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# Neuropsychological and Neuroendocrinological Disturbances Associated with Extracerebral Cysts of the Anterior and Middle Cranial Fossa

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**Summary.** In ten adult patients various clinical signs and symptoms led to the diagnosis of an arachnoid cyst in the anterior and middle cranial fossa. In this study a functional relationship between these cysts and disturbances of higher cognitive processes is described. In addition, neuroendocrinological impairment were caused by arachnoid cysts reaching into the suprasellar cistern. Considering the short medical history of our patients we presumed the disturbances not to be primary, but rather secondary, caused by an expansion of the cysts' volume.

**Key words:** Arachnoid cysts – Learning and memory – Temporal lobe – Gyrus cinguli – Neuroendocrinology

## Introduction

Arachnoid cysts are thin-walled, fluid-filled extracerebral cavities within the subarachnoid space. They may be congenital or acquired (secondary to trauma or inflammation). The conclusive diagnosis is a histological one. Sometimes, the differential diagnosis from other benign cysts, such as the subarachnoid hygroma or leptomeningeal cysts, can be difficult. Clinical asymptomatic and symptomatic (acute or chronic) developments have been reported (Shaw and Alvord 1975). Chronic developments generally occur during early childhood and are accompanied by deformity of the skull and mental retardation. The acute form is mostly seen in adults and manifests itself not only by signs of increased intracranial pressure but also by focal neurological disorders. For instance arachnoid cysts in the medial cranial fossa can lead to disorders of visual or oculomotor function as well as to focal seizures (e.g. temporal lobe epilepsy). Less frequent are reports in which arachnoid cysts have been demonstrated to be the direct cause of complex neuropsychological and psychiatric abnormalities. Perceptual disorders, impairment of judgement, cognitive functions and emotional lability have been described in patients with arachnoid cysts of the medial cranial fossa (Kuhnley et al. 1981; Colameco and Ditomasso 1982). Furthermore, impairment of memory has been noted (Heimans et al. 1979) but not examined with psychological tests. Admission and diagnosis of our patients with arachnoid cysts of the frontal and medial cranial fossa were based on various symptoms and complaints. All of them were examined neurologically, psychiatrically and by means of psychological tests. In patients with arachnoid cysts extending into the suprasellar space (suprasellar chiasmatic cistern), neuroendocrinological tests have been additionally performed.

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## Methods

Ten male patients between 21 and 26 years of age were admitted within 1 year and were included in this study. In addition to neurological and psychiatric examination, the diagnostic procedures included neurophysiological (EEG), neuroradiological (CT) and neuroendocrinological tests as well as test-psychological examinations.

By means of psychological tests we examined the crystallized (Cattell 1963) and fluid intelligence (capacity for information processing), the ability to store into the memory and to retrieve within different time-intervals, and the ability to concentrate and to react to different stimuli.

The multi-choice vocabulary-test (Mehrfachwahl-Wortschatz-Test; MWT-B: Lehrl 1976) records the crystallized intelligence via acquired verbal knowledge. This parameter remains unaffected by psychiatric disorders and estimates the premorbid level of intelligence. An additional test was applied to measure the actually available general and fluid intelligence (Kurztest für Allgemeine Intelligenz; KAI; Lehrl et al. 1980). This parameter estimates the capacity for information processing.

The long-term memory was examined with the help of anamnestic data. The Benton test (Benton 1962) describes the patient's ability to recognize geometric structures, memorize them for a short period of time and finally to reproduce them graphically.

To test "medium-term memory" (Bäumler 1974; Wickelgren 1972) the LGT-3 (Learning and Memory Test) was used. Several sub-tests gave information on the delayed retrieval of verbal and non-verbal (constructive, figurative) learning materials.

Furthermore, attention capacity and psychomotility were measured (Aufmerksamkeits-Belastungstest, d2, Brickenkamp 1978; Wiener Determinations-Test). To assess the personality of our subjects we employed the Minnesota Multiphasic Personality Inventory (MMPI; Spreen 1963).

Neuroendocrinological tests included the following hypothalamic and pituitary hormones: TRH, LH-RH, ACTH, TSH, LH, FSH, prolactin. Additionally $T_3$, $T_4$, cortisol (inclu-

**Table 1**

| | Cause of hospitalisation/ Symptomatology | Neurological and psychiatric findings | Psychological testing procedure | CT (diameter: = d) | Endocrinology EEG |
|---|---|---|---|---|---|
| 1 | Psychomotor *epilepsy*; for 3 years | Psychomotor epilepsy | No pathological finding | d: 1.5 cm; left anterior temporal lobe | |
| 2 | *Headache* dependent on posture, unsystematic vertigo; for 2 years | Impaired smooth pursuit (of eyes) vertigo (unsystematic) | Weakness of constructional memory and learning, reduced attention capacity | d: 3.5 cm; right temporal basal, extending to the suprasellar cistern | Hyperprolactinaemia |
| 3 | Right frontal *headache*, dependent on posture, disturbance of verbal memory; for 2 years | Mnesic disorder | Psychomotor reduction, information processing capacity reduced, fluid and premorbid intelligence different, global disturbance of memory and learning | d: 4.4 cm; paramedian, frontal and basal | |
| 4 | Postulated *vertebro basilar circulatory failure,* post-traumatic transitory perceptual disorder; for 3 years | Right-sided hemihypoesthesia | Verbal memory decreased compared to memory of constructional material | d: 3 cm; right temporal basal | |
| 5 | Anxiety, *derealisation,* failure in the faculty of thought, disorder in memory and learning, postulated psychosis (and neuroleptic treatment); for 2 years | Derealisation was proved to be a psychomotor epilepsy, decreased physical and mental power, MMPI: increased scale of psychasthenia, schizoidia and hysteria | Disturbance especially of verbal, but also of non-verbal memory and learning | d: 3 cm; rostral in the medial cranial fossa; amipaque CT: little communication with the liquor space | EEG-focus in left temporal leads |
| 6 | *Hemihypesthesia,* pronounced on the left leg; for 6 months | Hemihypesthesia pronounced on the left leg | Construct. memory decreased compared to verbal material memory | d: 3 cm; right temporal basal | |
| 7 | Psychomotor epilepsy, *headache,* galactorrhoe mnesic disorder; for one year | Epilepsy, galactorrhoea, mnesic disorder | Not tested; but in the anamnesis a clear indication for disturbance of memory | d: 3 cm; left temporal basal, extending to the suprasellar cistern | Hyperprolactinaemia |
| 8 | Right frontal or temporal headache, *deformation* of the skulls since birth | Affective instability | Psychomotor reduction, disturbance of constructional memory and learning | d: 8 cm; right temporal; extending from the medial to the anterior cranial fossa | EEG-focus in right temporal leads |
| 9 | Left frontal or temporal *headache,* progressive disturbance of memory (especially for names); for 4 years | Mnesic disorder | Disturbance of verbal memory and learning | d: 8 cm; left temporal postulated lobe agenesia; little communication with the liquor space | EEG-focus in left temporal leads |
| 10 | Affective instability, *reduced psycho-physical loading limit;* for 2 years | Affective instability, reduced psycho-physical loading limit | Reduced information processing capacity, fluid and premorbid intelligence differed, reduced verbal memory and learning | d: 8 cm; left temporal, extending from the medial to the anterior cranial fossa | EEG-focus in left temporal leads |

ding the circadian rhythm), testosterone and oestradiol were measured.

## Results

Individual data are presented in Table 1. In particular, headache, temporal lobe epilepsy and abnormalities in behaviour led to hospitalisation. The thorough neurological and psychiatric examination enabled a description of the psychopathological findings, the confirmation of a temporal lobe epilepsy, and only casually revealed focal neurological disorders such as an impairment of the oculomotor function or sensitivity (hemihypoesthesia). Electrophysiological signs (EEG) of an expanding process in the temporal area were found in four out of ten cases. Neuroendocrinological findings and an anamnesis with weakness in learning and memory indicated a process in the anterior or medial cranial fossa.

Finally, CT revealed arachnoid cysts in the medial cranial fossa in nine out of the ten cases, three of which extended into the anterior cranial fossa. One arachnoid cyst was localized in paramedian and basal left anterior cranial fossa. To complete the various clinical descriptions psychological tests showed partial mental disabilities corresponding to the cerebro-organic findings in nine out of ten cases ($P<0.05$). In patients with expanding arachnoid cysts in the medial cranial fossa we found a reduced ability to learn and memorize hemisphere-specific materials. In some, we also noticed a reduced attention capacity.

Cysts affecting the frontal lobe not only caused disorders of the memory, but also general psychomotor reduction, reduced capacity for information processing and a difference between fluid and premorbid intelligence. Arachnoid cysts connected with the suprasellar cistern led to elevated serum levels of prolactin. The prolactin concentration of two patients ranged between 20 and 30 ng/ml (normal range: 0.7–10.7 ng/ml). In one case galactorrhoea was noted. At the time of laboratory diagnosis, the patients were not being treated with any drugs which might have influenced serum prolactin level. The remaining neuroendocrinological data, especially the TRH, were found to be within the normal range. The CT revealed no incidence of pituitary adenoma.

## Discussion

The present study suggests a functional correspondence between expanding arachnoid cysts of the medial cranial fossa and mnestic disorders. These observations are similar to findings in patients suffering from temporal lobe lesions after surgical treatment for epilepsy or trauma: For instance, a generalized memory loss was found in bilateral lesions of the mediobasal temporal lobe including the hippocampus (Milner 1972; Penfield and Milner 1958), whereas unilateral lesions led to material-specific disorders of memory. Left-sided lesions (generally affecting the language dominant hemisphere) led to a deficit in verbal learning and recollection. Brain lesions on the right side produced disorders for non-verbal materials, for example, complex visual patterns, faces etc. (Milner 1974).

In eight out of nine patients arachnoid cysts of the medial cranial fossa led to material-specific weakness of learning and recollection produced by pressure on the temporal lobe including mediobasal and hippocampal structure. In addition, some patients showed a decreased attention capacity. A reduced attention capacity might explain weakness in learning and memory in general, but not material-specifity. Thus, this additional and non-specific finding does not contradict the close relation between expanding arachnoid cysts in the medial cranial fossa and material-specific mnestic disorders.

The lack of test-psychological abnormalities in one patient with an arachnoid cyst of the medial cranial fossa (case 1) can be explained by the very small size of the cyst. The extent of memory disorders has been correlated with the size of the lesion in the temporal lobe (Iversen 1977).

In two of the three patients with an arachnoid cyst extending into the frontal cranial fossa, we diagnosed, not only disorders of learning and memory, but also a general psychomotor reduction and a reduced capactiy of information processing. Besides the particular volume of these cysts there may be an additional effect resulting from a frontal lobe involvement in purposeful sensorimotor actions (Creutzfeld 1984; Lang et al. 1983), in motivation and will-power (Beringer 1944; Kornhuber 1980) and in the processing of short-term memory (Jacobsen et al. 1935; Gross and Weiskrantz 1964; Kornhuber 1973).

Of particular interest are the findings in a patient (case 3) with a cyst paramedian and basal in the anterior cranial fossa. This patient exhibited a significant generalized weakness of learning and memory in addition to a psychomotor reduction and a reduced capacity of information processing. Here, the cyst was localized next to the gyrus cinguli, lesions of which are known to cause an amnestic syndrome (Pribram et al. 1962). According to Kornhuber (Kornhuber 1973) the gyrus cinguli as part of the limbic system plays an important role in selecting the information for memory.

In three cases, abnormalities in behaviour, such as reduced adaptability to changing environmental demands or affective instability, led to hospitalisation. These disorders cannot easily be attributed to a certain region in the brain. The behavioural deficits rather appear to be produced by impairments of various partial functions.

To our knowledge hyperprolectinaemia in patients with cysts extending into the suprasellar cistern has never been described in literature. This hyperprolactinaemia can be caused by a reduced hypothalamic inhibition of the secretion of prolactin (Von Werder et al. 1977). Thus, it can be assumed that in our patients a narrowing of the portal vessels of the hypophyseal stalk by external compression led to a limited transportation of prolactin inhibiting factor to the anteropituitary.

In the majority of our patients the anamnesis was traced back for several years. We presume that the demonstrated neuropsychological and neuroendocrinological disorders, and the neurological abnormalities which are sometimes just as difficult to diagnose (e.g. temporal lobe epilepsy), extend beyond the time of clinical manifestations. However, the short clinical history is evidence for the expansion of the arachnoid cyst developing over several years. The expansion leads to impaired functions of the neighbouring cerebral tissue. They may increase in size because of osmotic pressure effects or because of one-way, valve-like communication with the subarachnoid space. Haemorrhages into the cyst caused by craniocerebral trauma have also been discussed as a pathogenetic factor (Varma et al. 1981; Williams and Gutkelch 1974).

In the literature arachnoid cysts are often described as incidental findings with no functional significance. According to our observations this statement should only be made after extensive test-psychological investigations.

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