Report on brain pathology in its relevance to neurology and psychiatry by Professor Karl Kleist

Translated and annotated by Marcelo Cetkovich-Bakmas[1] and Thomas Dorfmeister[2]

Introduction

Karl Kleist was born in Mühlhausen, Alsace January 31, 1879 (Neumärker and Bartsch 2003). His work was seminal in the study of higher brain function and phenomenology of endogenous psychosis within the Wernicke-Kleist-Leonhard school of psychiatry. Of note was his observation of catatonic phenomena and marginal psychosis, being perhaps the first one to propose uni- and bipolar affective disorders differentiation.

His paramount work, *The Brain Pathology* (Kleist 1934), was never translated. Based mostly on his experiences with traumatized persons during the first world war, it represents a scientific advance that put Kleist's wisdom in the position of a pioneer. This paper, presented at the Joint meeting of the Neurological and Psychiatric Departments on August 24th, 1936, in Frankfurt, was selected for translation because it represents a short sketch of the main subjects of his book. Only two short Kleist translations are available in English (Kleist 1952, 1960).

Significantly, Kleist always looked for traits or symptoms that link known brain lesions with those found in endogenous psychoses like schizophrenia. For example, he tracked speech disorders of confused schizophrenias with those found in aphasias. Additionally, he related moral flattening of hebephrenic patients with those having orbitofrontal cortex lesions.

The whole meaning of the Kleist Oeuvre is much more than a historioagraphic exercise. His mastering in clinical psychiatry and neuropathology predate by several decades the modern understanding of the Mind and Soul Relationship.

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Report on brain pathology in its relevance to neurology and psychiatry[3]

By Prof. Dr. Karl Kleist

The hour in which I am to speak to you today about the general significance of brain pathology is richly reminiscent of my first appearance before the assembly of German psychiatrists and neurologists -- 29 years ago and here in Frankfurt -- in which I made my first attempt[4] to advance from *one* point of brain pathology into psychopathology, from apraxia to psychomotricity. Eleven years later it was the *whole* brain pathology and its enrichment by the still fresh, but at that time not yet fully developed war experiences, which formed the content of my report at the Würzburg conference of 1918[5]. I would also like to remind you of 1924 and Innsbruck, when the then "Trends in Psychiatry"[6] were discussed, and it seemed to *me* that a *movement-based pathology* was again stirring vigorously, and that the next future belonged to it in association with the hereditary-biological-constitutional direction. Whether the first part of this prediction has come true as well as the second, you may judge at the end of my report!

When I speak of *brain pathology*, I mean with *Wernicke*, *v. Monakow* and *Henschen* the *general manifestations of brain diseases*, such as paralyses, sensory disturbances, aphasias or disorders of consciousness. The *importance of brain pathology for neurology and psychiatry* will then be seen mainly in the light which falls from it on the *general* neuropathological and especially on the *psychopathological manifestations of disease*.

The importance of general brain pathology for the *individual nervous and mental diseases* is also substantial, e.g., for the diagnosis, clinic and treatment of brain tumors, epilepsy, paralysis, schizophrenias; in regard to these, neurology is in the foreground. For lack of time, however, I must set aside presenting this as well, and I can refrain from doing so all the more because this subject will have its say sufficiently in the following lectures on brain injuries, *Pick*'s disease, schizophrenia and brain tumors, on neurosurgery and X-ray diagnostics.

It would also lead me away from the purpose of this report if I were to discuss here the *most general relations between performance, disorder and construction of the nervous system* and to justify the *principle of conditional dependence on form and localisation*, which seems to me to explain best the pathological findings of the brain. These general questions will also be treated in other lectures, e.g., in the one by *Beck* on word mutism and its anatomical bases.

I will deal briefly with these general questions only to the extent that is absolutely necessary for the understanding of individual observations used here. For a more detailed substantiation of these and other views, I may refer to my *Brain Pathology*[7].

I give references to the literature only as far as they are not contained in this book.

The nervous systems

The *spheres and zones that can be delimited at the cortex* correspond to the *systems of the individual senses* (Figures 1 and 2). From the sensory organ a *sensory part* of each system leads with different interruptions up to the cortex, from where in each case a *motor part* leads down to the assigned musculature. The sensory and motor system parts and their cortex zones respectively are joined by the psychic zone of each system, which is located only in the cerebral cortex.

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[3] Originally published in: Zeitschrift für die gesamte Neurologie und Psychiatrie. Volume 158 (1937), pages 159–193.

[4] Kleist, K. (1906) Über Apraxie. Monatsschrift für Psychiatrie und Neurologie, 19, 269-90. (Not quoted in the original).

[5] This seems to be unpublished.

[6] Allg. Z. Psychiatr. 82 (1925).

[7] Leipzig: Johann Ambrosius Barth 1934.

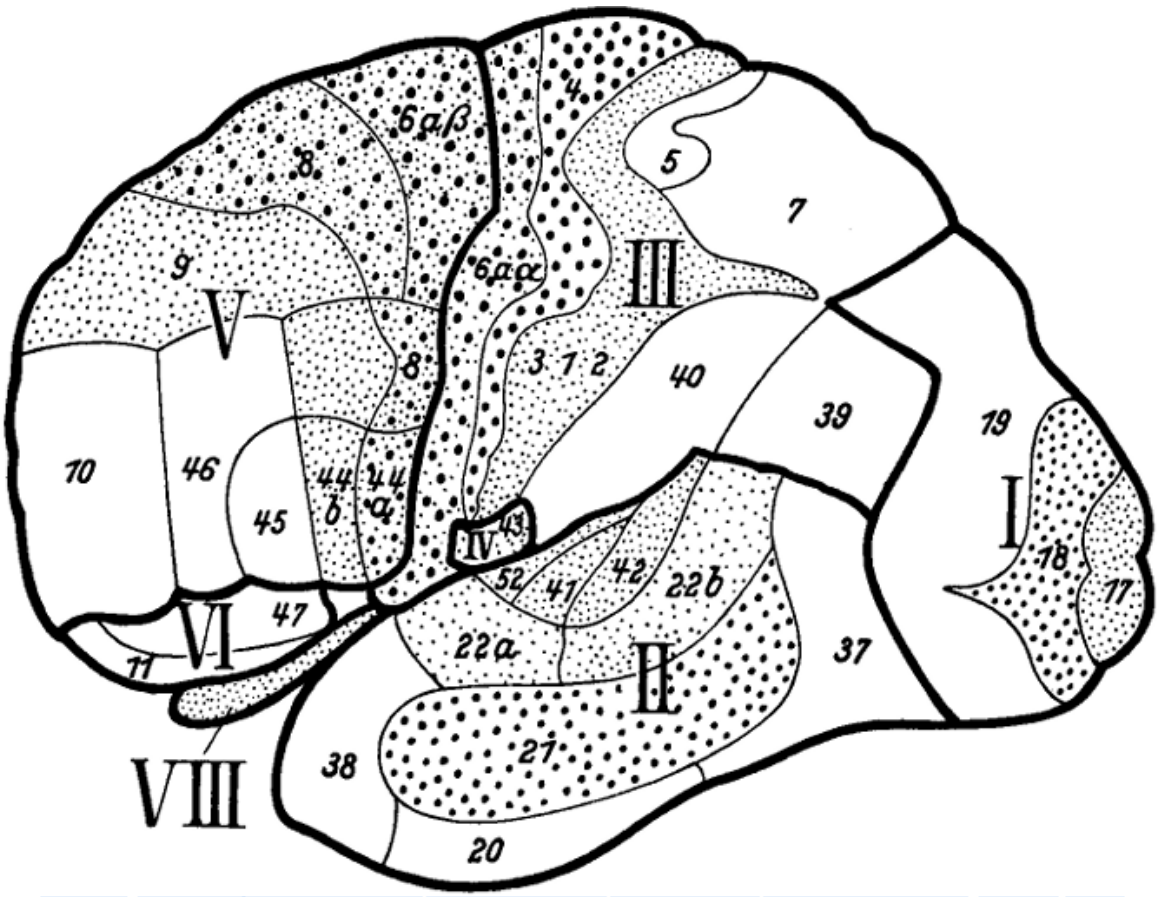
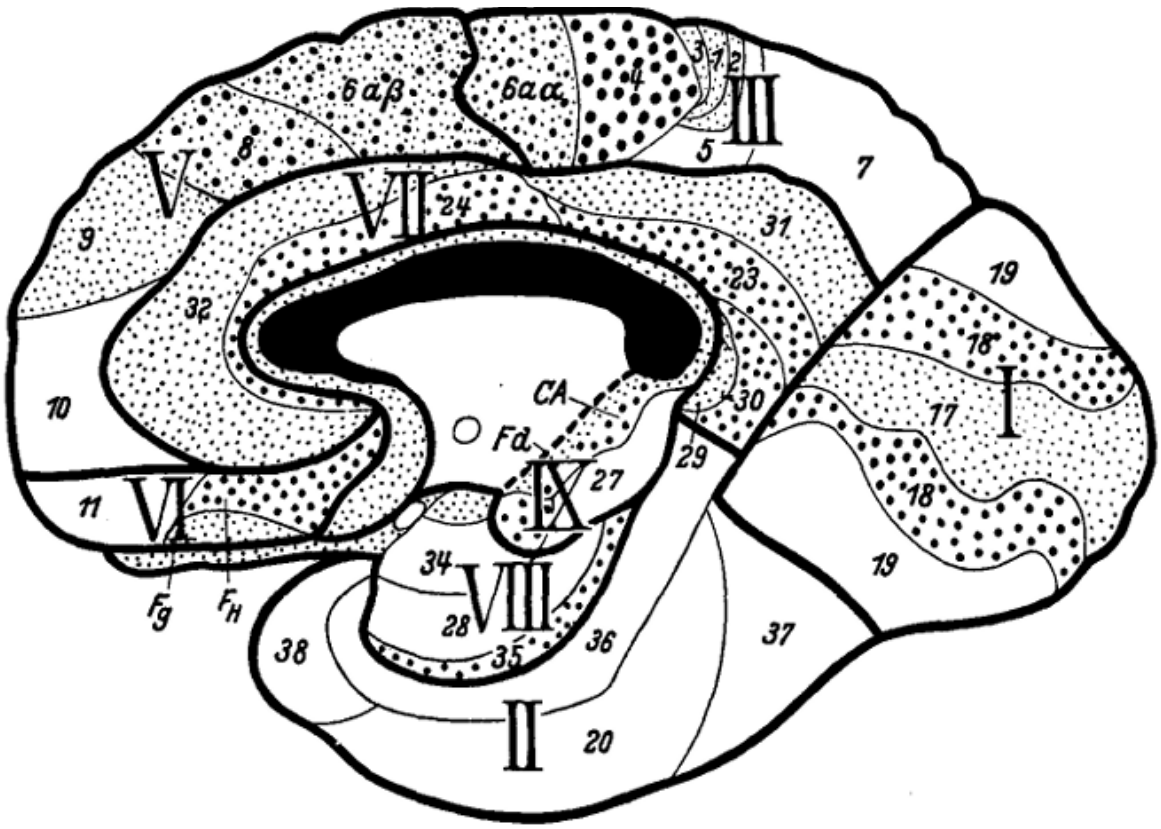


Figure 1. Spheres and zones of the cerebral cortex. Exterior. Architectural fields after Brodmann with individual modifications after v. Economo and Vogt.



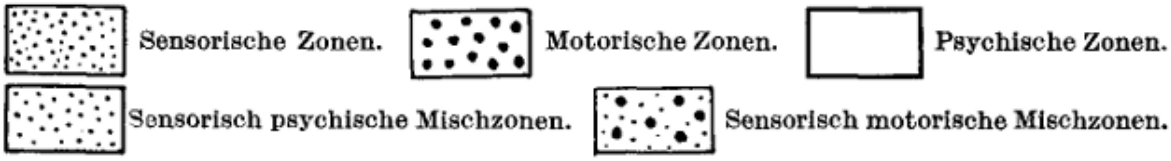


Figure 2. Spheres and zones of the cerebral cortex. Inside. Architectural fields after Brodmann with individual modifications after v. Economo and Vogt. I Lobus occipitalis = visual sphere. II Lobus temporalis = auditory sphere. III Lobus centro- parietalis = tactile sphere. IV Area subcentralis = sphere of taste. V Lobus frontalis = labyrinthine - myesthetic sphere. VI Lobus orbltalis VII Cingulum, retrosplenum I Sphere of inner sensations (ego sphere). VIII Lobus piriformis} IX Lobus ammonicus olfactory sphare.

The importance of brain pathology for neuro- and psychopathology results most clearly when pathological disorders are traced in these systems and their subdivisions. There are then three more or less clearly delineated sections on each system: 1. A section of *neuropathological* disease symptoms, 2. a section of disease symptoms based on brain pathology, and 3. a section of *psychopathological* disease symptoms.

The systems themselves fall into the three groups established by *Sherrington*[8]:

I. The *exteroceptive* systems: the tactile, auditory, vision, gustatory and olfactory systems.

II. The *proprioceptive* system of force, movement, rotation and position sensations from the sensory organs of the musculoskeletal system and the vestibular nerves.

III. The *enteroceptive* system of sensations from the viscera, glands and vessels.

I. The exteroceptive systems

*On the **tactile system*** -- and, first of all, on its *sensory part* -- 1. The *neuropathological section* has to do with the peripheral sensory nerves, the dorsal roots, the sensory nuclei and tracts of the spinal cord, and with the various sensory disorders and their propagations; these include the ataxias, tension and reflex changes dependent on receptive disorders.

2. The section based on brain pathology concerns the sensory pathways and centers -- from the dorsal column nuclei and the sensory cranial nerve nuclei on through the medial lemniscus to the thalamus and from there through the internal capsule to the Gyrus postcentralis -- with the corresponding sensory disorders, which include postcentral agnosia of form and touch.

3. The *psychopathological section* in the *left parietal lobe* includes disorders of *sensory recognition* (agnosia): tactile agnosia for objects and autopagnosia i.e., right-left disorientation, disorientation to the own body (so called finger agnosia). The haptic-psychic section also includes *sensory apraxias*, which are based on the loss and insufficient utilization of kinesthetic and other engrams for individual movements or for temporal or spatial movement correlations (so-called ideokinetic, ideational and constructional apraxia). Special types of apraxia are *agraphia* and certain inabilities to draw objects (Figure 3 and 4). Probably there are also *higher* haptic-psychic disorders, which involve the solution of purely *mental connections* in the tactile and movement processes; however, I would like to discuss them only in connection with corresponding disorders of the optical system.

[8] *L. R. Müller* does not completely agree with this and distinguishes between an environmental nervous system, a nervous system for involuntary muscle tension and for posture, and a vital nerve system.

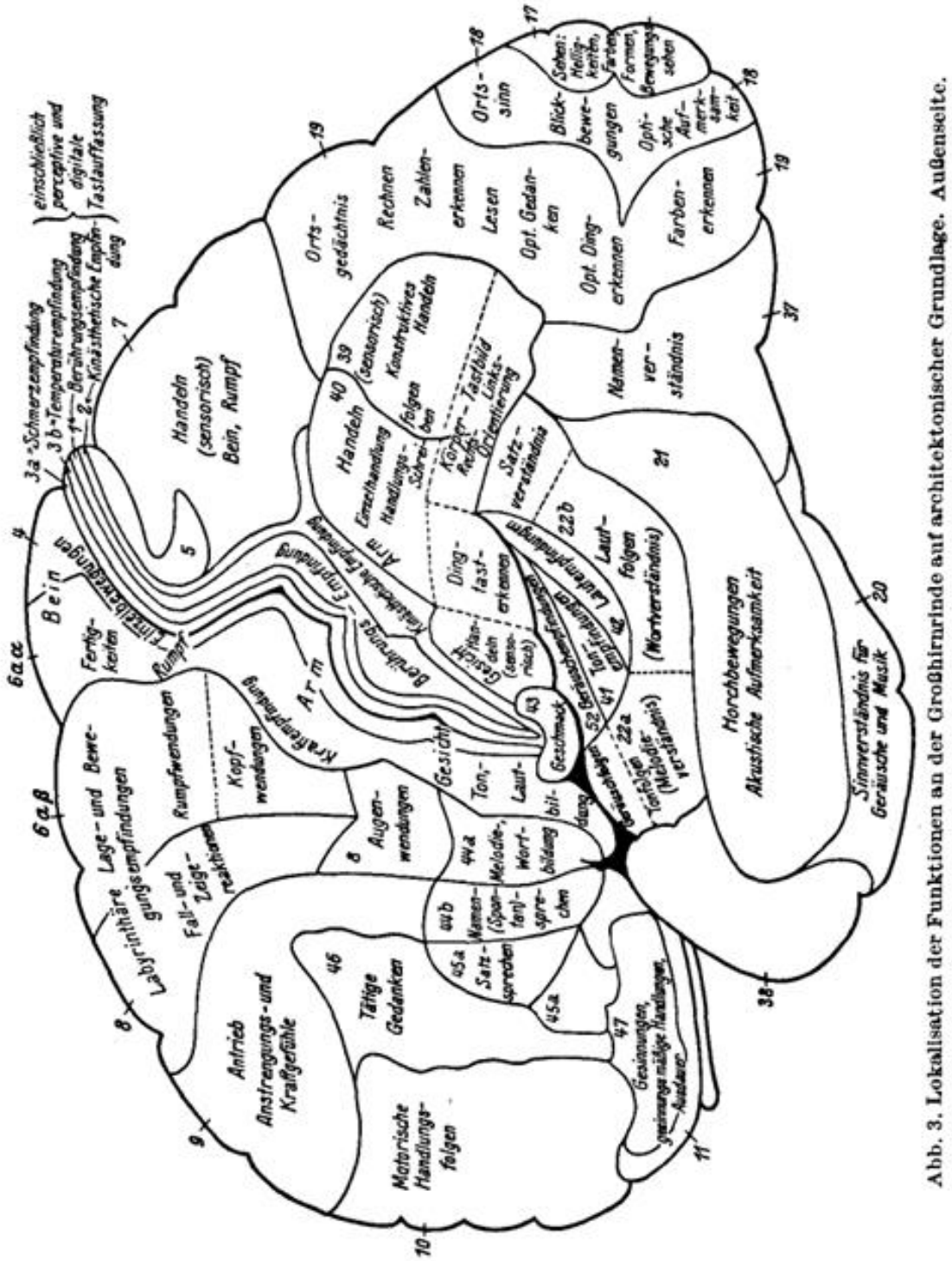


Abb. 3. Lokalisation der Funktionen an der Großhirnrinde auf architektonischer Grundlage. Außenseite.

Figure 3. Localization of the functions of the cerebral cortex based on architectonics. External Face.

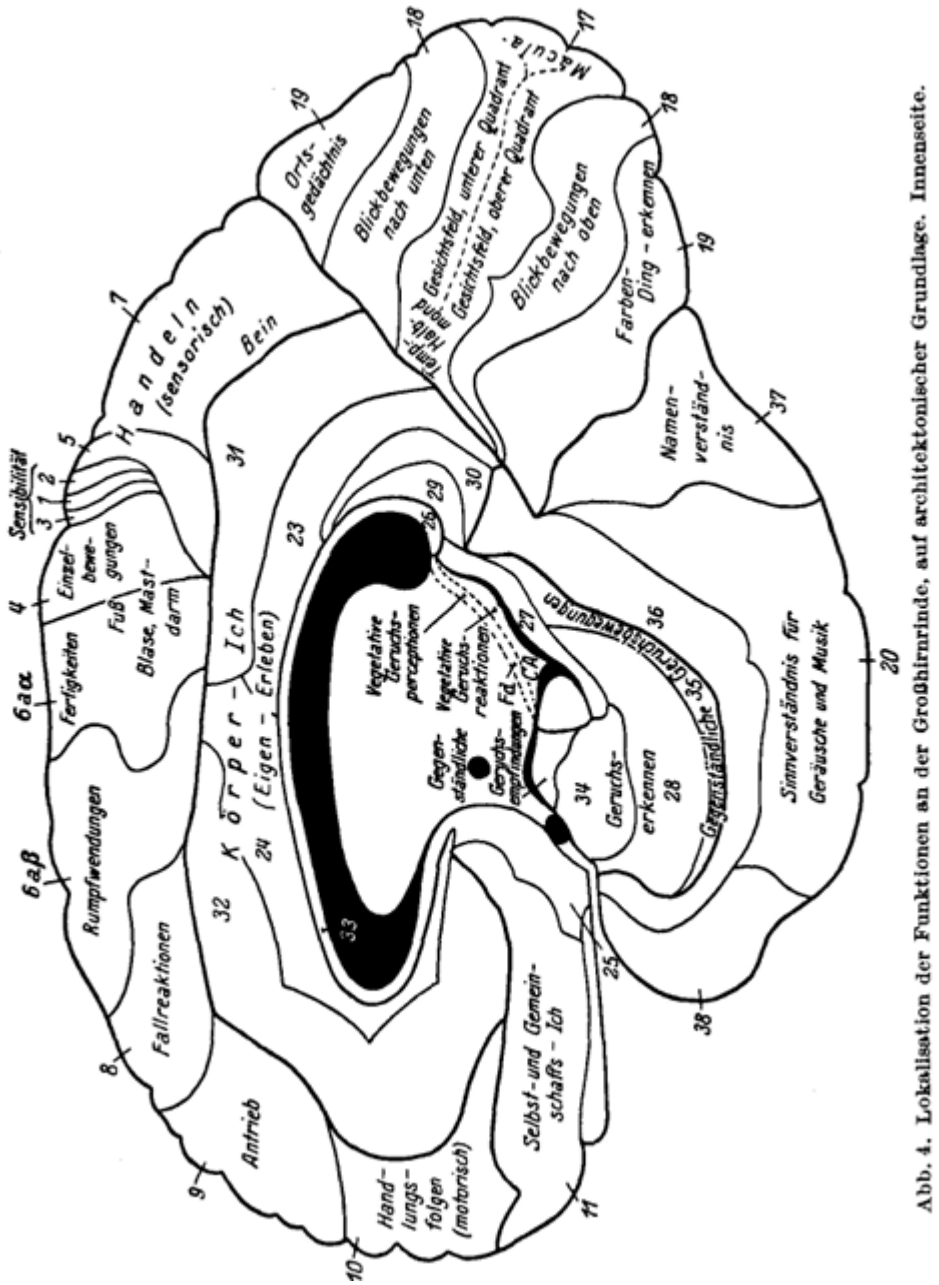


Figure 4. Localization of the functions of the cerebral cortex based on architectonics. Internal Face.

If I call the *3rd section* of the tactile system a *psychopathological* one, this is not to say that it is not also of the sort based on brain pathology. The disorders belonging to it are as closely connected with anatomical formations of the parietal lobe as certain sensory disorders are with the posterior central gyrus. This designation is only intended to indicate that the parietal disturbances of the tactile system are of a *higher*, *finer* and *more intricate* nature, that *memory traces* participate in them, and that they are therefore closer to the *core* of our mental constitution than a simple loss of sensation**,** which can arise from the posterior central gyrus down to the peripheral nerve. Also, such "neurological" sensory disturbances cause failures of mental material, but much simpler and more segregated, while the apraxias and the physical disorientation are very often accompanied by mental failures in other areas -- for example, in a widespread cerebral arteriosclerosis, a focal paralysis, a senile brain disease.

After all, it was just such widespread brain and mental diseases from whose "*dementia*" the disturbance forms of ideatory apraxia, etc., were first separated out. A simple loss of sensibility in one hand, on the other hand, occurs only rarely as a partial symptom of a cerebral

pathological manifestation of a paralysis -- by a focal disease in the middle part of the posterior central gyrus -- and only very exceptionally as a neurological symptom in an alcohol related Korsakoff with polyneuritis.

At the *motor section* of the tactile system only neuropathological and brain based pathological disorders can be distinguished: neuropathological are the neuromuscular paralyses of a progressive muscular dystrophy, the peripheral nervous arm paralysis of a plexus strain, the atrophic leg paralysis of a poliomyelitis, the spastic spinal cord paralyses, e.g., in a multiple sclerosis with foci in the lateral columns. However, the spastic paralyses occur in a similar way in brain based pathological disorders, such as due to injury of the crura cerebri, internal capsule, the thalamic radiation and precentral gyrus (due to encephalomalacia, tumors, solitary tubercle, etc.). A motor form of apraxia further belongs to the neuropathological section of the motor-haptic system, the *innervatory* or limb kinetic *apraxia*, which originates from the anterior part of the precentral gyrus corresponding to area 6aα and results in the loss of learned skills: of standing and walking from the upper part of precentral gyrus, of finger skills from the middle, of sound and tone formation and other mouth and facial skills from the lower part of the precentral gyrus.

At the ***auditory system*** I limit myself to the most important part of the same, the *sound and speech perception*. The *neurological and cerebro-pathological section of the auditory system* includes the disorders of *tone perception* in diseases of the cochlea, the auditory nerve, the cochlear nuclei and pathways, the lateral loop to the medial geniculate body and the acoustic radiation to the transverse gyri of the temporal lobe.

Even the *deafness for tone [Lauttaubheit]* resulting from partial injury of the left posterior transverse gyrus or from interruption of the left acoustic radiation (together with part of the corpus callosum) still acts as a partial hearing disorder (Figure 3). This is especially true because there are no paraphasias in spontaneous speech; hence *Déjérine*'s term "pure speech deafness"; *Wernicke*'s conception as subcortical sensory aphasia does not apply to all cases.

Psychopathological, on the other hand, are the *higher* disorders of language comprehension, which can be distinguished according to the gradual language structure as word deafness, deafness for name and sentence. Although these aphasic disorders are all well known in their brain connections -- with fields of the posterior transverse and temporal convolutions -- (Figure 3) and occur predominantly in focal diseases, it is *more intricate* and partly *mnestic* performances that fail in them. In auditory verbal agnosia the temporal structure of the phoneme is dissolved, which also leads to the literal paraphasia of speech. In agnosia for sentences the temporal and relational formula of the word order and its dependent word changes and word combinations that make up the sentence is disturbed. The dissolution of these also results in an expressive disorder and aggravation, a paragrammatism, and a non-occurrence of appropriate sentence formulas, an amnesic agrammatism. The deafness for names deprives the words of their indicative meaning of extra-linguistic contents and processes. This disorder manifests itself in speech, in that the words, when they are to be used as names, do not come to mind or only with confusion: amnesia and paraphasia for names.

It is a general law of sensory aphasias that the disturbance of comprehension is less severe and more easily to regress than the dependent order of speech. Therefore, sometimes aphasic disorders occur which appear to be expressive in nature only and are not motor aphasias but are easily confused with them. Such predominantly expressive temporal speech disorders are *conduction aphasia*, which is related to deafness for tone, and *amnestic aphasia*, which is related to deafness for name.

The loss of word and sentence formulas and of names also subjectively means a *loss of mental possession*, which is particularly evident in the loss of foreign languages in multilingual people who suffer sensory aphasia. In this sense, any sensory aphasia is a circumscribed *dementia*.

But also, their *frequent occurrence in the context of mental diseases* stamps the sensory aphasias, especially their higher forms, to *psychopathological phenomena*. From the jumble of "dementia" and "confusion" all these clinical pictures have been brought to light one

after the other. They appear in senile cerebral atrophy, in paralyses and widespread arteriosclerosis, but also in symptomatic psychoses, in epileptic stupors and stupefactions, and they play a special role in *schizophrenias*, especially in the schizophasic form.

Certain subtle disturbances of language, e.g., those of word transformation and the composition of words and parts of words, as practiced in declension, conjugation and comparison, never occur purely in focal diseases, but are sometimes supplied by schizophrenics virtually in pure culture. However, the aphasic nature of schizophrenic neologisms and word deformations can only be revealed if the patient is examined according to the rules of aphasia examination and if it is taken into account how the defective speech patterns can be caused by the arrangement of the examination and by no means depend only on delusional contents and pathological attitudes. The still common collection of spontaneous and random strange speech utterances can never solve their riddles, because the relation of the speech utterance to a certain task remains unclear.

Thus, a *paranoid schizophrenic*, who was conspicuous in her spontaneous speech by word *neologisms* such as wrinkled pills, yellow powder, chloroform chocolate, quicksause fever, referred to the fins on the image of a fish as *water wings*, a diver as a *water diver*, a lamp as a *flame lamp*. Sometimes a search for the word was quite obvious, for example, when she said to a black silhouette: fantasy dolls… night pictures… night dolls[9].

The expected finer disturbances of *speech comprehension* in schizophrenics could also be demonstrated by my colleague *Fleischhacker*[10]. The *anatomical basis* of such schizophrenic speech disorders is to be found in the cell failures in the cortex of the temporal lobe, especially in the posterior T_3, as demonstrated by *Miskolczy* and *Hechst*.

Kraepelin already concluded from the *speech disorders in dreams* similar to schizophrenia he observed in himself that there are functional disorders of the *temporal lobe*, and the same conclusion can be drawn from the linguistic lapses observed by *C. Schneider*[11] when falling asleep and during extreme fatigue. Apparently, the temporal speech areas are functionally switched off during sleep.

However, I would like to explicitly note that by far not every linguistic anomaly of a schizophrenic is to be interpreted in this way, as will become apparent from the progress of my report.

Finer paraphasic and paragrammatic speech defects also occur with *circumscribed linguistic development deficiencies*, so that then a rich conceptual vocabulary faces an insufficient linguistic expression ability. The two men I am thinking of were misjudged as schizophrenics because of their peculiar linguistic expression and came to the clinic for evaluation because of sterilization. One of them describes the *window promenade* (Figure 5) as follows: "A gentleman, who is very devoted to two ladies of attention, is with his interests involuntarily thrust into the path of a child and throws it."

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[9] *Kleist*: Münch. med. Wschr. 1914 I.

[10] *Fleischhacker*: Mschr. Psychiatr. 77 (1930). --

[11] *Schneider, C.*: Die Psychologie der Schizophrenen. Leipzig 1930.

Figure 5.

On the ***visual system***, the *neuropathological and the cerebro-pathological* sections of the *sensory part of the system* extend from the retina and the optic nerves through the chiasm and the tractus opticus to the lateral geniculate body and through the optic radiation to the visual cortex in the area striata (field 17) of the occipital lobe (Figures 1--4). Beginning from the tract, the disturbances are the same: hemianopic deficits of different extent and location, according to the type, reductions of brightness and color vision, vision of motion and form.

Blindness of form is not yet an agnosia, but a perceptual disorder corresponding to postcentral tactile paralysis and is caused by finer and partial cortical disruptions of macular vision in the polar area of the area striata.

I was recently able to prove this again in a particularly pure case of this kind, a gunshot injury of both occipital poles[12], and to show that the loss of the perception of form or shape then remains limited to vision and does not have the general significance attributed to it by *Goldstein* and *Gelb* for all mental areas. In *Goldstein*'s case, as can be seen from other indications, there were also extrastriate brain damages.

The *psychopathological section* of the optical system contains in Brodmann's field 19 first of all the *optical-agnostic* disorders in which the recognition of objects and persons is suspended despite the presence of form perception: *optical - material agnosia*. Special forms are the *alexia* for letters, numbers and notes. In the case of *color agnosia*, colors are distinguished, but are not classified in general color terms and are not perceived as colors of certain colored objects, e.g., red is not perceived as blood color. With the optical recognition disturbances also a loss of subjective optical conception goes along. However, these disorders are still on a lower level of psychopathology.

A *higher optical disturbance* behaves differently, where the individual objects can be recognized and imagined, but the meaningful connection between objects, persons and processes escapes or is misunderstood.

[12] *Kleist, K.*: Dtsch. Z. Nervenheilk. 138 (1935).

An occipital injury[13] victim interprets the *blindman's buff picture* like (Figure 6): "He has his eyes blindfolded and wants to steal something... that they do not know him."

Figure 6.

The meaning of blindfolding as a game is not grasped and is misunderstood as an intention not to be recognized, whereby, moreover, not being able to recognize and not being recognized are confused with each other. Next to it runs the misinterpretation of the grasping as an intention to steal, which then enters into the nonsensical relationship with the blindfolding: in order to be able to steal unrecognized, he had blindfolded himself. The persons and objects are thus all correctly recognized, as are their spatial relations; what is *defective* are the *mental relations* of the persons and objects to each other assumed by the brain-injured person, the meanings, justifications, causes and effects. The corresponding *abstract thought-experiences* -- whose knowledge one owes above all to the investigations of *Bühler* -- do not emerge in a proper way, and the faulty correlations, the mental *derailments* and *conflations* prevail over a simple missing of contexts. In other words, *paralogias* predominate over *amnestic-alogical deficits*. We can therefore also describe this higher recognition disorder as a *receptive* (here *optical*) *thought disorder with paralogias*.

If our brain-injured person then, when asked to name *metals*, says: "hammer and coal," he obviously tried to *imagine* metals *visually*; but the conceptual relations between "metals" and the corresponding individual ideas were so poorly reminiscent that he could only think of nothing else than a hammer, presumably as a metal object and as a mining tool; By thinking of coal, he lost himself from the conceptual circle addressed by the task. Thus, the same thinking errors as in *optical perceptions* also occur in the mental *processing of visual imaginations*. On the other hand, the thinking disorder receded in tasks which were not so much based on sensual material. *Binet*'s absurd sentences and questions of reason, proverbs and instructive narratives were almost always correctly explained by our brain-injured patient, and he was able to form thoughts with the three-word test without any problems.

[13] Brain Pathology p. 548.

In retrospect, one now recognizes that in the optical field, too, there is a *gradual build-up* from simple to higher and more intricate performances and a corresponding gradual series of disturbances:

- the color blindness and form blindness, which still belong to the purely brain-based pathologies of visual disorders -- starting from the area striata (field 17),
- the optical recognition disorders (agnosia for objects, alexia, color agnosia),
- the optical thought disorder with paralogias and occasional amnestic alogia.

The latter two perturbations are probably tied to separate entities within the optic-psychic field 19.

I have seen this *optical thought disorder* not only in several occipital lesions, but also in a *cerebral luetic* with cortical sclerosis and softening foci in the left occipital lobe, furthermore in *paralyses*, where they appeared especially again in illustrations of pictures and sometimes transitioned into *optical agnosia*. For example, one paralytic explained the image of the *window promenade* in this way: "The gentleman comes home and they finally hit each other, the gentleman may have killed the other gentleman because he is lying on the ground." Another paralytic is to name officers: "Frederick the Great, Bismarck, Zeppelin." A lie has no legs: "Those who have such short legs lie more." No rose without a thorn: "That is a sleeping beauty [Dornröschen]." The examples are taken from the scheduled mental examinations of paralytics conducted by *Schlesinger*[14] at my clinic.

Paralogical thought disorder also occurs in diffuse cerebral arteriosclerosis and in senile dementia, epileptic stupor and mental decay. But it is extremely frequent in *schizophrenics*, especially in the confusional and paranoid forms. And here, too, the paralogical misapprehensions and amalgamations show themselves with preference in *optical* conditions (perceptions and ideas). A schizophrenic explains the *snowball picture* (Figure 7):

Figure 7.

[14] *Schlesinger, Kl.*: Über alogische und paralogische Denkstörung im Rahmen der paralytischen Demenz. Inaug.-Diss. Frankfurt a.M. 1936.

"This is Max and Moritz, this is happiness and glass, and he has the slate in his hand and this is happiness. He has the snowball in his hand, he is the luckier one." He probably has on his mind: the vision slate will break like happiness and glass, the snowball is not fragile, so its owner is happier than the one with the slate. Difference between mountain and range: "There is Mount Sinai and the mountain Horeb." And the difference? "There is the desert in between" (confusion of difference and spatial in-between). With hat in hand, you can go through all the land: "To carry one's luck carefully in one's hand." A very tricky conflation: luck takes the place of the hat and is carried in the hand, but the hat does not completely disappear from this connection but is turned away to "carefully"; in the background there is probably the thought that one must be careful with one's luck.

In schizophrenics, too, the optical-paralogical disturbances sometimes turn into *optical-agnostic* ones. One patient describes the *circus picture* (Figure 8): "This is the window-danseuse."

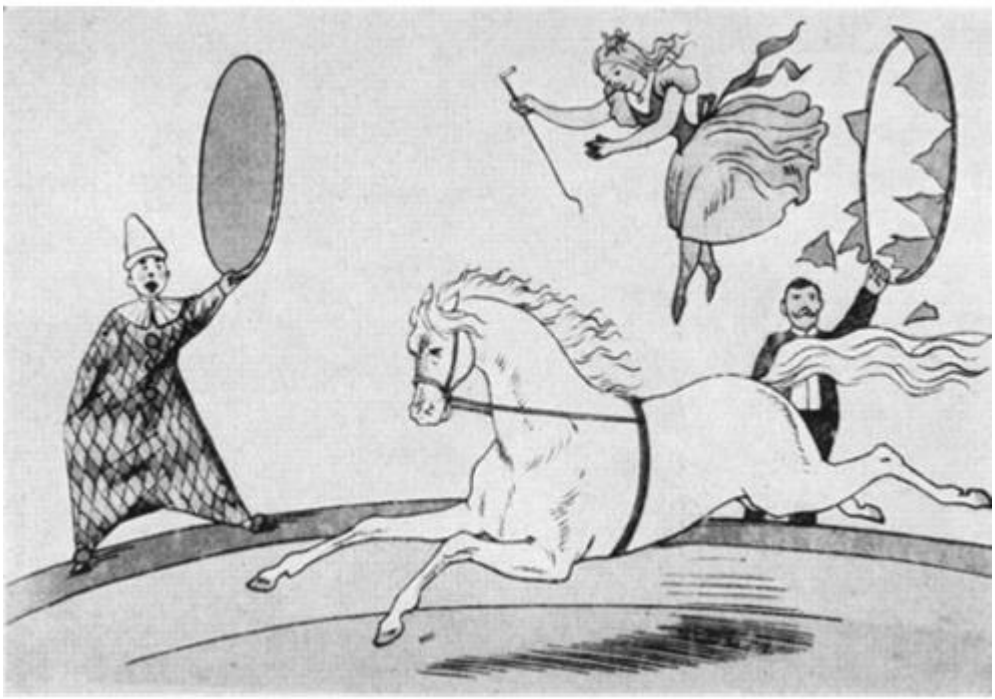

Figure 8.

This at first blatant derailment can be explained by the fact that the circus director holds up a hoop, which the patient mistakes for a window. (A window?) "The broken frame." Patient means the hoop through whose paper covering the circus rider has just jumped; broken refers to the hoop (frame!) instead of the covering. (What does the clown do?) "He has the whole frame." She wants to say: the still intact frame. "It is framed again" instead of still covered. (What is the tire covered with?) "Cellulose, or what is that, green glass is also available." Confusion of paper, cellulose and glass, the latter determined by the continued effect of "frame." Further the patient explains: "These are safety devices, while they hold this, she can jump." Apparently, the hoop is now misunderstood as a safety device. (What kind of safety device?) "When this riding starts and during this time, he has this captive balloon." Due to the continuation of the safety device, the hoop becomes a captive balloon, and in a short time the patient has successively misjudged the hoop as a window frame, covered with cellulose, with green glass, as a safety device, as a balloon. The transition from paralogy to agnosia is very clear here.

Often paraphasic and paragrammatic speech defects are connected with paralogical thought disorder, namely for reasons of anatomical adjacency. Our sick patient says about "old Fritz": he was "very complacent... benevolent." Difference between duck and chicken: "The duck has more colors, kind of wings..." Duck is a difference between a chicken." Hindered

naming in both examples, furthermore there is a paragrammatic error in the second one: There is a difference between duck and chicken. The already mentioned paranoid schizophrenic with paralogies called a newspaper "a drawing of attention for political conditions" -- wrong choice of names under wrong use of word combinations.

The schizophrenic disease process thus damages both in the optic and in the temporal brain area primarily the facilities serving the highest performances, namely those of the grammatical and the naming performance or those of the optical thinking processes and both often in the same kind of misattribution and mixing (paralogy, paraphasia, paragrammatism).

It should be added that there are also paralogic *action disorders*, "*crazy actions*" in *schizophrenics*, which may represent the highest form of *apraxia* already indicated earlier, a *mental parapraxia* or a *paralogical thought disorder in the tactile system*: A sick patient pours hot oil into his ear to destroy his hearing, so that he becomes a great composer (because Beethoven was deaf). The same patient goes on the street in the shirt at night, because all people had died and because the dead are buried only with their shirt dressed.

If it is therefore a futile attempt to derive all mental disorders of schizophrenics from one root, the schizophrenic disease process has a peculiar predilection for causing para-disorders, i.e., derailments, transgressions and conflations, in very different mental areas, both in speech and in thinking and acting. This is similar to the way in which paralysis favors ordinary failures in all mental areas, to the way in which memory deficiencies develop everywhere in senile dementia, and to the way in which the tendency to perseverate asserts itself everywhere in epilepsy. Only in this sense one can speak of a uniformity of the disease symptoms in one and the same disease.

We pass over the optical acalculias and take a look at the *motor part of the optical system*, which includes *eye and gaze movements*. The optic-motor cortical field is probably field 18 in the occipital lobe (Figures 1--4), which sends its impulses along the optic-motor pathway in the stratum sagittale internum of the occipital and parietal lobes to anterior pair of the quadrigeminal bodies and later through the posterior longitudinal fasciculus to the oculomotor nuclei in the midbrain and hindbrain.

According to *Vogt*'s stimulus experiments, the optic-motor field in monkeys is located in the cortical stripe surrounding the area striata, which, according to the new investigations of *Beck*[15], corresponds to *Brodmann*'s field 18 and is represented by a regio parastriata lying next to the area striata and a regio peristriata surrounding it again. The regio parastriata is also very suitable for motor performance according to its structure, because it contains large pyramidal cells in layer III, which form a limes giganto-pyramidalis in places, and it also contains, what seems to be even more important, particularly large cells in layer V here and there, while this layer is otherwise only poorly developed in the entire occipital lobe. *Vogt* describes the occipital eye field of Cercopithecus as field 19, but this cannot be adhered to, since according to *Beck* there is no cortical district corresponding to field 19 on the outside of the occipital lobe in the monkey. The occipital fields corresponding to *Brodmann*'s field 19 -- *Beck*'s om, os, oi -- lie in the monkey rather completely on the medial side and have not been stimulated by *Vogt* at all.

If *Förster*[16] did not obtain eye movements in humans from the small piece of field 18 located on the outside of the occipital lobe, it should be remembered that the largest extent of this field is on the inside of the occipital lobe. The fact that *Förster* was able to obtain eye movements from the whole extended lateral field 19, however, does not prove that this is the actual optic-motor region, since the eye movements may have originated from the optic-motor fibers running below the cortex.

The *fixation movements* from the environment of the macula field located in the polar region are indispensable for searching, finding and retaining optical impressions and thereby become carriers of the optical sense of place (the absolute optical localization), the optical attention and the certainty of optically guided movements. Therefore, in the case of bilateral cortical or subcortical disruption of fixation movements, the following also occur: locational

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[15] *Beck*: J. Psychol. u. Neur. 46 (1934).

[16] *Förster*: Verh. dtsch. Ges. inn. Med. 46. Kongr. Wiesbaden 1934.

cecity, restriction of the scope of optical attention with the consequences of comprehensive disorders of recognition, counting and reading, as well as *optical ataxia* (*Balint*), which is probably to be distinguished from constructional apraxia. There is also *local disorientation* as a result of loss of optical-topographical *memory images* obtained with the help of eye movements. The *optic-spatial agnosia* belongs to the optic-psychic field 19, namely to its upper sections. Thus, also the eye movements or their disturbances have a *psychopathological* superstructure.

These disorders are also found not only in focal diseases, but also in the case of widespread organic mental disorders, diffuse cerebral arteriosclerosis, senile diseases, paralysis, in epileptic and symptomatic clinical pictures.

I will only touch upon the ***taste and olfactory system***, since only a *few psychopathological* disturbances in a narrower sense have been observed in these systems. In another respect, however, both systems are very remarkable, because in contrast to the tactile, auditory and visual systems, they are not pure exteroceptive systems, but can at the same time be regarded as enteroceptive systems, since their own sensations, whose stimuli in fact come from the outside world, are only indirectly received from the oral and nasal cavities and partly also bring states of these vestibules of the body cavities to perception.

Perhaps it is related to the fact that the cerebral olfactory organ is a double organ and consists of an anterior, the lobus piriformis and a posterior olfactory brain, lobus ammonicus, each of which also has special supplies in the stria olfactoria lateralis (to the piriform cortex) and the stria olfactoria medialis via the corpus callosum to the lobus ammon. (Figures 1--4). The anterior olfactory brain apparently represents the exteroceptive part of the double organ. In case of damage of its sensory field, the periamygdalar region, *olfactory hallucinations* occur, and olfactory agnosia (in case of preserved olfactory sensations) probably results from injury of the corresponding psychic field in the entorhinal region. The posterior ammonic olfactory brain, on the other hand, is able to act powerfully through the fornix on the vegetative mechanisms of the hypothalamus and thus presents itself as a probably enteroceptive olfactory organ.

For *taste*, the organ of external, representational perceptions is the *taste field*, which, according to war experiences and other observations, is certainly located in the *subcentral region* (Figures 1 and 3). The subcentral region is joined inwardly by the *insula*, which possibly contains an enteroceptive taste apparatus acting on vegetative processes.

II. The proprioceptive systems

We turn to the *proprioceptive* system (Figures 1 and 2), which can be considered as a single entity, even though its sensory sources are distinct: Sensory organs in the *muscles*, tendons, fasciae and joints, as well as the *vestibular apparatus*, which convey sensations of movement, position, rotation, force and gravity. The *neuropathological* section of the system includes the disorders of spinocerebellar conduction (cerebellar tract of lateral funiculus and posterior funiculus of spinal cord) that we encounter in *Friedreich*'s ataxia, for example, as well as the vestibular disorders with vertigo, falling, pointing past, gaze distractions and nystagmus.

The *cerebral pathological* section includes the functional disturbances of the central vestibular conduction through the posterior longitudinal fasciculus to the red nucleus and to the medial thalamic nucleus and further to the frontal brain (*Le Gros Clark*) (Figure 10). In contrast, the vestibular and spinal excitations collected in the cerebellum are conducted to the middle ventral thalamic area (nuclei va_1 and vb) via the cerebellar superior peduncle and reach the precentral gyrus. In this section, therefore, the symptoms of the cerebellum -- the asynergy, dysmetria, hypotonia, adiadochokinesia, the intention tremor and the reduction of gravitational sensation -- also have their place.

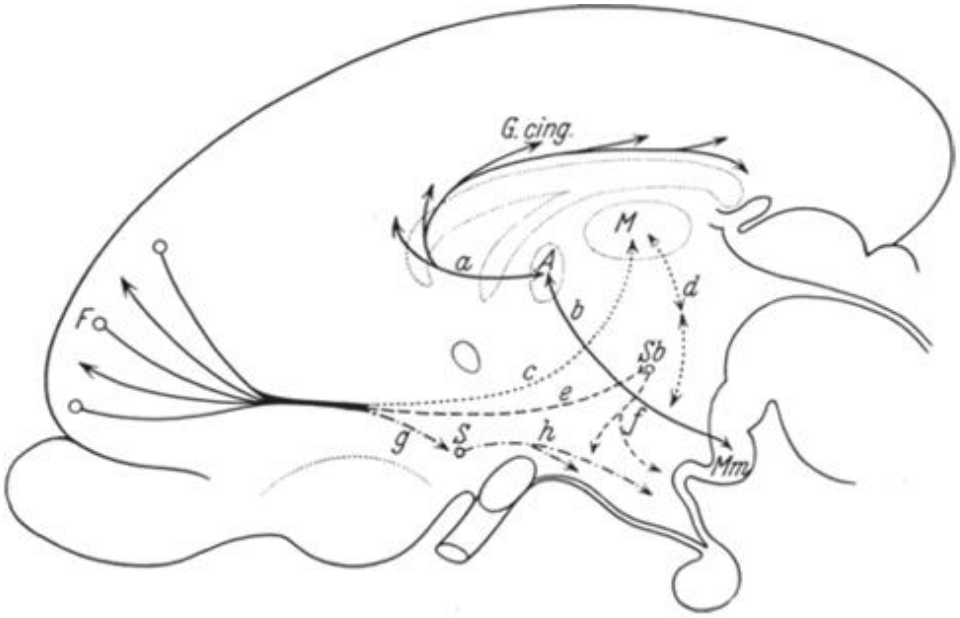

Figure 10. Connections of the tIypothalamus with the cortex. A Nucl. ant. thal., G. cing. Gyrus cinguli, b Tr. thalamo-mammillaris, F frontal cortex, M Nucl. med. thal., Sb sub-thalamic kerne, S Sept. pell., Mm Corp. mamillare, a--h connecting fibers. (After Le Gros Clark.)

A connection of the vestibular apparatus with the temporal or parietal lobe, which has been assumed several times, has not been proven and is very improbable according to the cortical architectonics. *Spiegel*, who has recently been referred to (*Stauder*), in his also otherwise dubious attempts, did not stimulate a vestibular center in the temporal lobe, but parts of the auditory cortex (see Brain Pathology, p. 951).

In the frontal brain (Figures 3 and 4), fields 6 a ß, 8, and 9 form the *terminal sites of frontopetal conductions*, but *motor pathways peculiar to the proprioceptive system* also originate from 6 ß and 8, some of which lead to the red nucleus and through it indirectly to the spinal cord, others to the pontine nuclei and indirectly to the cerebellum, and others to the thalamus, pallidum and subtantia nigra. Through them, the frontal brain acts on the lower reflex centers of the proprioceptive system in the diencephalon, midbrain, cerebellum and medulla oblongata, to which the cervical and positional reflexes, the co-movements and automatisms belong and whose disturbance leads to immobility with rigidity or hypermobility with chorea, athetosis, tremor, etc., in short to *myostatic* phenomena.

Of the *pathological disorders of the frontal system section*, the most important are the aggravation of trunk, head and gaze turns, nystagmus, spasms with eye, head or trunk turns to the opposite side, originating from fields 6 a ß and 8, as well as word mutism originating from a cortical district corresponding to field 8 at the base of F 3 (pars. basil.).

No trunk ataxia emanates from the frontal lobe, which was also confirmed by the presentations of *Claude* and *Barré* at the last international neurological congress in London in agreement with me[17]. In contrast, *pseudofrontal* ataxia occurs in tumors and injuries of the anterior frontal brain as a result of extensive pressure and remote effects on the midbrain, cerebellum and hindbrain, unless the vestibular apparatuses themselves are damaged in skull injuries.

Pseudofrontal are also the *perseveration of contraction*, *grasp reflex*, *Gegenhalten* and *grasping*, *catalepsy* and other motor disturbances sometimes observed in frontal brain lesions that actually originate in the brainstem. According to new experiments by *C. Richter*[18], grasp

[17] *Claude* et *Barré*: Revue neur. 64, 4 (1935).

[18] *Richter*, *C*. and *M*. *Hines*: Nerv. Dis. Monogr. Ser. 13 (1932).

reflex and grasping appear in monkeys after ablation of field 6, not of the frontal brain fields. In humans, however, field 6 is for the most part no longer located on the frontal brain, but on the precentral gyrus. Therefore, Richter's finding is consistent with my observation that minor damage to the c. a. or its thalamic radiation and minor paresis enhances the grasp reflex along with grasping and Gegenhalten, which in itself is due to thalamic injury.

In the case of the "compulsive grasping" referred to the posterior superior frontal brain by *Schuster*, there was also always a slight paresis of the hand concerned and anatomically a demonstrable damage of the c.a. or its thalamic radiation.

The *psychopathological manifestations* of the *frontal brain system* consist in *lack of drive*, in certain *apraxic* and *aphasic* disorders and in a peculiar *thought disorder* which I have called *alogical*.

The *lack of drive* may occur as a very general inactivity, including speech and thinking, or is more or less limited to individual areas:

1. to the *overall movements* of *walking* and *standing* and carrying out nature's call.

2. to *language*; as such it forms the well-known clinical picture of muteness with preserved or improved repeating abilities, automatic speech and affective speech, which *Wernicke* interpreted as transcortical motor aphasia and which can be better simply called *spontaneous muteness* or *name-muteness*, because on the level of spontaneous word usage it is just about the use of words *as designations*, as names.

3. to the movements of the mouth, tongue, chewing and swallowing.

4. to the mimic of attention and thinking and thinking itself.

The drive performance originates from the area of *Brodmann*'s field 9: in the F_1 for general movements, in the F_2 for mimic related to thinking and thinking itself, in the F_3 *ascendens* for *speech*. The foci or lesions were mostly bilateral, for speech and thought sometimes only left-sided (Figure 3).

Restriction of drive is often accompanied by a pathological increase in the perception of strength and effort. The drive acts as an exciter against the other cortical motion devices and the infracortical auxiliary sites (red nucleus, pons, nigra, etc.). The frontal drive deficiency is as such free from rigor, flexibilitas, catalepsy and other abnormal states of tension originating from the brain stem. This has been assured only by the war experiences.

In the case of progressive brain and mental diseases, such as *senile* and *schizophrenic* diseases, the *lack of linguistic drive* increases only very gradually to a disorder comparable to the focal aphasias, whereby the *linguistic vocabulary shrinks more and more* and certain words are used more and more *monotonously*. Our patient Gretchen H. had, when I met her in an already advanced state of her *Pick's cerebral atrophy*, only four small stories, e.g., the one about a train ride, with which she thought of everyone and at every opportunity; later there were only a few words, in the end a few croaky sounds. In the case of a *paranoid schizophrenic*, the progressive impoverishment of language manifested itself in the fact that she referred, for example, to every useful activity as "build," to every kind of expansion as "run" and to every well-meaning person as "father." The restriction of the set of names is thus accompanied by an expansion of the scope of meaning of the *remaining names*.

The frontal brain disorder, which used to be known as *motor agrammatism* only in the field of language and which depends on the most anterior part of the motor speech area (pars ascendens or triangularis of the F_3), penetrates even further into the psychic, in the sense that this place or its fiber system of projection, association and callosal fibers must also be injured if agrammatism is to occur in a motor speech disorder. As a permanent disorder, agrammatism -- like other aphasias -- remains only if the right motor speech area is also affected or the callosal connection between the two is interrupted.

The motor-grammatical performance is the formation of regular *word sequences*, whereby the words are subject to certain variations and compositions. It is an *active, formative performance*, even if it uses to some extent memory-acquired sentence formulas. An example of *agrammatism* in a *catatonic* is the following picture description.

On the first picture you see a mother with three children at a table, the two girls are busy writing, the boy is screwing on the burning lamp, over which he holds a written sheet to dry. The mother raises her finger in warning. On the 2nd picture the lamp has fallen over and

exploded, the older girl flees with burning dress, the boy cries, the mother has taken the youngest girl in her arms and turns to escape.

Picture 1. that is table with children... (the writing girl?) a mother... (the boy?) is to light... properly on... (the other girl?) is right of the mother.

Picture 2. (Mother with the child on the arm?) Lady and son. (Fleeing girl in oblique posture?) Lady is lying… (Crying boy?)… Son is also a pity.

A performance *analogous to the grammatical word sequence*, however, also exists in the field of *action*. Also, the composite actions need the temporal and meaningful sequence of single movements. And this requires not only kinesthetic and optical stimulation and order emanating from the parietal lobe, but an effort building up, which, as war experiences and focal diseases have shown, is performed by the *frontal brain*. There is a *frontal apraxia* of the *sequence of actions*, in which the sequence of partial movements is not confused as in the parietal form, but does not come about at all, does not go beyond the first steps, or is completed in a shortened and coarsened manner, e.g., one drinks immediately from the pitcher instead of pouring into the cup and drinking from the cup.

However, frontal apraxia of the action sequence occurs also and even more frequently than in local injuries and focal diseases in *frontal brain atrophies*, in *Pick*'s or *Alzheimer*'s disease, in *focal paralyses*, and only with the help of such observations I succeeded in working out the clinical picture which is related to the area of *Brodmann*'s field 10 on the F_1 and F_2. These were always bilateral brain changes. Also, in the case of impaired movement in *schizophrenics* and especially in *catatonia*, not only frontal lack of drive, but also apraxia of the sequence of actions must be considered; however, it is very difficult to distinguish them from psychomotor symptoms of the brain stem, such as akinesia with flexibility or catalepsy, Gegenhalten and negativism.

Finally, brain injuries, focal diseases and brain atrophic processes (*Pick*'s and *Alzheimer*'s disease, paralysis) have taught me that *thinking from the frontal brain* can also be disturbed not only as a mental activity drive but also as a *formative, constructive performance*. The establishment of mental relations is then missing or remains imperfect, derailments and entanglements, however, recede. The resulting *alogical* thought disorder is the motor counterpart of the occipital and parietal sensory and at the same time paralogical thought disorder. *Thinking*, as well as language and acting, are of *dual nature* comprising sensory (receptive and ordering) and motor capacities, and thus alogias, as location-bound brain disorders, may stand side by side with the older sisters of aphasias and apraxias.

Only the disorders of thought formation and thought drive, which also include certain arithmetic disorders, originate in the frontal brain. Memory failures, memory disorders, confabulations, drowsiness and insomnia, which one tried to connect with the frontal brain (*Berger*, *Woerkom*, *Goldstein* and others), have nothing to do with it and are based on distant effects on deeper parts of the brain or on concomitant disorders of such parts.

The *brain location of frontal alogia*, according to the evidence of local injuries and diseases, is the area of field 46 on the middle F_2. The left hemisphere also has a predominance for thought formation.

The occurrence of "mental disorders" in bilateral *frontal brain tumors*, although not described in detail, has recently also been confirmed by the French brain surgeon *Cl. Vincent*[19]. In one case, the tumors were located in the two middle quarters of F_1 and F_2 on each side, which is also consistent with my findings. In another patient, there was a huge glioma of the left frontal brain, which had also damaged the adjacent part of the right frontal lobe. Extensive unilateral resections of the frontal brain remained asymptomatic.

The *alogical thought disorder* participates considerably in the presentation of actual *mental illnesses*. The following examples come from *paralyses*, whose preference for the frontal brain is well known. (Thought-formation from table, lamp, chair): "The table is used for writing. The lamp serves to illuminate, the chair to sit on. Fortune and glass soon break, alas!: "Fortune is fortune, glass breaks, then broken." (Note the accompanying agrammatism!)

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[19] *Vincent, Cl.*: Dtsch. med. Wschr. 1936, 2.

Alogical thought disorder in *hebephrenics* is shown by the following examples. (Hunter, hare, field): The hunter is running across the field and sees the hare and the hare is also running... (What does the hunter have to do with the hare?) Field, hare... the hunter runs across the field and sees the hare. Picture window promenade: This is a festive card, a draft, a man... a woman with a child.

III. The Enteroceptive Systems

On the inner sensations of the bodily openings, the sexual organs, the intestines, glands and vessels our inner experiences are built up. While we can sharply separate the individual exteroceptive types of sensations from each other and follow their peculiarities up to their highest psychic formations and complexities, the individual types of inner sensations are more difficult or even impossible to distinguish from each other, but flow together into general states. Therefore, we must refrain from tracing individual enteroceptive systems, like, for example, that of the heart or lung sensations or single abdominal organs, separately from the sensory organs up to the highest mental emanations, but we must stick to the higher mental entities, which we are better able to distinguish from each other. According to this, the enteroceptive processes are divided into three large groups:

1. The group of the *person*, the drives, aspirations and impulses.
2. The group of the *inner body* and feelings.
3. The group of *enteroceptive effects* on extero- and proprioceptive processes.

The *neuropathological* parts of the enteroceptive systems coincide with the disorders of the *autonomic* (sympathetic and parasympathetic) nerves and their conduction pathways outside and inside the spinal cord.

The *cerebro-pathological* enteroceptive disorders are those of the vegetative cell groups and fiber bundles from the bottom of the 4th ventricle to the hypothalamic area of the diencephalon. This includes the central *metabolic disorders* and the related desire disorders of *thirst* and *hunger* (diabetes insipidus, hyperorality), as well as the decrease, increase or alteration of the *sexual drives*, etc. Furthermore, according to *Ranson*[20], electrical stimulation of the hypothalamus can cause *sympathetic excitations*, which correspond to those of *anger* and *fear*: Constriction of small arteries with increase of blood pressure, acceleration and deepening of respiration, inhibition of gastric and intestinal movements, excretion of adrenaline, secretion of sweat and saliva. If the irritation continues, the animal becomes wild, tries to free itself, scratches, bites, hisses, bangs with its tail. On the other hand, stimulation of a site located just in front of the hypothalamus, in the vicinity of the anterior commissure, results in *parasympathetic stimulation*, bladder contraction, slowing of pulse and respiration, lowering of blood pressure. On the other hand, *Ranson* and his co-workers were able to achieve a *weakening of the emotional excitability* by circumscribed destruction at the mammillary bodies and their nearest surroundings. Monkeys operated on in this way first became *dormant* and then were transformed into real pet animals; they were indifferent and fearless and allowed themselves to be taken and stroked.

Accordingly, the *inner sensations* arise not only directly from stimulation of the sensitive terminal bodies on the inner surfaces of the hollow organs and their oral and aboral openings, but more frequently indirectly from the movements and activities of the internal organs, vessels, and glands stimulated from the autonomic centers. Originated just like that, the internal receptions -- such as the exteroceptions and proprioceptions of the skin and musculoskeletal system -- are fed to the diencephalon (Figure 9) and, according to *Wallenberg*'s investigations, reach in part directly the thalamus (medial nucleus area), in part the *hypothalamic* and *subthalamic* nuclei -- the mammillary bodies, the zona incerta, and *Forel*'s field -- from where they can also reach the thalamus through the Tr. mammillo-thalamicus, the thalamic bundle H_1, and through ventricular fibers.

[20] *Ranson*: Publications from the Institute of Neurology, Bd. 6 u. 7. Chicago 1934/35.

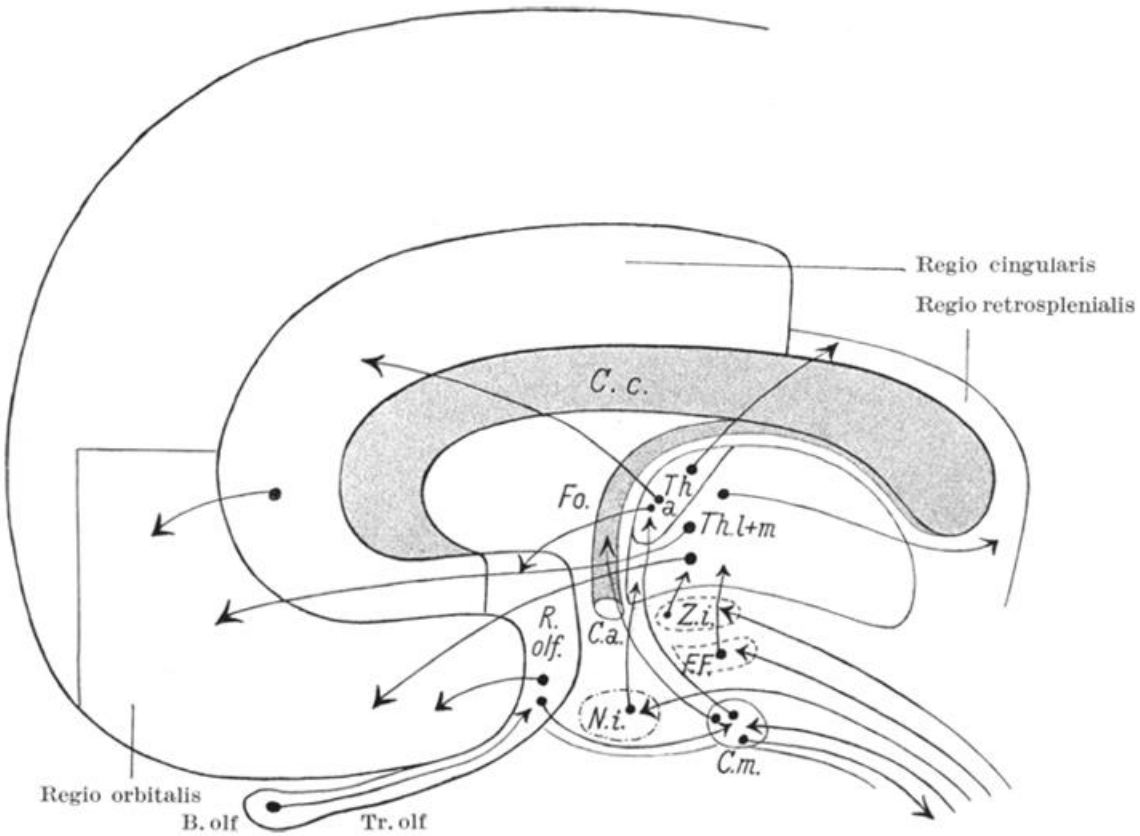

Figure 9. Fiber connections of the regioncs orbitalis, cingu]aris, and retrosp]enialis. C.a. Commisura anterior, C.c. Corpus callosum, C.m. Corpus mamillare, Fo. Fornix~ tha. Nucl. ant. thalami, Th.Lu.m. Nucl. lateralis et med. thalami, F.F. Forel's field, Z.i. Zona incerta, N.i. Nuclei infundibularis, B.olf. Bulbus olfactorius, Tr.olf. Tractus olfactorius.

The anterior thalamic nucleus, however, is connected to the cingulate gyrus[21] and to the retrosplenial region[22] by ascending and descending fibers, and the medial thalamic nucleus sends fibers through the anterior thalamic radiation to the frontal and orbital brain and receives them from there *(Le Gros Clark*, Figure 10).

Therefore, *cortical organs of the enteroceptive systems* must be located in the *cingulate gyrus* and the *frontal brain* (in the broadest sense).

The close relations between autonomic centers and performances and the endocrine glands are shown not only by the spatial and functional contact of the *pituitary* and *pineal glands* with the diencephalon, but even more by the secretory activity of individual vegetative cell groups themselves (nucl. supraopticus and paraventricularis) (*Scharrer*[23]).

In the *psychopathology of the* ***person***, it should be noted that the person (or character) is composed of sensory experiences (dispositions) and motor expressions (volitions). In another direction one can distinguish: The “autopsyche” [Self-Ego; Selbst-Ich], which is based in itself; the “koinopsyche” [Community-Ego; Gemeinschafts-Ich], which consists of the moral attitudes and behavior patterns of communal living; and the individual's integration into the world as a whole, the “holopsyche” [Welt-Ich oder religiöses Ich; World-Ego]. While the lower ego level of instincts, strivings and impulses cannot be disturbed higher than from the diencephalon, the self, communal and world ego has a diencephalic and a cortical level, which according to the war observations is located in the basal frontal brain, the *orbital brain*.

As for the *psychopathology of the person*, the war observations have shown that in the case of *orbital brain injuries* there were deficiencies in the community spirit occurring,

[21] *Le Gros Clark*: J. ment. Sci. 1936.
[22] *Takeuti*: Fol. Psychiatr. et neur. jap. 1, 1, 2 (1933/34).
[23] *Gaupp* und *Scharrer*: Z. Neur. 153 (1935).

disloyalty and deceitfulness, fraud and theft, insubordination and agitation, while in the case of others injured in the same area the *ego-self* was degraded in the sense of immaturity, boyishness, foolishchildishness, witzelsucht, and in the field of the *will* there was a tendency toward instability and addictive behavior. The injuries frequently penetrate the orbital brain through the *eyes* or the nasal root (Figure 11).

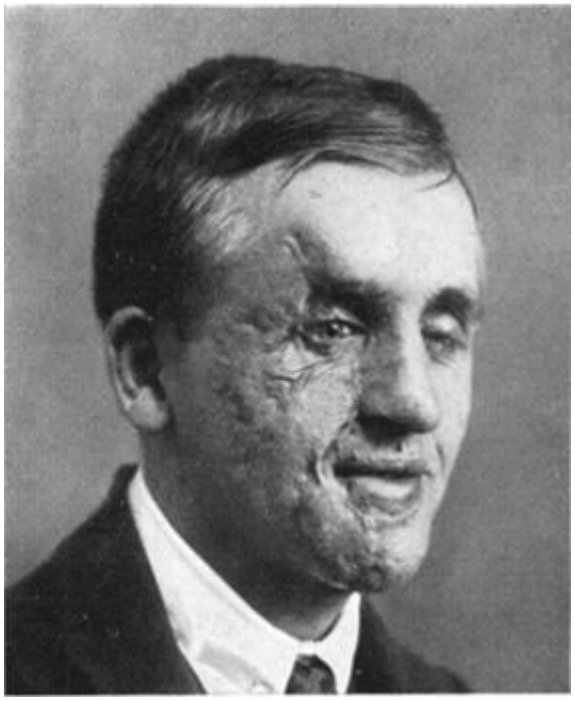

Figure11. Orbital brain and eye lesions. (R.glass eye!). (Gehirnpathologie, Abb.395)

The old observations by *Leonore Welt* thus received a late confirmation (Figure 12) after much contestation, and since then *Guttmann*, *Spatz*, and *Grünthal*[24] have added similar observations in cerebral lesions and *Picks*'s cerebral atrophy.

[24] *Grünthal*: Über die Erkennung der traumatischen Hirnverletzung. Berlin: S. Karger 1936.

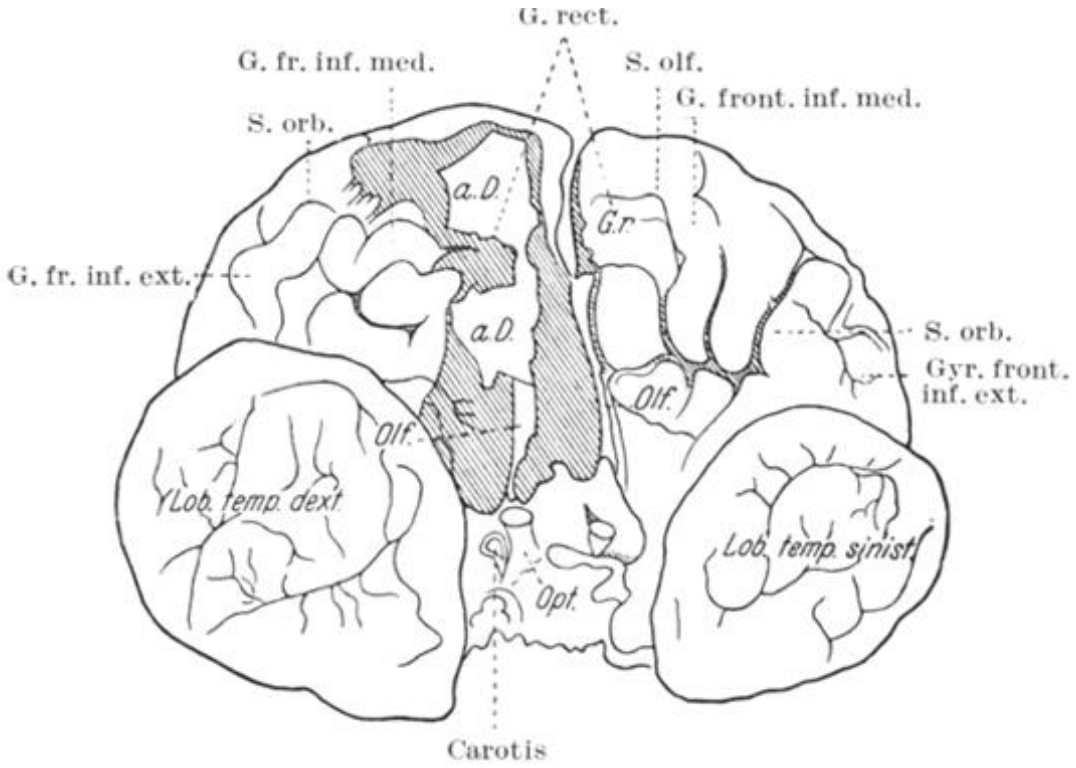

Figure 12. Orbital brain injury with character change. Pieces from the dura. Injured areas hatched. (After L. Welt.)

The not rare kind of psychopaths, more correctly *characteropaths*, whose futile nature is combined with immaturity and boyishness as well as with a higher intelligence weakness of alogical kind, from an anatomical standpoint, probably consists of individuals with orbito-frontal defects. However, encephalitis has shown that the person can also be disturbed from the diencephalon. Also, in the case of personal changes in *schizophrenics*, especially in *hebephrenics*, it would be necessary to search for cell foci in the diencephalon and orbital brain.

In the case of personal disorders originating from the diencephalon, in addition to the symptoms of failure and even more frequently without them, there are also *temperamental increases* or *decreases* in the excitability of the associated feelings and activities emerging, *biotonic* disorders in the sense of *Ewald*. Elevations of the *self* with arrogance and bragging was only occasionally found in brain-injured persons. Such *expansive* states are most frequent and best known in paralysis, where they are not coincidentally so often accompanied by character deterioration.

Underlying the paranoic states are increases and decreases of *communal feelings*, often in conjunction with those of a *religious* nature: *ecstatic* states with excessive charity, sacrifice and religious blessing – or persecutory upsets with suspicious and hostile infection of communal feelings.

Only occasionally this is seen in brain injured patients, the more frequent are persecutory conditions in involutive, senile and arteriosclerotic brain lesions. But also, in *psychoses without permanent anatomical damage*, in circular, degenerative and symptomatic diseases, the same sites in the diencephalon must be the seat of the then compensable brain disorders. And paranoic psychopaths with a tendency to over-valued ideas and querulous or sensitive reactions are likely to be malformed in the diencephalic fundamentals of their communal feelings.

Brain-injured persons, who suffered character changes, often showed, especially immediately after the wounding, also disorders of drive, especially libidinal rage and defensive outbursts, sometimes also sexual overexcitabilities and hyperorality. In such cases, the anterior injuries spread through the orbital brain to the anterior *diencephalon* and the

hypothalamus. Also, *tumors* of the diencephalon can cause impulsive restlessness (Figure 13).

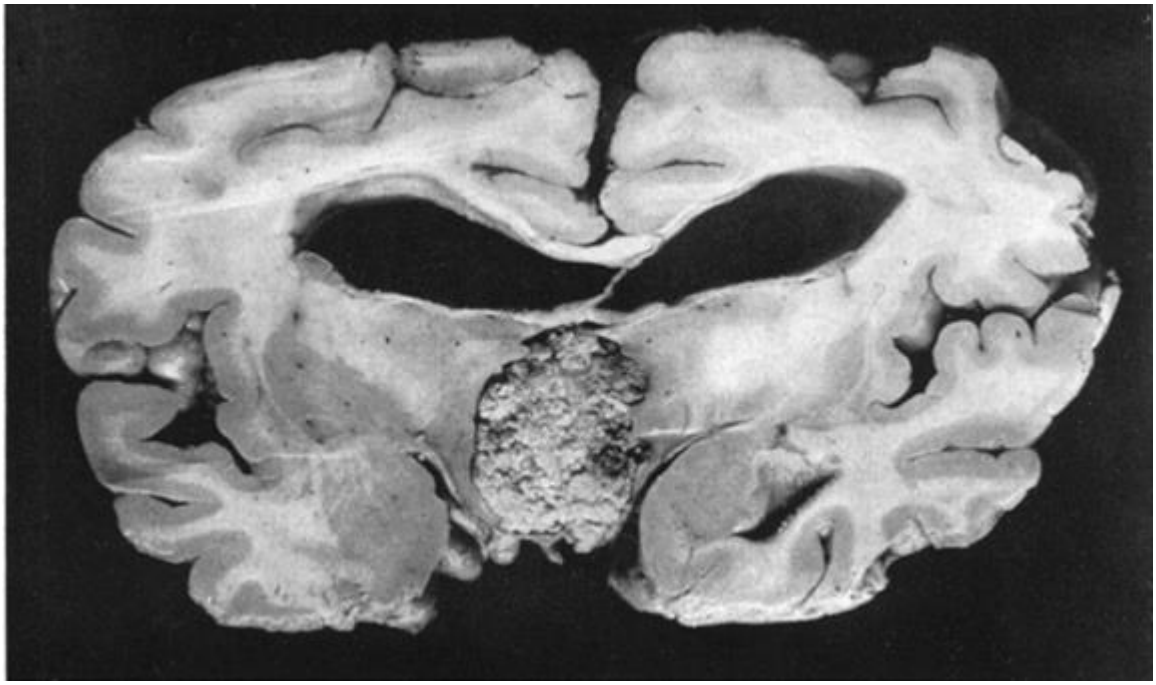

Figure 13. Tumor of the diencephalon (3rd Ventr.) with libidinal excitation (anger, defense, screaming, yelling, scolding). (Brain pathology, Figure 404.)

More rarely, a *reduction of libidinal behavior* up to dull-stuporous behavior occurred with profound brain injuries. Then the brain wounds were mostly over the temporal, parietal or occipital lobes, and their deep effects were on a *posterior region of the diencephalon*. Similar to the drives, the related *urges* and *compulsions* are to be judged in terms of brain pathology, especially since they are often pathologically increased in *encephalitis*. *Meyer*[25] found severe destruction of the medial thalamus in an encephalitic condition that had become malignant with the most violent compulsive excitations.

From here it is only a step to the *strivings and impulses* which are less often pathologically altered in brain-injured and focal patients, but which, on the other hand, dominate the symptomal characteristics of *catatonia*, *motility psychoses* and certain *symptomatic psychoses*. The strivings in the sense of persistence (stereotypy), repetition (iteration), duration (catalepsy), imitation (echo phenomena), obedience (command-automatism), affirmation and negation (negativism) originate like the drives from emotional experiences, and the same applies to the playful *impulses*, which, pathologically disturbed, move between the opposites of a hyperkinetic increase and an akinetic dampening of the motor temperament.

By approaching the brain pathology of psychomotor disorders from *another* side, namely from *myostatic* disorders (chorea, athetosis and rigidity), I also reached the *diencephalon* on the basis of *focal diseases with psychomotor manifestations*. However, it was found that not only the *thalamus* and the subthalamic area, but also the ventral basal ganglia, especially *caudatum* and *pallidum* (externum) were involved (Figures 14 and 15).

[25] Arch. Psych. 80 (1927).

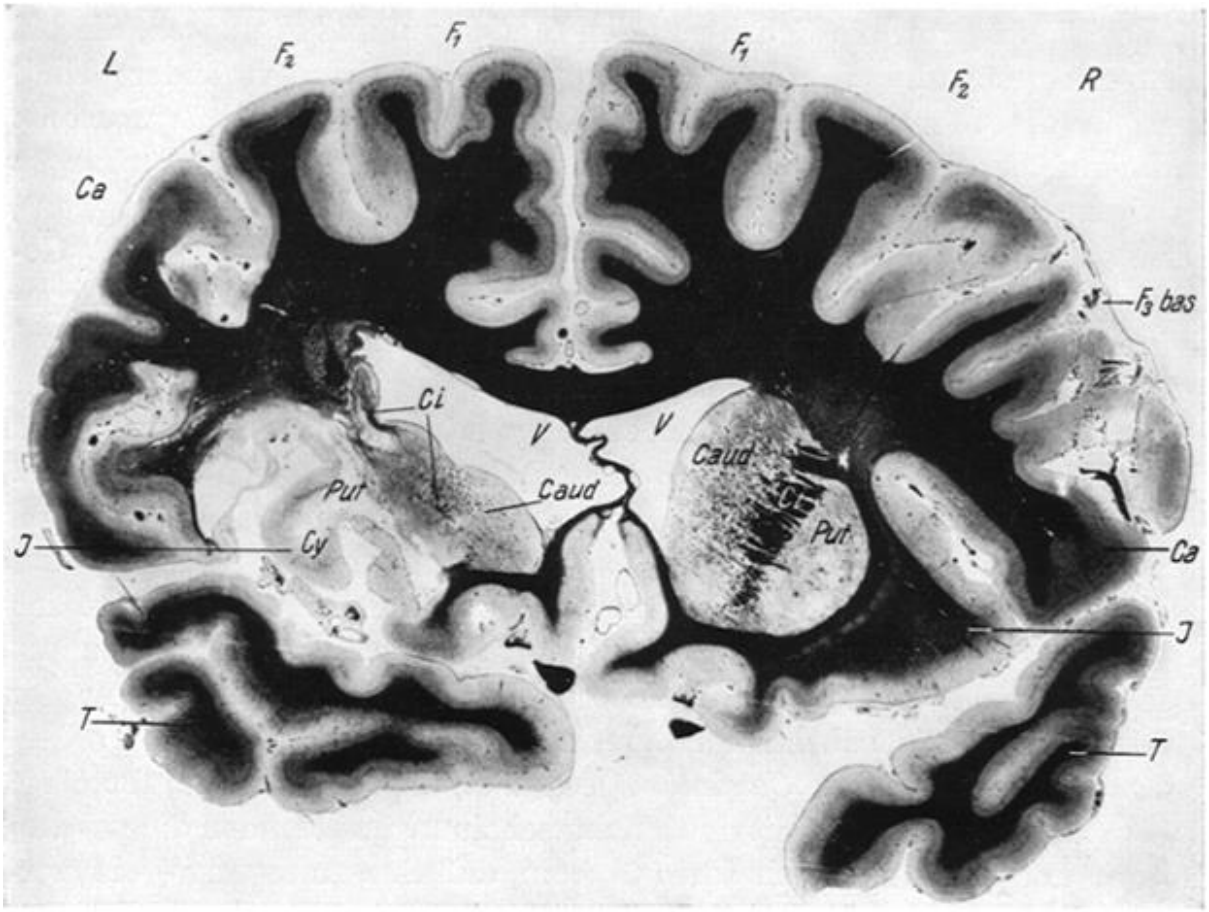
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Figure 14.

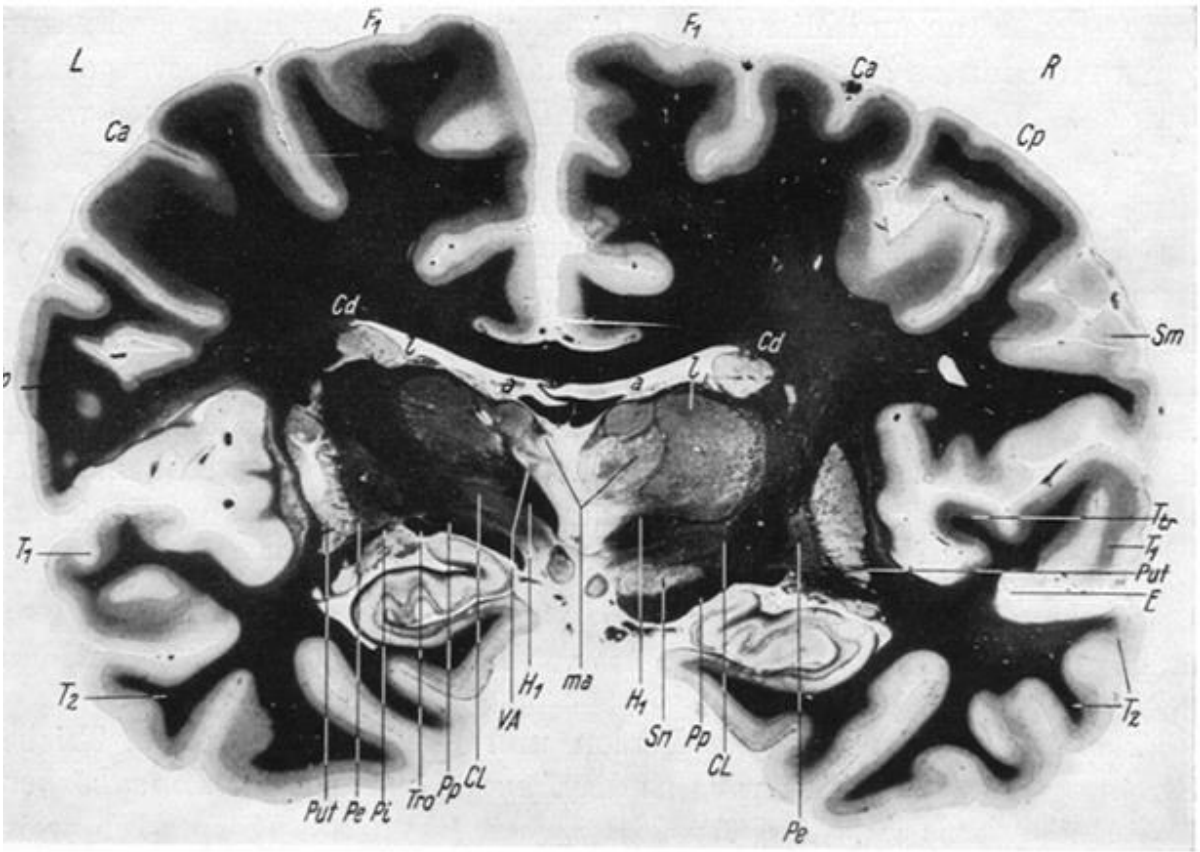
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Figure 15.

Figures 14 and 15. Psychomotor (parakinetische) excitation. Softening foci in left caudatum, putamcn uud thalamus, right dilated perivascular spaces. (Brain pathology, Figures 360 and 363.)

The psychomotor processes also match with the drive phenomena in that their stimulation -- hyperkinesis, parakineses, iterations, stereotypy -- in focal diseases originated from anterior stem brain sites (caudatum!), whereas akineses with Gegenhalten and Mitmachen, catalepsy and proskinetic behavior were elicited from more posterior parts (thalamus, pallidum). Brain injured subjects with akinesia, Mitmachen or catalepsy were more often wounded in the temporal, parietal, or occipital lobes than above the frontal brain, so that deep actions were more likely to affect posterior diencephalic sections. According to *Ranson*, *catalepsy* and sleep can be caused in the cat by injuries in the area of the mammillary bodies. According to the description, however, it was not a catalepsy in the narrower sense, but an *akinesia with flexibilitas* ("plastic malleability"), partly with a persistent grasp reflex, i.e., probably with grasping and Gegenhalten.

In animals with very pronounced catalepsy and somnolence, the fibers running dorsolaterally from the mammillary bodies and descending from the hypothalamus were destroyed. However, in these experiments it is not possible to decide with which of the destroyed structures sleep was related and with which akinesia. The same kind of akinesia is caused, according to observations of myself and *Fernandes* by bulbocapnine intoxication.

After the disorders of the person and its related drives, aspirations, etc., we turn to the *psychopathology of the* ***inner body*** *and the feelings*. Pointing to failure of the body-ego, one brain-injured person complained that his spinal cord was injured and that pain wandered from the spinal cord to the brain; another one: the blood in the brain was spoiled, the brain was dried out, the abdomen was dead. More frequent in brain-injured patients were a *general physical hypersensitivity* and *nervousness*, which easily turned into impulsive defensiveness, or temperamental sinks and lifts of the whole physical sensation: *dysphoria* and *euphoria*. Since the brain wounds were almost always above the orbital or frontal brain, one could assume that the inner body -- as well as the person -- possessed a cortical organ there. However, autopsy findings and accompanying clinical symptoms speak more in favor of the diencephalon as the seat of such changes.

Similarly, the *pure feelings* close to the body-ego were disturbed in brain-injured persons: rarely affective defects with unbalanced, fluctuating emotional state or with pathological laughter and crying, more often *temperament shifts to the cheerful or unhappy side*. The brain wounds were widely scattered along the pallium, although with some preference for the frontal and orbital brains; but more essential was probably the *depth of the injury* and the involvement of the *diencephalon*. In one case, a bomb fragment that had penetrated the occiput was detected radiologically at the sella turcica. Focal diseases teach us that affective instability, pathological laughter and crying can originate from the *thalamus*, affective instability from the medial nucleus and compulsive expressive movements from the median center[26]. These insights gained from patients with brain injuries and focal disease also serve to understand the peculiar *hypochondriacal* and *affective* disorders found in *paralyses* and *schizophrenias*. The paramount role of affectivity in the circular, as well as in degenerative and hysterical psychoses, need not be particularly emphasized.

A *third group* of disease symptoms involves the ***effects of internal performances on exteroceptive and proprioceptive processes*****.** First, the disturbances of *self-experience*. One of my patients no longer seemed to experience the parts of his body as his own property. When he was supposed to show his nose, forehead, neck, right eye, he pointed to the corresponding parts of the examiner's body. His left half of the body was not present for him at all; if he should show the left hand, the left foot, he showed the corresponding right parts of the body. Where is the left hand? "It's not there." When it was shown to him: "Oh, that's

[26] *Gonzalo* u. *Kleist*: Über Thalamus- und Subthalamussyndrome und die Störungen einzelner Thalamuskerne (in press).

nothing, that's the little finger." The prompting to movement was also unable to reach the left side of the body, or only with great effort and assistance. This and an accompanying dyspraxia of the left hand indicated softening of the *corpus callosum*.

By the way, the patient was also somewhat apraxic on the right side, and rarely he showed his left hand instead of the right one. These minor signs of right-sided apraxia and of right-left disorientation are suggestive of disease of the *left inferior parietal lobe*; but this does not explain the loss of self-experience for the left half of the body and partially for the right side and for the unpaired parts of the body.

The disruption becomes understandable only by the assumption of a *separation between the tactile image of the outer body* in the left inferior parietal lobe and the *representation of the inner body*, whose cortical organ we assume in the *cingulate and retrosplenial gyrus* and thereby also presuppose a preponderance of the left cingulate gyrus. A focal point in close proximity or perhaps within the left *cingulate gyrus* itself could explain the disease symptoms concerning the right half of the body and the unpaired body parts; in addition, a softening of the *corpus callosum* would be necessary to cause the loss of self-experience for the left half of the body, including the left-sided lack of drive and the left-sided dyspraxia (Figure 16). In fact, the corpus callosum and cingulate gyrus, which are fed from the same blood vessels, usually soften together.

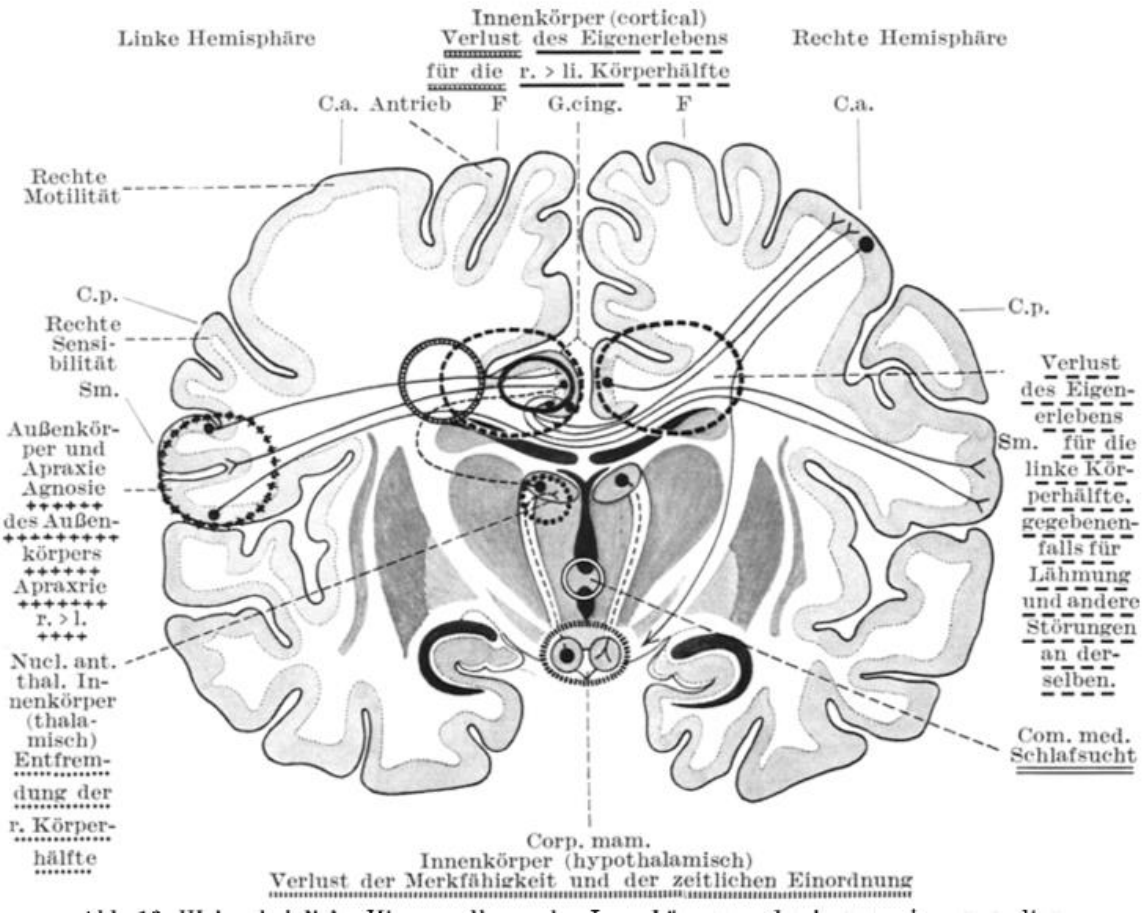

Figure 16. Probable brain bases of the inner body and its related disorders (using a brain chart by Müller-Spatz).

In our case, there are no anatomical findings proving these assumptions, but it can be shown that in many cases, that the *cingulate gyrus* and the *corpus callosusm* were injured, which have been described by *Anton*'s *syndrome* of "loss of self-awareness for disturbances"

(paralysis, etc.). Obviously, *Anton*'s syndrome is only a special case of *loss of self-awareness of one's own body*, i.e., of deficiencies of one's own body[27].

Cortical disturbances of self-experience have another psychopathological meaning and are included in some signs of physical or personal disorientation observed in diffuse arteriosclerosis, in paralytics and seniles, in involutive diseases and in schizophrenics.

Even greater, however, is the general psychopathological significance of *related disorders* that do not originate in the cerebral cortex but in the *diencephalon* and the substrates responsible for body- and self-ego located there. I mean the phenomena of *alienation* (depersonalization); for the *loss of vividness* of perceptions and ideas peculiar to alienation is accompanied by an embarrassingly felt *diminution of the sense of self*. In the field of action, alienation corresponds to the loss of the volitional sense of self, of *free will*; all action seems to run down automatically or to be caused and influenced by other people. Therefore, experiences of manipulation of actions or thoughts, thought withdrawal and the like, which are so frequent in schizophrenics, also belong here. Possibly, also in these phenomena, there is no separation between the diencephalic ego system and the extero- and proprioceptive processes, but a malfunction or deficiency of the diencephalon itself.

Alienation and paranoic experiences predominate in schizophrenic, circular, degenerative, epileptic and hysterical diseases, but are by no means foreign to focal diseases and brain injuries. In a tumor of the temporal lobe observed by me and in other local conditions, the seat and accompanying symptoms clearly pointed to the diencephalon. *Pötzl* and *v. Stockert* have described alienation in thalamic tumors. Also in paralysis, encephalitis, in senile and presenile diseases such phenomena are not uncommon, as our own[28] observations and those of *Fünfgeld*[29], as well as the compilation of *Haug*[30] prove. The same is true for the disturbances of other *impression qualities* related to alienation, of familiarity, of reference and *meaning* with perplexity or revelation and inspiration, which, however, are partly not based on a reduction but on an increase of influences emanating from the diencephalic ego on extero- and proprioceptive experiences.

In other disease symptoms *influences* are disturbed which work f*rom the ego or from enteroceptive processes on the imaginary life* in the broadest sense. It is not only that sensory impressions leave traces in the soul and accumulate a memory treasure from which ideas can resurface, but *I* perceive, *I* memorize my perceptions, *I reflect* on them and *imagine them again*. Pathological changes of these ego-like sides of the imaginary life are the disturbances of the *retentiveness* (of the time marking of the experiences), certain *delusional perceptions* and the disturbances of consciousness (marked or moderate disruption, perseveration and fantasy). Possibly not only retentiveness (Gamper), but the whole subjective side of imaginary life is related to the system of *mammillary bodies*, anterior thalamic nucleus and cingulate gyrus, which is also likely to be the carrier of the body-ego (Figure 16). Within this system, enteroceptive impressions can meet with those supplied by the external sensory organs and with proprioceptive sensations both in the thalamus and in the cerebrum, if one thinks of the association fibers striving from the cingulate gyrus in all directions.

Whether this also explains the case of *Mabille* and *Pitres* with permanent memory loss in the presence of bilateral softening in the frontal white matter previous to the caudate nucleus head is very doubtful, since only macroscopic brain findings are available and nothing precise is said about the hypothalamus[31].

Overall, this is the creation and mastery of a *mental world independent* of the ever-changing perceptions and movements. In any case, experience in brain-injured patients and in encephalitis has proven the general association of memorization, hallucination, perseveration and confabulation with midbrain damage, and these insights have already been useful to us in the focal diagnosis of tumors of the diencephalon and adjacent cerebral parts,

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[27] More detailed s. Brain Pathology p.1223 ff.

[28] *Kleist*: Arch. f. Psychiatr. 103 (1935).

[29] *Fünfgeld*: Nervenarzt 9 (1936).

[30] *Haug*: Die Störungen des Persönlichkeitsbewußtseins. Stuttgart 1936.

[31] *Mabille* et *Pitres*: Revue Med. 1913.

especially the temporal lobe. For the understanding of aphasic and other cortical disorders, the separation of the perseverative symptoms, which are so often mixed with them, means an extraordinary clarification, if one thinks of the repeated and always futile attempts to understand perseveration as an aphasic disease symptom similar to paraphasia.

The *last* psychopathological phenomena I have to discuss are different *interrelations of mental processes of exteroceptive and proprioceptive origin under the dominating influence of ego-related, enteroceptive performances*.

The onset of unconsciousness in case of injuries of the hindbrain in the area of the vegetative vagus nucleus presupposes that from this part of the brain a primordial connection of external, individual and internal mental experiences is controlled, which we may regard as a *primordial consciousness* [Urbewusstsein]. At this total experience there are fluctuations between clarity of consciousness and obscuration of consciousness, which probably depend on alternating sympathetic and parasympathetic excitations, i.e., precisely on enteroceptive processes.

At a higher level, in the *diencephalon*, we encounter related mechanisms in the sympathetically and parasympathetically controlled *interactions* of *waking* and *sleeping*. Foci that interfered with this regulation system were located in my observations, consistent with the stimulation experiments of *Hess* in the frontal areas of the *middle commissure*, which also contain the mammillary bodies and the mammillothalamic tract (Figure 17). *Ranson* saw sleep occur in experimental lesions of the diencephalic base in the area of the mammillary bodies.

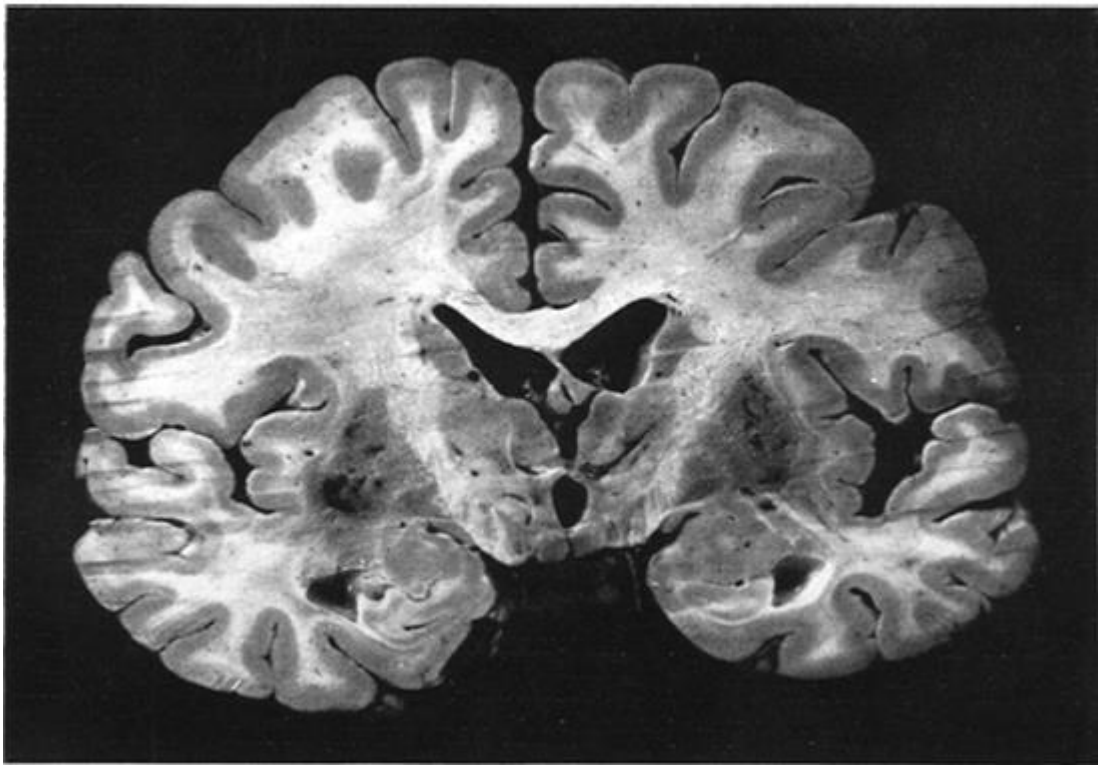

Figure 17. Sleep addiction in softening foci in the cavernous gray and thalamus with comm. med. (Brain pathology, Figure 424.).

The core of unconsciousness and sleep is surrounded by absences, twilight states, narcolepsy, dreamy states, i.e., symptoms which are frequent in *brain tumors* and which originate not only from the diencephalon, but also from the closest *temporal lobe*, and from the adjacent lower parietal lobe, by which they may enable the localization of tumors, especially in the right temporal or parietal lobe, which is otherwise symptomless. Outside of brain pathology in a narrower sense, these symptoms are important in *epilepsy* and the various epileptoid diseases and conditions. *Seizures* may also be triggered by the brainstem and may also be related to altered autonomic nervous control.

The more static total states of consciousness and wakefulness and their opposites, bringing about a selection from a mental whole, are faced by dynamic processes of paying attention and suggestion. They are also dependent on vegetative or ego-related performances. *Attentiveness to sensory impressions* and the corresponding adjusting movements apparently emanate from the tectum of midbrain; nonattention and overattention, may it be by tactile impressions from one half of the body or by auditory or facial impressions, are observed in focal injuries and diseases of this area. Related to mindfulness is sensory startle reaction. Different from mindfulness, also in its physiological conditions, is *attention* in the narrower sense, which is bound to a considerable extent to the *diencephalon* and, as a regulator of thought, action and speech, finds its expression in flight of ideas and inhibition of thought, incoherence and thought paralysis.

A different *selection* takes place under the *stirring* and *displacing* influences of *suggestibility*, which is connected with the diencephalic body-ego. Brain injuries with damage to the diencephalon sometimes make the affected person suggestible to a heightened degree, making him react hysterically without this being psychologically conditioned, as in constitutional hysteria. I must limit myself here to hints and refer to the more detailed explanations in my brain pathology[32].

By walking the various paths of the extero-, entero- and proprioceptive systems, all of which have led from a neurological through a cerebro-pathological into a psychopathological realm, the intrinsic relationship of the three parts of each system and the importance of one to the other has come clearly to our attention. As necessary as the boundaries between the three areas are for factual and practical reasons, they are only *conditional* boundaries, not walls, but barriers, which can and must be raised at any time when certain demands are made. *Such* a requirement is the striving for *complete clarification of psychopathological phenomena*, which will only be achieved when all mental signs of illness will *also* be understood *in terms of brain pathology*, and all normal mental processes will also be understood in terms of brain physiology.

In this direction, as you have convinced yourself, substantial progress has been made recently. There is no field of psychopathology today in which brain pathology has not gained a firm foothold and advanced to a greater or lesser extent, and the *active* benefit derived from it for the recognition and treatment of brain diseases in the broadest sense is increasing with every step. It is therefore necessary and worthwhile to continue along this path and to unite the forces of many for this task.

[32] The mental processes supported by the diencephalon are also summarized there as *phenomena of being* (p. 1295 and 1311 f.).