170/100 mmHg. Palmomental reflexes were present bilaterally. Tendon reflexes in upper and lower limbs were exaggerated symmetrically and equivocal pyramidal signs on both sides were observed. The gait was apractic.

The EEG showed widespread bilateral theta and delta slowing. DSA revealed occlusion of the left internal carotid artery.

She cooperated poorly during neuropsychological testing. She was euphoric and she dissimulated occasionally. Difficulty in sustaining attention was prominent. Her conversational speech was restricted and perseverations were observed. Verbal and visual memory was seriously impaired; the Benton test could not be reliably assessed because of perseverations of errors. Dyspraxia, dysphasia and dysgraphia were present. She was unable to calculate above the threshold of 10. Her IQ could not be reliably assessed.

Discussion

The clinical diagnosis of SAE in the studied group was based on the CT findings, the presence of intellectual impairment and focal neurological signs and other data suggesting a vascular aetiology. Such diagnostic criteria make the diagnosis of SAE highly probable [21].

Intellectual deterioration was present in each patient and in 14 cases it was advanced to the point of dementia. Disturbances of concentration, and psychic retardation with apathy were characteristic of the patients with mild and moderate dementia. These were responsible for the results of psychological tests being better than expected from the low level of patients' daily activities and capabilities.

The results of neuropsychological studies showed that symptoms of diffuse damage of frontal lobes dominated, although it should be noted that Luria's tests do not allow for quantitative evaluation of neuropsychological deficits. Frequent abnormalities of gait (10 patients) and urinary incontinence (5 patients) could also be interpreted as symptoms of frontal lobe disorder [11]. However, in most cases signs of lesions in other cortical areas, more or less pronounced, were present. This could be expected from the cortical infarcts visible on CT scans.

The pattern of intellectual impairment in the group studied in some ways resembled that of subcortical dementia. The slowed speed of cognition, apathetic personality and depressed mood were present in most cases. The memory disturbances, however, were severe and do not correspond to the "forgetfulness" of subcortical dementia. One of the reasons for the only partial similarity of dementia in this group to subcortical dementia might be the presence of cortical infarcts. Cerebral infarcts are relatively common among SAE patients: they were observed in about 30% of cases diagnosed at autopsy [3]. On the other hand, it cannot be excluded that in some cases the degenerative process of Alzheimer type coexisted and was co-responsible for the dementia. At present, it is not possible to differentiate, using clinical data only, between pure vascular dementia and the mixed forms of dementia.

The dementia in SAE seems to have features different from dementia of Alzheimer type. It can be presumed that SAE patients require different rehabilitation techniques, which could improve their self-sufficiency.

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