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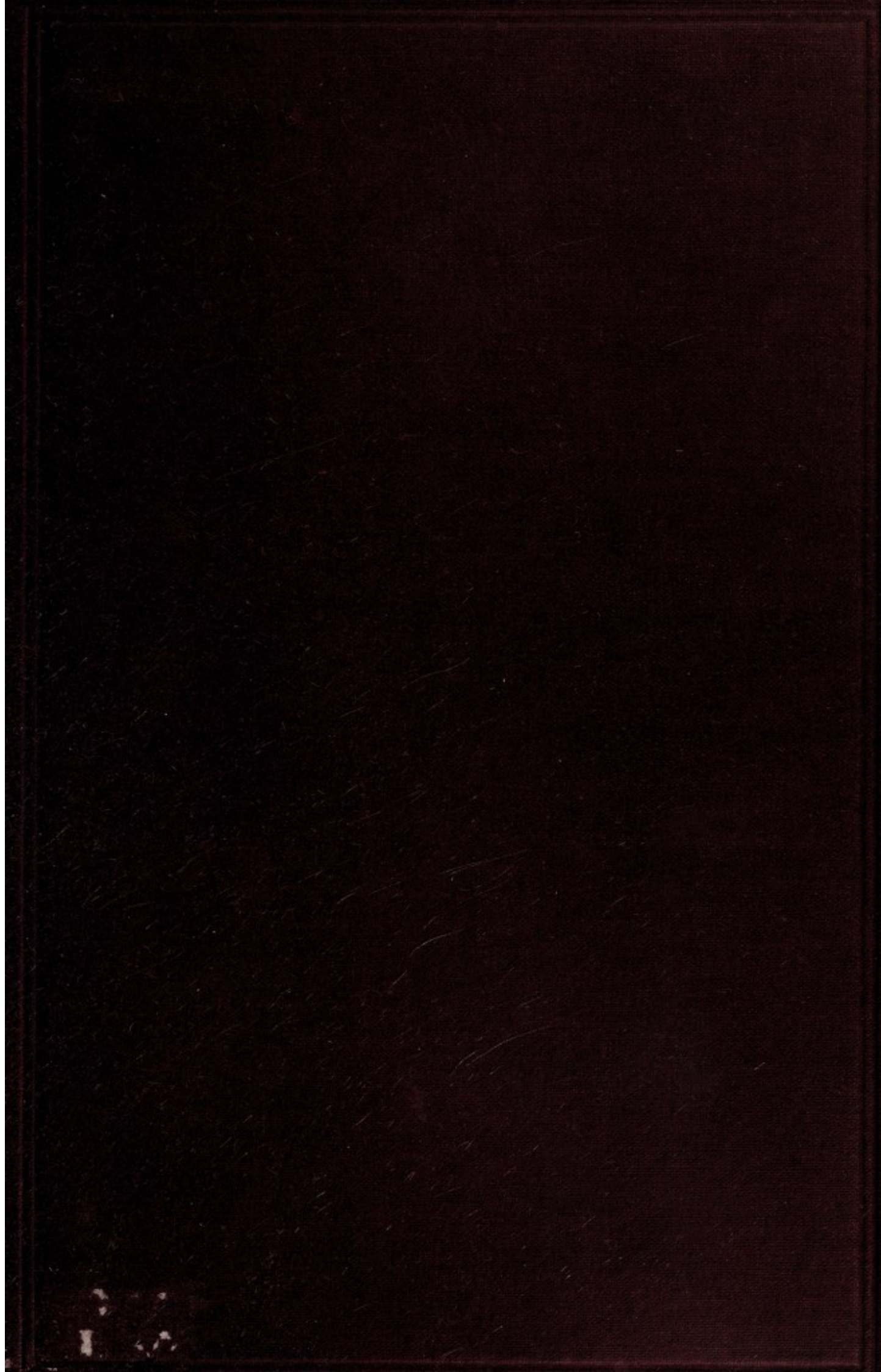
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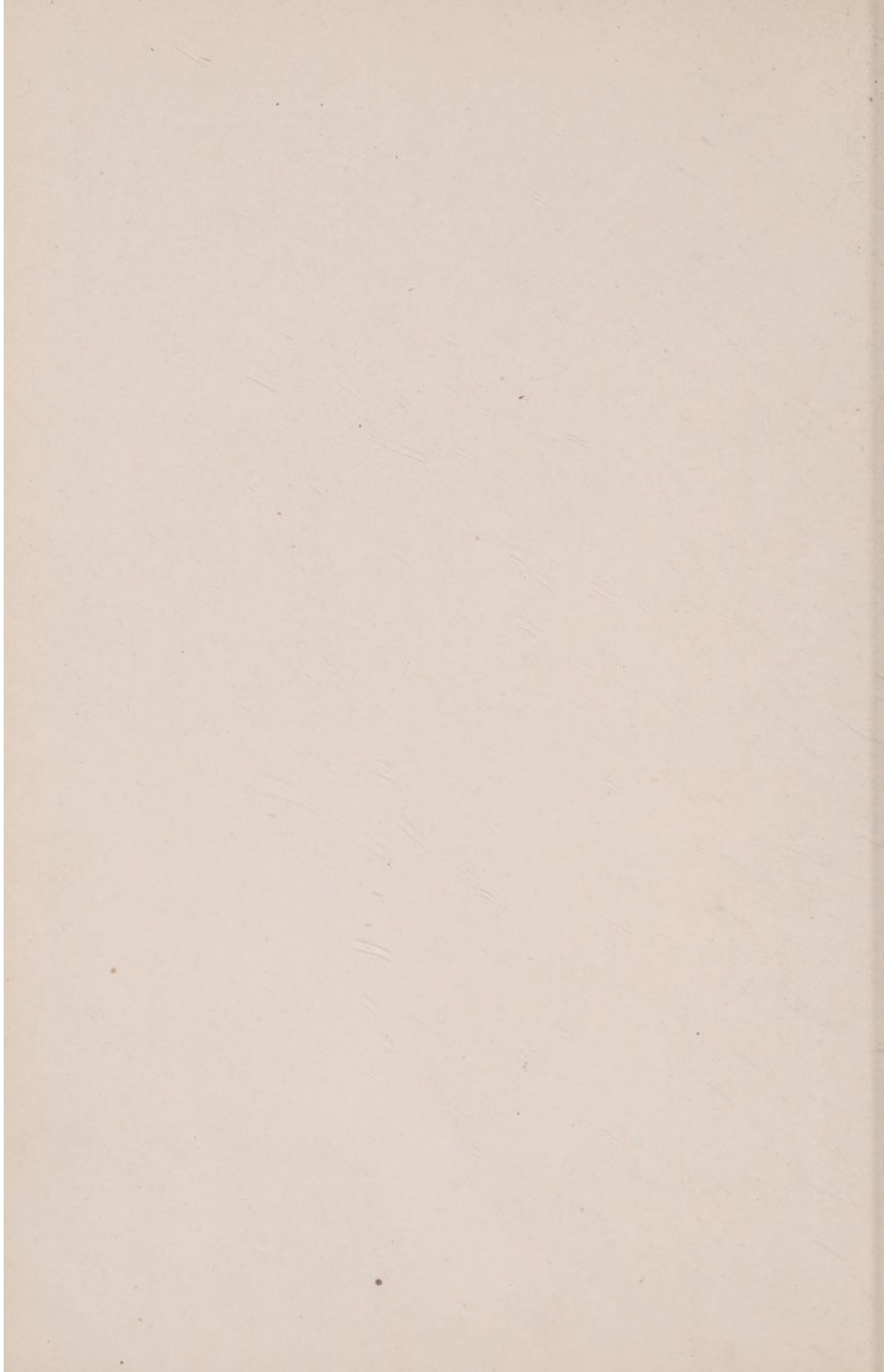
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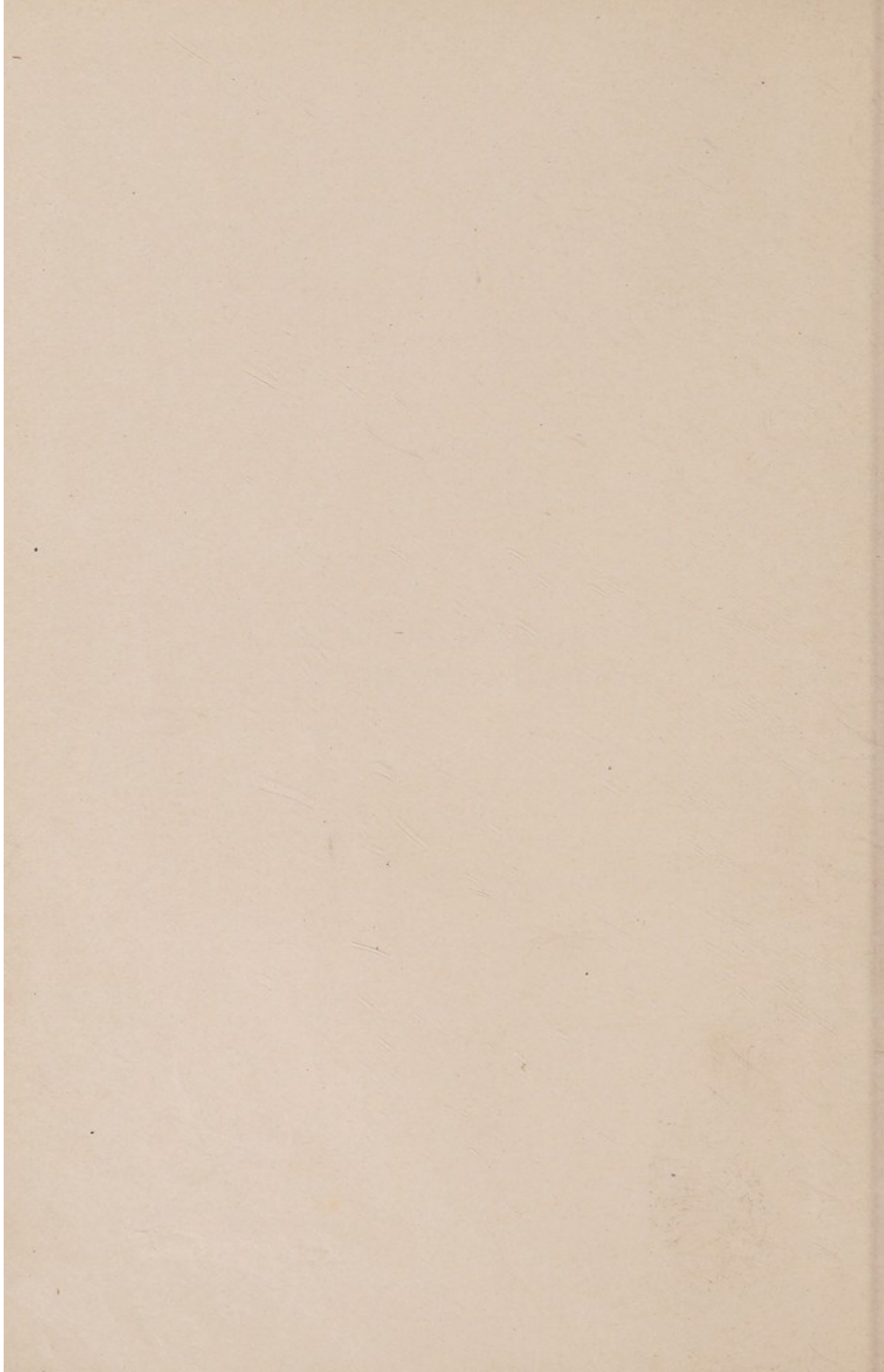
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PSYCHIATRIC-NEUROLOGIC
EXAMINATION METHODS



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PSYCHIATRIC-NEUROLOGIC EXAMINATION METHODS

WITH SPECIAL REFERENCE TO THE SIGNIFICANCE
OF SIGNS AND SYMPTOMS

BY

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EXAMINATION METHODS

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PREFACE

In the introduction to his compendium Doctor Wimmer states that it is meant to be a guide in making psychiatric-neurologic examinations for the use of students and physicians in general.

Such a guide, he says, must of necessity include a part of the general symptomatology belonging to psychoneuropathology, and he trusts that he has neither omitted anything essential nor lost himself in unimportant practical points. He has in several places made a short introductory presentation of the normal psychology or anatomophysiologic data with the hope of giving a better understanding of the diseased disturbances.

Doctor Wimmer says, "My work does not claim to be a real treatise on 'diagnostics.' In order to make a correct diagnosis here, as elsewhere in medicine, it is necessary to be acquainted with the special pathology and to be in possession of clinical experience. However, an examination methodologically carried out is in itself quite a step in the direction of the goal. The subject of simulation has deliberately been omitted. Compendious references to 'unmasking tricks' can but do mischief in the hands of a 'nonspecialist.'"

I hope the book will be found useful to the American student in psychiatry, especially in connection with his work in the mental clinics. The time given to this branch of medicine is as yet so limited that abbreviated outlines of a thorough examination giving references to fundamental features in symptomatology should be welcome to those seeking a practical guide.

ANDREW W. HOISHOLT.

San Francisco, Cal.



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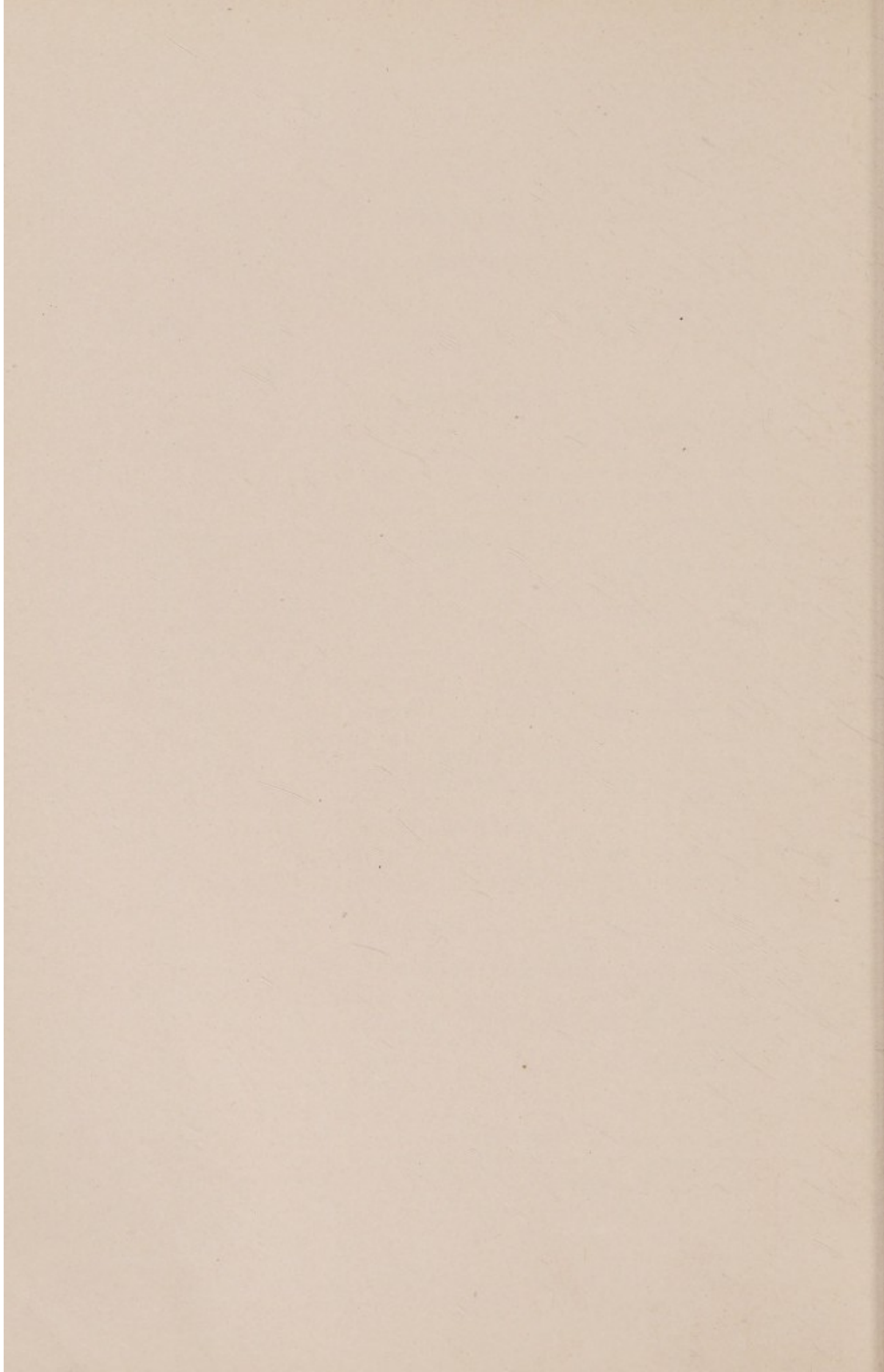
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PSYCHIATRIC-NEUROLOGIC EXAMINATION METHODS

CHAPTER I

ANAMNESIS

The examination of the insane may, like all other medical examinations, be divided into two parts: the *anamnesis* and the *present condition* from a psychic and somatic standpoint.

The same psychotic disease pictures may vary in diagnostic importance according to cause and mode of development. *A careful and full anamnesis is therefore always required.* This should be obtained from the relatives of the patient, from friends, etc.; also where indicated from hospitals, other institutions, etc. The patient himself may be able to give reliable information especially as to circumstances prior to his sickness. At other times his memory is poor and he may be subject to memory falsifications by reason of delusions and personal interests (hysteria, moral degeneration, crime).

Anamnesis Schedule

1. Are there any *insanity, neuroses*, (epilepsy, hysteria, etc.), *inebriety, suicides*, morbid individuality or criminality in the patient's family? Has either of his parents contracted *syphilis*?

2. What have been the characteristics of the history of his life in general? What has been the general *bodily development* during childhood and later? In cases of women: What has been the history of the menstruation

and conditions concerning *pregnancy, births, miscarriages, etc.*?

3. What sickness has he suffered in childhood (*brain diseases, convulsions, "enuresis nocturna,"* sleep disturbances, etc.); later (infectious diseases—in women: puerperal diseases—chronic organic diseases and diseases of metabolism, defective nutrition, etc.)? Has he suffered from *convulsive attacks, paralysis*? Is he *addicted to liquor*? Has he contracted *syphilis*? Has he been exposed to severe mechanical or *psychic trauma*?

4. What was his general *mental habitus* prior to his present insanity? Has he *previously* suffered from *attacks of insanity*; when, in what form and of what duration? Did he fully recover or not?

5. When did his present mental illness begin? What were the suspected causes, the *symptoms, etc.*?

1. *Inherited predisposition (heredity)*. Among the near and distant relatives of the insane we frequently find a history of insanity, neuroses, nervous disturbances of all sorts, etc., although the percentage of frequency may not be to any great extent higher than among normal individuals. This *general neuropathic disposition* is not therefore a safe etiologic indicator. We must know (a) the nature of the psychic and nervous diseases in question, and (b) where in the family they occur (*special neuropathic disposition*).

(a) *Exogenous* psychoneuroses (traumatic, senile, luetic, febrile, general apoplexies, etc.) have hardly any predisposing influence of importance. This on the contrary is true of the endogenous mental diseases: the *affect psychoses* (manio-depressive group), the *dementia precox* group (hebephrenia, katatonia, etc.) and the *paranoid states* (paranoia, paraphrenia). The predisposition frequently reveals itself not through real mental disease, but alto-

gether as *pathologic peculiarities of character*, (morbid personalities, psychopathic constitution, psychic degeneration): eccentric persons, misanthropes, swindlers, vagabonds, drunkards, criminals, prostitutes, etc. *Suicide* may be the outcome of such constitutional psychopathy, or it may in other cases occur in misunderstood or latent mental disease (melancholia, chronic paranoia, etc.). Among the principal neuroses, *epilepsy*, *hysteria* and *neurasthenia* are considered to have a neuropathic effect on the progeny. The influence of heredity is also seen in certain *organic* nervous diseases (*Huntington's chorea*, Thompson's myotonia congenita, amaurotic idiocy, progressive muscular diseases, etc.).

(b) These diseases have the greatest neuropathic influence when occurring among the *direct ascendants* (parents) of the patient. *They have less effect through side lines (collateral inheritance).** Psychoneuroses in the *sisters or brothers* of the patient (or in his children) may indicate the inherited predisposition. Sometimes the mental disease occurs in the *same form* in the ascendants as in the patient (*homologous inheritance*). In affect psychoses and dementia precox, epilepsy, etc., the clinical forms at times vary in character (*heterologous* or transformed inheritance, which has been contested by many). Sickness among the patient's relatives on both sides (*convergent inheritance*) is thought to increase his neuropathy, especially where *numerous* cases of sickness occur among ascendants. Under such circumstances one may sometimes find that the neuropsychoses show an increase in the severity of the clinical forms with downward progress through descendants (*progressive degeneration*), finally terminating in the dying out of the family. Regeneration is, however, possible.

*The clinical history should not make use of such expressions as grandparents, uncles, aunts, etc., but the more correct genealogic names: paternal grandmother, paternal aunt, paternal uncles, etc.

Consanguinity among the parents of the patient is only of importance where these show inherited neuropathy. When in an individual who does not show marked evidence of neuropathy we meet with mental or physical abnormalities which may be traced to a distant progenitor, we speak of *atavism*. It is also to be remembered that a morbid inheritance may be transmitted through a *healthy* intermediate member of the family. Especially is this true of women.*

2. Of great importance to the development of diseased states in the child (imbecility, epilepsy, general psychic degeneration, various nervous diseases, etc.) is the *direct injury to the embryo or germ through chronic alcoholism or syphilis* in the parents. It has not yet been definitely determined whether the general condition of health of the parents, especially that of the mother during pregnancy and other exogenous diseases, etc., can have any influence upon the brain of the infant.

3. The outward life of the patient is a reflection of his general mental habitus, and often reveals facts which are of assistance in understanding the mental disease present. One should know the social origin (illegitimate birth?), conditions in the home, nature of early training, attendance in school, later education, success at examinations, occupations, position in life, domestic relations.

4. *General psychic development*: State of health, circumstances of growth during childhood, age at which patient learned to walk, talk, was taught cleanliness (especially of importance in connection with imbecility). *Sexual development*: Age at first *menstruation* and circumstances of later menses, signs of developing sexuality (masturbation, etc.). *Climacterium*: Date of last menstruation,

*The patient as well as his relatives will frequently deny (in noncriminal cases) the presence of insanity in the family, but willingly admit "nervousness," "despondency," "eccentricity," etc.

concomitant climacteric symptoms. Facts relating to *pregnancies, births, puerperal periods, etc.*, are to be carefully ascertained as well as facts concerning miscarriages, *stillborn children*, the number of living and deceased children, and the state of health and cause of death of the children.

5. *Diseases during the early part of life* of the patient, exclusive of psychoses. During childhood: *Rachitis, infectious diseases* (fever delirium), trauma of the head, and *brain diseases* (meningitis—infantile paralysis), *infantile convulsions* (age, frequency of attacks), chorea, periodic headaches, *disturbances of sleep*, (insomnia, pavor nocturnus, somnambulism), enuresis—occurrence day or night, continuous, in series, episodic. In later years: *Infectious diseases*, (typhoid, influenza, tuberculosis, etc.), *heart and kidney disease* (uremia), *metabolic diseases* (diabetes, rheumatism, anemias, Basedow's disease, etc.), painful nervous diseases (neuralgias, tabes), serious surgical injuries and operations, chronic malnutrition, overexertion, loss of sleep, etc. In cases of women one should inquire concerning puerperal fever, puerperal eclampsia, protracted lactations, prolonged uterine hemorrhages, genital diseases, etc. Careful inquiry should be made concerning *convulsive attacks* (p. 116), *paralyses* (p. 122), *speech disturbances* (p. 106).

Chronic alcoholism: When did alcoholic abuse begin? Was it steady or periodic [*Dipsomania*: Imperative, frequently with marked mental depression (*dysphoria*) when alone, to excess]? The extent of the abuse and the kind of liquor used? The possibility of an *abnormal alcohol reaction* (atypical alcoholic intoxication by relatively small consumption of alcohol with quick development of an extreme degree of intoxication or a dream state, p. 52). Attacks of furor, etc., are seen in psycho-

paths, epileptics, chronic alcoholics, following trauma of the head, etc.

Other chronic poisons: Morphine, opium, cocaine, tobacco, lead, mercury, poisons in gas form?

Syphilis? Time of infection, outbreak of symptoms in the past, treatment, circumstances of the *Wassermann reaction*? Evidence of disease of the central nervous system during previous outbreaks (headache, dizziness, loss of consciousness, attacks of confusion, convulsions, temporary paralyses, speech disturbances, ocular pareses). Where infection is *denied* by married people inquiries should be made as to miscarriages, stillbirths, children that died at an early age, psychoneuroses in wife or husband (dementia paralytica, tabes); diseases among the children, etc.

Trauma of the head (or general mechanical concussion of the nervous system as in falls from heights, etc., the date of such occurrence and its severity, signs of *comotio cerebri*, fracture of the skull? Direct or subsequent mental or nervous diseases (headache, dizziness, memory defects, mental depression, anxiousness, irritability, changes in character, diminished ability to work, etc.)?

6. *Mental causes* ("psychic trauma"): *Continual* worries, griefs, self-accusations, disappointments, etc., of a domestic, economic, social, erotic nature; *mental shocks*: deaths, accidents, fright, etc., sexual attacks, arrests, confinement in prison. Psychic contagion.

7. The *general psychic habitus* of the patient: Did the development of intelligence take a normal gradual course with marked excursions? Is he *imbecilic*, backward, below par, especially intelligent? Has he special gifts or interests?

Temperament and character? Well balanced, vacil-

lating, lively, apathetic? Is he sensitive, inclined to *episodic outbreaks* of *moodishness*—dysphoria: of short duration, suddenly arising and subsiding despondency, frequently showing fear, restlessness, wanderlust (poromania, fugue), craving for liquor (dipsomania, p. 13), impulsive acts? Or is he *habitually elated*, moody, easily influenced, talkative, restless, a schemer, a visionary? Does he show a *lack of balance* and *extreme reactions of affects*, irritability, etc.? Have the excursions in mood shown any *periodicity*? Has he been peculiar, seclusive, suspicious, self-referrent, querulous, self-righteous, exalted? Has he revealed any *moral defects* [laziness, meanness toward people (children) or toward animals, untruthfulness, inclination to steal, sexual excesses, tendency to violate the law, loss of feeling for family, relatives, etc.]?

8. *Earlier attacks of mental disease?* When, how often, of what duration? Suspected causes? Sudden or gradual development? Most important symptoms (see below)? The course of the disease (continued, with remissions, influenced by external conditions)? Treatment and where received? Complete restoration or with defects (residual symptoms: change in character or mood, loss of interests, intelligence, memory, emotional life; presence of fallacious ideas, hallucinations; peculiar ideas, mannerisms, bad habits, diminished ability to work, social decline)?

9. *Present mental attack: When did it begin?* (The patient himself as well as his relatives often overlook the first signs of mental disease—as long as the patient is not confused, dangerous to himself or to others, etc.; careful inquiry without suggesting anything should therefore be made, especially in all *criminal cases*). As

to form of mental outbreak, etc., see preceding paragraph.

Symptoms

This inquiry presupposes a certain knowledge of the clinical disease pictures. As the examination of the patient progresses one will often be inclined to add to the inquiry in support of an anticipated diagnosis.

(a) *Signs of depression*: Psychic inhibition, failing ability to work, a shunning of society, despondency, weeping, fear, self-accusations, *suicidal thoughts*, *suicidal attempts* (this should always be inquired into).

(b) *Elatedness*: A joyous state without motive, laughter, speech-craving, meddlesomeness, passion for finery, extravagance, mischievousness, meaningless purchases, trades, journeys, etc.; sexual and alcoholic excesses, immoral or illegal acts. Irritability, willfulness, intolerableness, domineering disposition, querulence, quarrelsomeness toward surroundings, fits of anger, violence, etc.

(c) *Disturbed train of thoughts*: Senseless talk, confused acts, confusions as to surrounding persons, place and time. *Retardedness*, defective memory, loss of interests, psychic sluggishness, negligence with regard to work, home, personal appearance, cleanliness; peculiarities, bad habits, mannerisms.

(d) *Fallacious ideas and hallucinations* (see pp. 55 and 60): Has the patient shown a noticeable suspiciousness or enmity toward certain persons? Has he talked about being spied upon? Has he complained about being subject to spitefulness of other persons, to persecutions, injurious influences? Has he shown seclusiveness, moving about from place to place, etc.? Has he thought himself scolded, mocked, criticized, etc.? Has he revealed a marked increase in self-esteem, claimed to be possessed

of special gifts, meant for particular missions, or to have marked distinction due him? Has he expressed pronounced grandiose ideas, shown a certain noticeable zealousness to reform? Has he preached religion, talked politics, etc.?

(e) *Somatic nervous symptoms*: Headache, dizziness, vomiting, fainting spells, spasms or convulsions; paralyse (localization, degree, duration), mode of development (sudden, gradual); speech disturbances (was the patient unable to speak, write, read, or was he unable to make himself understood?), mode of origin, duration? Ocular paralyse, optic defects? Disturbances of sleep, degree of emaciation or loss of weight, appetite? Symptoms pertaining to the heart, lungs, kidneys? Fever (expressed in degrees of temperature)? In making this inquiry concerning symptoms which is often of diagnostic importance the chapter on "the somatic state" should be consulted.

CHAPTER II

THE PSYCHIC STATE

The psychic state had better be considered before going into the somatic examination which often frightens the insane patient when taken up first.

Examination Schedule

I. THE EXTERNAL DISEASE PICTURE

1. *Fundamental emotional state:* (a) Despondent, anxious, perplexed, silent, crabbed, suspicious, etc. (b) Elated, merry, arrogant, erotic, angry, etc. (c) Superficial, blank, etc. (d) Is the mood the same or changeable (labile), spontaneous or responsive to outer influences?

2. *Psychomotor conditions:* (a) Motor inhibitions, complete immobility. Does the patient react when addressed, when pricked with a pin, when one makes threatening motions in direction of the eyes, etc.? Does he take the initiative in speech, does he answer questions or is he silent? Are the arms and legs freely movable? Is muscular tension, resistance, catalepsy present? Does he lie down assuming twisted, uncomfortable positions? Is untidiness, urinary retention, refusal of food present? (b) Is motor unrest, restlessness, craving for occupation, mannerisms, stereotypic movements, loquacity present? (c) Is the motor unrest continuous or does it cease spontaneously? Is it influenced by external conditions? Is it in keeping with his emotional state, speech?

II. THE FUNCTION OF CONSCIOUSNESS

1. Questionnaire.

2. Course of ideas: (a) Flight of thought, thought abstraction (perseveration, verbigeration), thought confusion. (b) Thought inhibition; thought block. (c) Emptiness of thought, or possibly attempts at thought association.

3. Attention (spontaneous, sluggish, divertible). Bourdon's test.

4. Apprehension: (sluggish, failing, perverted). Heilbronner's picture test.

5. Orientation: The lucidity of the patient as to time and place, surrounding persons, personal conditions, etc.: Disorientation, delirious states, confusion, dream states.

6. Sense deceptions: Illusions, hallucinations, (domain of sense intake, contents, intensity, localization).

7. Fallacious ideas [contents, date and mode of origin (fully developed from the beginning or through misinterpretations), further course of development, systematization, with involvement of affects]. Imperative ideas.

8. Imperative thought habit: Imperative ideas (query mania, insanity of doubt, phobia, impulsions), autochthonous ideas, overvalued ideas.

9. Memory: (a) Recording faculty, Ranschburg's method. (b) Power of reproduction (inhibited, defective; memory falsifications). Limited loss of memory (extent, mode of origin as regards normal experiences or experiences during illness). (c) Memory contents (personal experiences, practical information and school knowledge).

10. Individual mental work (judgment, power of discrimination, ability to combine, etc).—Ebbinghaus' test.

It is self-evident that all these examinations should not be undertaken continuously one after the other. The patient would thereby, among other things, become so mentally exhausted that the result of many of our special methods of examination would become quite misleading. At the first sitting we confine ourselves therefore as a rule to (1) description of the picture of the external condition; (2) a record of answers to the questionnaire; (3) a few catch questions involving the patient's power of apperception and memory for new and old impressions, after which comes (4) the somatic examination.

I. THE EXTERNAL DISEASE PICTURE

The external disease picture is especially influenced by (a) the *mood*, (b) the *reaction in behavior* as shown in *mimicry, gesture, and speech*. The different forms of the picture are dependent upon (1) the nature and stage of the disease; (2) the *individual characteristics* of the patient: age, sex, mental development or state of intelligence,

refinement, wealth of feeling and imagination, etc. The principal pictures of the external mental state are:

1. *Depressive inhibition*: The *mood* is depressed, sad, desperate (depression, dysthymia). The facial expression and gaze are worried, despondent, hopeless, anxious, etc.; there are frequently tears, quiet weeping, sighing, lamentations, melancholy exclamations and self-accusations (p. 60). *Marked motor inhibition*: Taciturnity, slow, soft, hesitating replies or silence (mutism—mutacism). Cautious, hesitating or anxious movements, helpless picking, wringing of hands or complete immobility (stupor). Skin is often pale, cheeks hollow with prominence of facial wrinkles and slow pulse.

2. *Agitated depression* (most frequently an "anxious restlessness"): The facial expression shows deep despair and fear, a perplexed, anxious look, and there is restlessness, wringing of hands, clinging to persons around, lamentations, screams of fear and off and on despondent loquaciousness (p. 42). Congestion, perspiration, dilated pupils, tremor, startled movements as from fear, a quick, tense, now and then irregular pulse; increased, superficial (often "jerky") respiration.

Depression in hysterical persons, in children, old people or demented cases (hebephrenics, paretics) is often more noisy, blubbering, with many-worded lamentations, monotonous whining, extreme theatrical mimicry and gesticulation, martyr-like facial expression, a certain degree of self-satisfaction or we find sulkiness, querulence, to a certain extent divertible by external impressions or increased by them, by visitations, round of physicians, etc.

3. *Thought block* (schizophrenic resistance) must not be confused with depressive inhibition. The former is a momentary or more continuous stoppage in the train of

thought or behavior (reply, handshake, eating, etc.) which does not have its origin in other psychotic symptoms (for instance in the mood). It is sometimes accompanied by a sudden discontinuance after which the patient talks, acts, etc., without inhibition which is contrary to what one sees in the despondent patient in whom the act, when it finally gets started, is most frequently hesitating and slow. This negativism is sometimes like a falling to pieces, at other times it reminds one of the fable of the frog who "crawls up three steps and falls back two." It is undoubtedly often due to a bringing into uppermost thoughts of idea complexes markedly involving the affects which completely absorb the patient's consciousness. At other times it might seem due to imperative hallucinations (p. 58), to sudden fatuity of thought, etc., which the patient often describes "as if his thoughts had been taken away from him." The mental state is to a certain extent characteristic of dementia precox (schizophrenia). It often alternates with catatonic restlessness.

4. *Stupor: Complete immobility* emanating from the mind, which now and then leads to assuming very uncomfortable or twisted positions (doubled up positions, "à la vache," etc.). *Silence:* No reaction when addressed, when skin is pricked with a pin, when threatening movements are made in the direction of the eyes, etc. Winking of eyelids is infrequent, there is an absence of warding-off movements (flies moving about the face, into the mouth). Play of features is almost stationary, molded (compare below); eyes are often shut or there is a distant vacant stare. Driveling is frequent as well as collection of saliva in the mouth. A *refusal of food* (sitophobia) is present as a rule and often urinary retention, or the urine and feces are passed in bed.

We find either *complete muscular relaxation*—the passively lifted arm falls to the side when it is released (equal on both sides); or we find *cataplexy*: if an arm or a leg is moved passively and put even in an uncomfortable position the patient will let it remain for an unlimited time* without giving evidences of fatigue (tremor in the extremity, tachypnea, rise in pulse rate, perspiration, congestion of the face). The attempt to make this test is sometimes met with a slight, elastic, quickly overcome resistance (*flexibilitas cerea*).

Or we find that when we try to move the patient's limbs, to open his eyes (the lids being generally pinched together), the mouth, etc., we meet with an *energetic muscular tension*, which is often present when the patient is left to himself. Or a direct resistance (*negativism*) may sometimes be shown in other ways; for instance, when the patient turns away when spoken to, forces lips together when spoon fed or spits out the food. Or he retains his urine and feces when brought to the toilet, afterwards urinating or defecating on the floor, in bed, etc.

In contrast with this, certain patients will imitate movements made before them (*echopraxy*), repeat words said in their presence (*echolalia*). The muscular symptoms in stupor are always bilateral, the tendon reflexes always present, now and then with hyperreflexia, but clonus and the Babinski reflex are never present. Pupillary reaction to light is practically always observed although it may frequently be sluggish.

The stupor may be interrupted by episodic, spontaneous movements, often of a peculiar character (p. 27) or by *fulminant*, impulsive acts (a sudden rushing out upon the floor, breaking to pieces, violence, self-mutilations, suicidal attempts).

*The time limit is usually put at 55 minutes.

The picture of the stuporous state varies with the nature of the mental disease originating it. The picture just described applies particularly to the catatonic form. In the *depressive stupor* of the manio-depressive and psychogenic psychoses, etc., the despondent affects may reveal themselves by a set, sad expression of the face, by a hopeless staring or anxious look, tearful eyes, by sighs, etc. Making references to the personal affairs of the patient, giving feigned scoldings, etc., may bring a blush to the face, tears to his eyes, a restless and startled gaze or quickened respiration. Threatening movements in the direction of the eyes, pricking the nose, temples, soles of the feet, may startle the patient or cause sluggish evasive movements, bring expressions of fear to the face, etc. The patient's resistance toward passive movements which is not stubbornly invincible but intermittent-evasive, is often indicative of marked apprehensiveness. Real catalepsy is not observed. By reason of the presence of a despondent complaisance, etc., the patient may let the lifted arm remain in the given position, symptoms of fatigue will, however, quickly show themselves. Driveling and collection of saliva in the mouth are rare. Where there is untidiness about the bed the patients plainly show expressions of shame or unhappiness. Retention of urine is observed now and then, often with an appearance of apprehension when the patient is brought to the toilet.

At times a distinct motor inhibition with an elated state (*maniacal stupor*) may be found: The patient will lie in bed almost immovable, silent, but with a beaming face, jolly, attentive look, small outbreaks of laughter and may make fulminant, mischievous acts, etc.

In catatonic stupor the facial expression may also appear to show a cheerfulness with a vigilant, surprised expression, grimacing mimicry, etc.

Pseudostupor may be based upon imperative hallucinations (p. 58); it may mask thought concentration on delirious or hallucinatory experiences; or it may be a dream-state, etc. The patient is self-absorbed—absent-minded, more or less immobile, statuesque and silent, or he may occasionally be so taken up with his hallucinations as not to answer when addressed or only after repeated questions. The mimicry is sometimes quiescent—dreamy, sometimes anxious, suspicious, angry, sometimes languishing, blissful, ecstatic or in accordance with the internal psychic experiences. There is no real negativism, rarely catalepsy.

A *voluntary reservedness* (silence, reticence, suspiciousness satiated with contempt, and an exalted facial expression and poise) is often seen in paranoics.

5. *Unconscious states* are as a rule of *organic* origin. They include:

(a) *Somnolence*: The patient is drowsy or sleeping but may be awakened by *slight* stimulation (loud address, pricking, etc.), is however confused, more or less disoriented and falls immediately asleep again.

(b) *Sopor*: The patient can only be awakened for the moment by *severe* stimulation, such as pricking of the forehead, nose, plantæ, the use of the faradic current, etc.

(c) *Coma*: Complete loss of consciousness, during which the patient can not be awakened even by the most *powerful* stimulation. There are no spontaneous movements. When the arm or leg is passively lifted it falls inert (observe a possible difference of the two sides and the rigidity that may be present—see Paralysis). The eyeballs may frequently be seen to move slowly under the half shut lids (concerning conjugate deviation, see p. 100). Cyanosis of the face is frequent. There is an *absence of the pupillary, corneal, skin reflexes* and sometimes also of the tendon reflexes. The Babinski reflex, changes in

pulse and respiration, retention of urine (practically important) are often present.

Loss of consciousness may among other things be due to (1) *acute exogenous intoxications* [alcohol (habitus, feter ex ore), hypnotics such as chloral, veronal, etc., morphine (pupillary contraction), hyoscine (mydriasis), etc.]; (2) *autointoxications* (uremia—then often convulsions, diabetes—odor of acetone, etc.); (3) *preceding convulsive attacks* (compare p. 117); (4) *meningitis* (usually sopor or somnolence, screaming, gritting of teeth, stiffness of the neck, Kernig's symptom, ocular paresis, etc.); (5) *cerebral apoplexy* (p. 134); (6) *commotio cerebri* or *fracture of the skull* (anamnesis, external signs of lesion, hemorrhages from the nose, ears, mouth, slow pulse, vomiting, etc.).

In *all* unconscious states an *examination of the urine* (albumin, sugar) is *absolutely necessary*; possibly lumbar puncture.

Lethargy (psychogenic) most frequently originates suddenly following psychic shock, convulsive attacks, etc. It is a *sleep-like* state, usually with muscular relaxation, tremulous eyelids, a few spontaneous movements, an anesthetic state over the entire body so that the patient can not be awakened by pricking the skin (contrary to experiences in normal sleep) associated now and then with spasmogenic zones. The color of the skin is natural or livid. Heartbeats and pulse slow, sometimes with *minute long intermissions* (cases of suspended animation). They frequently result in sudden awakening with loss of memory.

“*Absenses*” or petit mal attacks (epilepsy, hysteria): Momentary pause of consciousness, sudden turning pale, immovability, dropping of things held in the hands by the patient, absent-minded staring, occasional slight clonic

jerkings of the body, smacking of the lips, unintelligible grunting; all of these features *lasting seconds* followed by a *sudden* awakening, the patient resuming his work, his conversation, etc., as if nothing had happened. Amnesia as regards the attack is found to be present.

Narcoleptic attacks resemble a sudden falling asleep (?). They frequently set in while the patient is standing up, do not lead to falling or to change of color of the skin, are entirely without reaction to stimuli, last only seconds, show a sudden awakening and amnesia. The attacks often repeat themselves, frequently on the same day (in epilepsy, hysteria).

6. *Maniacal restlessness: Elated mood* (exaltation, hyperthymia); he is joyous, giddy, optimistic, mischievous, rakish, superior, etc. He shows a vivid, elated facial expression with gesticulations, risibility, *loquaciousness* (p. 20), *craving for activity*, running about, busybodyness, meddlesomeness, dancing, singing, mischievous acts, etc. As the excitement increases, the patient shows more and more purposeless polypragmaty, noisiness, destructiveness, untidiness, filthiness, violence, and confused talk (p. 41).

According to the increase in intensity of the disturbances we differentiate between (1) hypomania, (2) mania levis, and (3) mania gravis.

The mood is often very *changeable*, passing suddenly into anger, fury, cursing, fighting, or vice versa into emotionalness, tears, frequently through impressions from without, or for selfish reasons. *Prolonged exaltations with anger* have especially been observed in certain psychogenic degenerative manias characterized by hypersensitiveness with violent explosions, hypercritical mood, domineering, meanness, obscenity, etc. The excessive functional

activity does not always affect simultaneously the three domains: mood, life of action, train of thought or speech. We meet with maniacal restlessness without any special loquacity and vice versa, motor restlessness and speech-craving without real elatedness, etc. (*manio-depressive mixed states*; see "maniacal stupor").

7. *Catatonic restlessness* is characterized by (a) a frequently striking discrepancy between mood (or train of thought) and motor restlessness, (b) by a special form of motor restlessness.

(a) *The mood* seldom has the "vivid" stamp of elatedness, is frequently quite shallow, and is more reluctantly sluggish or roughly superior than really joyous. The mimicry is often quite stationary ("empty smile") or altogether apathetic (*paramimia*). The mood is but to a slight extent influenced by impressions from without. The frequent *revulsions of feeling* (anger, blubbering, violent sobbing) are usually spontaneous and seem to outsiders quite without motive.

(b) *The motor restlessness* which is more or less severe and continuous, is predominantly *monotone* and *stereotypic*: speech-craving of peculiar nature (p. 37), reviling, swearing, thrusting out of the tongue, grimacing, turning of the eyes, caricaturing gestures, twisting around like a snake-man, turning somersaults, boxing attitudes, destructiveness, smearing, untidiness. "The patient is in constant movement without in reality accomplishing anything." His motor restlessness is not dependent upon external incitations as is that of the maniacal patient who is "ambitious" and whose restlessness often subsides when he is left to himself. The catatonic patient does not require much space while the maniacal case roams about. The former is "machine like," his actions being made up of twisted, unpractical, clumsy,

individual movements requiring an unnecessary strain of muscles. *The movements often show a monotone repetition* (mannerisms, motor stereotypy) lasting for a considerable time. There is an awkward twisting about in bed, a running around the bed, a hammering with an arm or a leg (and always the same one), a gesticulatory putting up of arm and leg in twisted, often "pathetic" positions, a constant stripping off of clothing, making attempts to destroy the bed, etc., after which the patient may be found sitting quite apathetic or with a broad grin on his face among the ruins that he has wrought.

The restlessness often shows *pauses* with an appearance of thought-block, stupor of short duration and apathy. The speech-craving varies greatly, sometimes with slight or considerable motor restlessness and almost complete mutism (very seldom seen in mania, p. 27). Or the speech contents may seem entirely out of keeping with the patient's individual acts; in the midst of the wildest unrest he may suddenly give a sensible reply, show himself fully oriented (for instance often when he is being visited).

His acts are frequently impulsive (catatonic raptus). The patient may suddenly bolt, get upon the table, the window sill, upset a chair, throw his dishes on the floor, make assault upon his surroundings; or he will rush around the bed a few times, turning cartwheels, scold, weep and sing at the same moment. All of a sudden he may become quiet (at such times we may find imperative hallucinations present, p. 58).

8. The restlessness met with in *confusional states* may resemble the one just described; it, however, more frequently has a stamp of perplexity, apprehension, helplessness, complete lack of understanding of the situation. The *motor reaction* is usually quite purposeless, vacil-

lating as to direction, seldom especially violent, only to a certain degree monotone, not really stereotypic, frequently influenced by external impressions; viz., getting out of bed, confused roaming about, constant reaching out for doors and windows, rushing into the toilet or in obscure corners, undressing, helpless fussing with bed clothes, tearing at the hair, scratching the skin, picking inquisitively at all objects, hurling the dishes along the floor, smearing, etc.

The *mood* is usually changeable: apathetic, anxious, easily excited, etc. Sometimes silence, sometimes confused chatting is present.

In profound confusional states (for instance in infection-delirium) the motor restlessness is often limited to a restless twisting and turning about in bed, picking with the fingers at the blanket (carphology) unintelligible muttering ("mussitating delirium"). The restlessness is of a peculiar character in *delirium tremens*; the patient thinks he is engaged at his usual work, drives his horses, picks his rags, waits at the table, is taking his drinks, etc.; he is tremendously busy in a quiet satisfied way ("occupation delirium").

9. The *stuporous states* are first and foremost characterized by an *emotional blankness* (apathy). The patient lies about in a dull state, is uninterested, sleeps much or lounges on a chair unoccupied, does not often talk spontaneously to any extent, answers sluggishly and in a few words, is poorly able to attend to himself at meals, to attend to his personal cleanliness, etc. Now and then again there is a sulky resistance, seldom catalepsy. At other times the *motor restlessness* is more marked, monotone, and he may often be rather quiet about it in a gentle, fussing way; constantly making the bed, undressing, twisting at the door knob to get

out, picking at the beds of his fellow patients, prating in a low voice, etc. The restlessness is often most pronounced at night (senile demented, paretics). The motor restlessness, or meddlesomeness, is especially marked in the *demented euphoria* (particularly in the parietic form). The patient feels impelled to arrange numerous things, to help the other patients, to gather all kinds of trash, to decorate herself with finery; is in a constant state of well being, satisfied with everybody and everything, showing nonsensical speech-craving, baroque, grandiose ideas (p. 63).

In organic stuporous states we now and then see *compulsory* (imperative) *laughter or weeping*; more or less without cause and usually without being in a corresponding mood, the patient gets to laugh or cry without being able to stop himself.

Erotism (libidinousness, erotic excitement) may be found in all clinical pictures of mental states (even in depressions, for instance of the climacteric period), most frequently in restless states: Bold phrases, obscenities, coquetry, exposures, masturbation, sexual attacks on surroundings (even of the same sex), sexual perversion, etc.

II. THE FUNCTIONS OF CONSCIOUSNESS

A temporary estimate of these may be obtained by filling out a *questionnaire* according to *Sommer*; i.e., putting a series of questions which arrayed in natural series are to substantiate whether the patient presents certain *fundamental psychic disturbances* or not.

The patient's name, the date and time of day when the examination was made must always be noted. His answers are to be put as *full* and as much *word by word* as possible. The *way* in which the patient expresses himself is often much more important in psychiatry than

whether his reply is correct or not. A plus or minus placed opposite the questionnaire is always of double meaning and confusing. If the patient does not reply we note down "no answer."

I. QUESTIONNAIRE

- | | |
|--|---|
| 1. What is your full name? | 17. What kind of people do you find here? |
| 2. How old are you? | 18. Who am I? |
| 3. What are you? | 19. How long have you been in this place? |
| 4. Where do you live? | 20. What day of the week is this? |
| 5. Why have you come here? | 21. What month is this? |
| 6. Are you sick? | 22. What year is this? |
| 7. Are you downhearted? | 23. Where were you yesterday? |
| 8. Are you in fear? | 24. Where were you eight days ago? |
| 9. Are you being persecuted? | 25. Where were you a month ago? |
| 10. Have you been threatened by anybody? | 26. Where were you last Christmas? |
| 11. Do you hear voices? | |
| 12. Have you any visions? | |
| 13. Who has brought you here? | |
| 14. Where do you come from? | |
| 15. How long had you been there? | |
| 16. What kind of a place is this? | |

After the patient has given his answers one should at once or later on, go more profoundly into one or the other questions by further additional inquiry. It is best to note down on the schematic form whether the patient replies quickly or slowly, willingly or reluctantly; whether he answers only upon repetition of the question, whether he is attentive or must be constantly aroused to reply, whether certain questions (and in that case, which ones) excite him, etc.

The questionnaire should give one information as to (a) the orientation of the patient with regard to personal circumstances (Questions 1 to 4), with regard to place

and surroundings (Questions 16-18), as to time (19-22); (b) as to his *memory* for more recent experiences (Questions 13, 15, 23-26); (c) as to certain important *elementary disturbances* (mood, fear, fallacious ideas, hallucinations (7-12)); (d) as to his *realization of being ill*, i.e., whether he has disease insight (5-6). Facts may furthermore often be ascertained with regard to thought inhibition or flight of ideas, thought confusion, blankness of thought, perverted understanding, confabulation, etc.

The more thorough examination of the different sides to functions of consciousness must lay stress upon these aspects which the clinical picture as a whole may show to be of special importance.

II. COURSE OF IDEAS (IDEA ASSOCIATIONS)

Sensory impressions or ideas which have been present in consciousness at the same time or one after the other will frequently show a tendency to combine, to form associations so that when one of these again turns up in consciousness it will arouse the others. Of all the possible associations of ideas, however, only a small part will assert itself in normal consciousness; such as those frequently experienced having external connections, those practiced again and again, those related rationally or according to significance (i. e., logically). The impressions and ideas which are experienced *during severe emotional excitement* show a special tendency to association or preservation in memory. These often reveal a close tie between essential and unessential elements (unimportant persons, words, etc.). *Such memory complexes involving the affects* are frequent in normal persons and play a considerable *pathogenetic role* in psychopaths, hysterical patients and others. The complex

operates in different ways in consciousness: (a) As a more or less complete memory picture with dejection, dimness of consciousness ("reminiscence delirium," p. 53, from which fallacious ideas may take their origin, p. 70). (b) As a complex with the "unconscious," i.e., without distinct (continuous) contents of consciousness but with marked affects ("groundless" dejections, dysphorias, moodiness, etc., p. 15). (c) It may operate through abnormally severe *irradiation of feeling*; the sight of the person who accidentally enters into the idea complex of the patient (the mail carrier bringing a letter containing bad news, etc.) becomes *as such* the cause of a new emotion (idiosyncrasies, antipathies, and sympathies). (d) Or the idea complex operates by *clustering*, choosing the ideas (even without the patient being fully conscious of doing this); contrast ideas are sidetracked, congenial ideas are adhered to (thought "displacements:" hysterical amnesias, p. 70, the Ganser syndrome, p. 54, etc.).

The above-mentioned idea associations may be collectively designated as "*personal*" *memory associations*. In addition to these we have in man the very important *word associations*. They are symbols of sense impressions and ideas of another nature (optic, tactile, etc.), often "condensed" terms for *groups of ideas* ("general concepts"—army, hospital, gratitude, etc.), and admit an abbreviation of the activity of consciousness: The ideas (individual memories) X, Y, Z, etc., do not need to be reproduced in toto each time, the mental process being able to get along by going over the corresponding language symbols (we usually "think" predominantly in word pictures). It is, however, essential to the normal, "regulated" train of thought that the muscular movement in

connection with the word pictures is first exercised from the very beginning in intimate cooperation with memory associations; secondly that whenever the motor activity repeats itself, it takes place under a certain control (in the manner of a more or less conscious "joint vibration") of the individual, experienced idea associations, so that these can be easily reproduced by "after-thought."

Information concerning the patient's train of thought we obtain (1) through his conversation, spontaneous or in reply to questions; (2) through his *writings*; (3) through his description of himself (many are very poor at describing their own ideas of apperceptions, others, among them psychopathic and hysterical patients, are apparently too melodramatic and metaphoric to accomplish this); (4) one may by exercising caution be able to draw conclusions as to the patient's train of thought from his *general behavior*.

Disturbances in the idea associations relate to tempo, contents, coherency.

1. *Flight of ideas* shows itself outwardly as a rule in *speech-craving* ("internal flight of ideas," see p. 42): The patient is garrulous, talks rapidly, can hardly say as much as he wants to, makes remarks about everything (which are often indiscreet, impertinent, criticizing, mean; compare with statement below), has an opinion of everything and everybody, attempts to show off with set phrases, is full of good stories, jokes, puns, murders foreign languages, tries to make an impression with big words, oaths, coarse language, obscenities, brags, makes a parade of exalted ideas (p. 62), sees everything in a rosy hue, especially himself and what belongs to him, declaims, sings in a loud voice, etc., doing this al-

most until he froths at the mouth or until he is as hoarse as a frog—his hoarseness sometimes amounting to a rasping whisper.

The *train of thought* is frequently accelerated (?), is first and foremost *quite vacillating*. The patient jumps from one subject to another, loses himself in side issues, so that he finally forgets what he wished to say. The train of thought is to a great extent influenced by impressions from without, *very divertible* (p. 46); words picked up from the conversation going on around him, from the dictations to the clinical stenographer, from the peculiarities of the individuals present, from the appearance of a stranger, etc., will at once give a new direction to the course of ideas. It is only with the greatest difficulty that a patient can be made to give accurate replies to concrete questions; but the replies are then as a rule correct.

The idea association takes place only to a slight extent in harmony with rational relationship, in part as common experience associations (side issues), more frequently through routine idea connections (play on words, witticisms, citations, etc.), but particularly in cases where the flight of ideas is on the increase as pure *word associations* (word-completions or word distortions, rhyming, connections by assonance). It is especially the *clang associations* which to a certain extent are characteristic of flight of ideas (the association test on p. 45 only shows it to be present in 2 per cent of normal, not brain-fagged persons). The flighty idea association is therefore characterized by what is *on the surface*.

With the increase in flight of ideas the talk becomes an oratory of individual words or scraps of sentences loosely tied together (*logorrhea*), or a completely confused disconnected (“*secondary incoherency*,” p. 40).

The following is an example of what a maniacal patient may produce of this sort:

“I am the Commander in Chief of the Wolverines—long live the marines—I am loony—I can speak Chinese, Yiddish and German—I have a number seven foot and a number nine foot—I am Commander in Chief of the K. S. P. O.—let us up and go—it means cobra de capello—I can make friends with wild animals by making the Wolverine sign—you can have the flag—it might help you—I am here to help do—I’m a Wolverine—I have no bones—it won’t bleed because I am a Wolverine—once upon a time Abe and I went fishing—we saw a big bear dishing—he just licked his paws and asked him to give him some fish,”—etc., etc.

The individual flight of ideas is richer in words than in thoughts. The *productivity* is dependent first upon the severity of the disease; secondly, upon the individual aptitude of the patient. In cases of *feeble-mindedness* the speech is often very meagre, nonsensical, monotonous, shows strained witticisms, oaths, frequently perseveration (compare with catatonic speech-craving). The flight of ideas observed in certain *demented states*, especially in cases of paresis, may to a large extent resemble the maniacal, but is frequently more pratingly unproductive, as for instance in the following “jingle speech:”

I saved the American fleet
 And I sank the Spanish too.
 My teeth have not grown yet
 But they’ll soon grow too.
 The ground is swell and I’m swell
 And we’ll all swell together too.
 I’ll tell the word together
 That I’m the American God too.
 A ding is a ding and a dong is a dong,

A bell is a bell and a gong is a gong,
A clock is a clock and a 3 is a 3,
And the clock tells 'me I'll soon be free.

In cases of *frontal lobe tumors* we now and then find euphoria with speech-craving and rakish jokes ("witzelsucht" witticism-mania); the mental production which is below par and the presence of other brain symptoms should assist in the diagnosis.

In alcoholics (dementia, Korsakow, delirium tremens) we find now and again a certain coarse-grained good-naturedness, grim humor, etc., but without productiveness.

Circumstantiality of speech (p. 41) must not be confounded with speech-craving, or flight of ideas. Now and then we find something of this sort in normal, uncultivated persons but little versed in conversation, or in connection with embarrassment, as in children, feeble-minded, etc.

The *scribbling* of the patient presenting flight of ideas reveals in the main the same disturbances as his speech: masses of paper daubed with large, hasty, jumbled-together characters, one line sometimes running (cross-wise) over the other and with extremely disconnected contents, forgetting parts of follow-up sentences, with dashes, exclamation points, underscorings in unlimited quantity, with different colored ink, illustrating sketches ("humoristic," obscene, etc.). In the absence of letter paper, the maniac will make use of newspaper, toilet paper, walls, etc. (graphomania).

2. *Catatonic thought abstraction*: The *speech* may be quick though it is more frequently slow, sometimes amounting to an episodic silence, less rich in words (productive) than that of the maniacal patient and is characterized by (1) *poverty of thought*: Outwardly related associations (tautologic phrases, far-fetched play on words,

hackneyed witticisms, *few real clang associations*). The thought connections are frequently quite baroque ("unintelligible"), often with perverted ("highly pathetic," "important") pronunciation of certain words, and inarticulate exclamations, oaths, obscenities, etc. (2) Perseveration, *a sticking to action once begun*: The same word, sentence, phrase, appears again and again; the patient ruminates on a certain thought or a certain question, gives the same answer in ten different ways, never reaches his conclusions in deliberating for or against a thing, etc. All this is in contrast with the maniacal train of thought which is constantly shifting in direction. In further contrast with maniacal conditions we usually find (3) *a diminished divertibility*, at any rate without the quick progressive association seen in the maniacal case, which may often show itself entirely as a perseveration (compare the speech specimen below). While the maniac wants an "audience," the catatonic speech-craving is usually (4) *monologic*, the talk being only to a slight extent directed at the surroundings. The talk may even come to a halt when the examiner approaches the patient, speaks to him, etc., or he may on the other hand talk away when left to himself. (5) In the midst of his unintelligible flow of speech he may often surprise one with quite intelligent replies, remarks, etc., which reveal a full orientation, good apperception, and ability to record things, etc.

The *articulation* of the words themselves is often peculiar: lispings, falsetto, baby voice (also observed in hysterical disturbances of consciousness), words pronounced with rhythm, scanning, stammering, hem and hawing, hissing, simulating voices of animals (*speech-mannerisms*), etc.

Verbigeration is a stereotypic and prolonged, frequently rhythmic repetition of the same, often meaningless words and phrases. He makes use of *neologisms*, self-made words ("Pedar," "Amonphoto," etc.), a brand new language which the patient may speak and write for years and which may possibly remain his only way of expressing himself.

Speech Specimen No. 1. (The talk quite rapid): "You will get along fine, Carlo, if you stick to mathematics—you are to be beheaded in a hurry, boy (completely without emotion)—without reason—in a hurry—you are to go through the intermediary links—you may select planet—(at this moment he discovers the closed hydrant) you are to smite water from a spring—"drinks" from the faucet) that was well, my boy—you may select planet—it is too plain, Carlo—you can smite water from it—you shall believe in the Christian God—strike against planet, Carlo—there is plan in that—you will get along, Carlo—when you don't care a pin for woman—you may wander out into the desert," etc.

Speech Specimen No. 2: "I have been flapping—gapping—lapping—I have crutch—Dutch—Oh, say can you see—but she has been aping me—but I have never been taken there by a tiny molehill—she tries to put down my viewpoint—I was very tidy, when I was a drunkard—and so is Peter Marquard for sure a silly coward—to have me appear green all over the globe—though I have been the first to stamp and tramp and been burned for all of you in England and Denmark and Iceland and Creta," etc., etc.

The *thought abstraction* also shows itself in the patient's *scribblings*, which otherwise are characterized by flourishes, mystic under Scorings and parentheses, cabalistic figures or sketches a la cubism, futurism, etc. At

other times the patient may on the contrary surprise one with formal and good letters full of interest, containing at the most a single sentence having a baroque character or peculiar application.

The catatonic thought abstraction is quite different in nature from flight of ideas (they may become complicated as to tempo): The baroque skipping about in the thought activity, the "senseless" expressions, etc., may *in part* be interpreted as the result of (1) *indirect association* (concerning unconscious or dimly conscious intermediate links): "Goat—(moat)—lotus leaf," "punish"—(punishable) observable, etc. (2) By a *sliding transition* to rationally allied terms or more frequently to expressions related from a standpoint of motor-speech: "Did you say last night your sleeping draft?" (3) Motor-speech *malformation* or *condensation* (contamination): "I have never *conjured* you ("from consulted and injured"). (4) *Perseveration tendencies* (p. 38). (5) *Interruption of the train of thought* by suddenly developing complexes, fallacious ideas, hallucinations—often in the form of "thought block, thought disappearance" (p. 20). (6) Where there is speech-craving we probably will also find imperative "*thought swarming*" (p. 43). (7) *Perverted application of the words* in a "symbolic" meaning to express peculiar inner, mental experiences (thought abstraction, hallucinations, etc.).

3. *Confusion of ideas* (incoherency, dissociation) may be (a) *secondary*, following maximum of flight of ideas or a catatonic thought abstraction and will then retain a part of the characteristic peculiarities in question, such as clang associations, divertibility, or perseveration, neologisms, etc.; (b) *primary* (infectious, alcoholic, epileptic, hysterical delirium, etc., p. 52). In its simplest

form this confusion shows itself as a difficulty in, or impossibility of, bringing the thought to a conclusion, or fixing the point in a question: the conversation comes to a stop every little while, it stays with the attempt to start it again, or it leads to deflections, questioning without comprehension, the uttering finally of fragments of sentences or certain exclamations; i. e., the train of thought and speech becomes *fragmentary*.

Speech Specimen: "You should certainly be able to see that—however—may I not—well—that is—what I wish to say—but—yes—how shall I—because I know nobody—that is just it—that I haven't thought at all—how can one properly speaking—I did think at the moment"—etc.

Where the *speech-craving is confused*, the speech is quicker, more wordy, skipping; the thought connection is on the surface but without essential clang associations and without real divertibility. All ideas, essential or nonessential, seem to assert themselves with equal weight, deciding the train of thought but for the moment, which gives to it a stamp of the accidental, the disconnected, combined with a sudden turning up of rationally indifferent elementary ideas ("rudimentary associations").

Speech Specimen: "Who knocks—thrashed out—you are just performing with your masque-ball—Well, thanks—right now I have a little cold—musical mammae—are you in love—the one most Chinese—confound it, of course—numero—quatorze—or number seventeen—you may begin at Pentecost—I hope for ducats—yes, many thanks—I will affiance you—my beloved—good-night," etc.

In dealing with catatonic thought abstraction the differential diagnosis often requires to be on the look-

out for defects of attention, apperception and orientation met with in mental confusion.

Concerning the *Ganser* symptom, see p. 54. Speech confusion must not be confounded with paraphasia (p. 110) and vice versa.

4. *Thought inhibition* occurs as a rule with depression or depressive inhibition (p. 20). The train of thought is slow (in spontaneous speech, replies to questions, letter-writing) and the mass of ideas is often so reduced that the patient complains of "emptiness of thought." Every thought activity entails an exertion on the part of the patient, his apperception is slow, he has difficulty in giving information, which one should be careful not to confound with impairment of memory. He is exceedingly long in doing the simplest problem in arithmetic or gives them up altogether, etc. The thought connections are of an "inner" nature; the experience-associations, which are often a review of all his earlier recollections of life, are predominantly depressive (self-accusations). Clang associations (assonance) are wholly wanting. The train of thought is characterized by *monotony* but of quite another kind than that seen in catatonic disease pictures.

Now and then we meet with complaints of an "inner flight of ideas," i.e., the patient is not able to fix his thoughts, or he finds that depressive ideas are steadily forcing themselves upon him.

Sometimes the *depression is associated with speech-craving* (in spite of thought inhibition) and often has an *apprehensive origin* (talks "to relieve his feelings"). He is monotonous with a somewhat jabbering talk using the same words and phrases: "Oh—will you help me—won't you help me once more—Oh, am I then really dead—am I dead—what—what—" etc.

Thought inhibition must not be confounded with *thought block* or negativism. The patient often describes this as a pronounced feeling of compulsion (also now and then said about the "*catatonic thought-swarming*": "It is thinking within me—they are reading my thoughts").

Concerning *imperative ideas* see p. 66.

Neurasthenics (psychasthenics) have frequently similar complaints concerning fatuity of thought, etc., as the melancholic patient; usually, however, the examination (association tests, spontaneous speech) will show the associations to be predominantly external, such as assonance, etc., and the train of thought will be found monotone, but no real inhibition will be detected. In *hysterical patients* the train of thought is essentially unaffected if we exclude from consideration pronounced attacks of despondency or confusion, but it is frequently characterized by *complex-activity*: memories with emphasized affects are easily aroused. The train of thought of the *paranoic* also circulates about his fallacious ideas, etc., but may not otherwise be differentiated from that of the normal person.

5. In *stuporous states* (dementia) the train of thought is sluggish, clings readily, is very slightly divertible, is impoverished in ideas, even those close at hand, often characterized by ego-centricity (tedious descriptions of life-experiences often entirely without relation to the point in question, undisguised personal wishes or unrequested appraisements). The speech is drawling, circumstantial, tautologic, the answers vague, often deficient and the words of the questioner often recur in them, or the patient makes them his own. The speech contents are as a whole impoverished, few opinions or fan-

cies of the patient's own are expressed, at the most spunt-out phrases.

6. *The feeble-minded*, especially of the torpid type, thinks and speaks slowly, has difficulty in explaining himself, in catching the main point in an argument, is apt to lose himself in indifferent side issues, makes use of idle chatter, helps himself with general terms, definitions learned by rote, tautologies ("word symbols"), is only to a slight extent able to make use of his personal experiences in a given case or sees everything from his narrow personal point of view. At other times, in cases of the elated type, he may show loquaciousness: Empty prating of childish contents, imitative babbling, etc., without productiveness.

These various disturbances may often be made to show up distinctly in the association tests: A series of 50 (100) test words (nouns, verbs, adjectives, mixed together) are dictated to the patient one by one, distinctly and with neutral emphasis, the patient being told that he is to "say what happened to occur to him" but is not to exert himself to find "something that fits in." One should test in advance whether he has understood the instructions, by letting him experiment with the association of a *few words, not in the series*. If it is impossible to instruct him (in severe mania, confusion, apathy), the *test words* are immediately dictated.

The *reaction time* is estimated with a stop-watch showing $\frac{1}{5}$ sec. which is started when the pronunciation of the test word begins and is stopped when the patient utters the first sound. If possible the patient should be *questioned* after each reaction, (it is less satisfactory after the entire test has been made): "What did you think about?" (In this connection information should be obtained concerning intermediate, more complicated associations, as-

sociation involving vision or other senses, personal recollections, etc.).

The train of thought is here subject to the same forms of disturbances as previously mentioned. The *reaction time* (in adult normal persons about two seconds) is prolonged in mental depression, apathy or dementia and in cases of congenital feeble-mindedness. It varies fitfully in confusion, catatonic restlessness and in negativism. In hysterical patients we frequently have "complex-reactions" (suddenly developing memory with affects): prolonged reaction time, often strange (baroque, blank) reactions succeeding previous sensible ones ("cover reactions").

It is of great importance that the patient remains *attentive* during the test. Inattentiveness causes postponement of the reaction due to external associations. A renewal of the test will then frequently give proper reaction (time, contents) contrary to that brought out by the complex-activity. Many patients become at once bewildered by the test ("emotional stupor"), are void of thought and react slowly. Reassurance and further instruction usually bring satisfactory results.

ASSOCIATION SCHEDULE TESTS

1. Journey	14. Sing	27. Brother	39. Man
2. Pricking	15. Punishment	28. Strike	40. Crime
3. Head	16. Pretty	29. Die	41. Party
4. Money	17. Blood	30. Visit	42. Forget
5. Physician	18. Despise	31. Wish	43. Laugh
6. Heart	19. Wise	32. Mother	44. Church
7. Dance	20. Right	33. Honor	45. Little
8. Sickness	21. Love	34. Child	46. Moon
9. Believe	22. Flower	35. Poor	47. Hate
10. Afraid	23. Mockery	36. Happiness	48. Weep
11. Pay	24. Old	37. Tall	49. Kiss
12. Evil	25. Ring	38. Home	50. Hope
13. Bread	26. Accident		

III. ATTENTION

A sensory impression, a spontaneously arising idea may awaken the attention (1) by its intensity, duration, heterogeneousness; (2) by the ideas that it brings forth; (3) by the feeling-tone or affects that it elicits. We differentiate between the spontaneous attention and that awakened from without and here again between the one which is awakened with difficulty and the one awakened with unusual ease. We are also interested in whether the attention is fixed by the impression, etc., or is quickly fatigued by it; i.e., whether it is easily diverted to new impressions.

Increased spontaneous attention is seen, for instance, in patients suffering from imperative fears (p. 66), in paranoics with marked persecutory ideas (reference delirium, p. 65). *Diminished spontaneous attention* is met with in connection with intense emotional excitement, confusion, stupor and pseudostupor, in catatonia (?), demented states and in many cases of congenital feeble-mindedness. Whether this is also true of thought inhibition is doubtful. In confusion, pseudostupor, dementia, etc., the attention is at the same time *aroused with difficulty* (hypovigility). Attention is *aroused with abnormal ease* especially in maniacal flight of ideas and in certain forms of confusion (infection psychoses, alcoholic deliria). In these cases the attention is at the same time either quickly fatigued (hypotenacity) or *abnormally divertible* (hypervigility).

The determination of the condition of the attention is of decided importance in appraising the results of many of our examination methods.

The *attention* is tested (a) in making out the questionnaire of Sommers; (b) by dictating to the patient mean-

ingless sentences ("the snow is green," etc.); (c) by showing incorrectly or defectively drawn pictures



Fig. 1.

(*Binet*, Fig. 1*); (d) by letting him make a number of serially continuous subtractions (*Kraepelin*) which is

*For clinical use the pictures are drawn of a size of about 4 to 5 cm.

best done (e) by making use of *Bourdon's striking out test*: in a casual makeup of 200 letters, the patient is to pen through all the therein contained 20 *a*'s or 20 *o*'s and *l*'s, etc. The time it takes to strike out is to be measured. The relation of the correctly penned out or omitted letters to those incorrectly done serves to gauge the certainty or perseverance of the attention. The coincidence in the striking out of *o*'s and *l*'s will demonstrate the possibility of dispersion of the attention. Normal adults require a half minute for the test; children sometimes a little more.

IV. APPREHENSION

The correct apprehension of external impressions is dependent (1) upon the strength, duration, and *distinctness* of the impression; (2) upon the condition of the power of attention (3) upon the supply of old memory pictures related by association to the new impression and the power of reproduction of these pictures.

The power of apprehension is *sluggish or defective* in cases of psychic inhibition (?), congenital feeble-mindedness, dementia, mental fatigue, many confused states (concerning illusional falsifications see p. 55). It is *superficial and capricious*, but often very quick in maniacal elatedness; distorted in a baroque manner, for instance in certain catatonic disease pictures and in some hysterical psychoses (compare the *Ganser* syndrome p. 54). Profound disturbances of the power of apprehension may, for instance, be met with in epileptic confusions, while *memory defects* (dementia, hysterical amnesias p. 70) may likewise give rise to disturbances in apperception. With regard to agnosia see p. 112.

The preceding tests will also have given one an opinion concerning the power of apprehension. Other tests,

however, are: (1) The patient is made to name objects observed in everyday life or is to state their use (keeping in mind the possibility of agnosia); or (2) he is made to reproduce dictated short sentences or stories (Köppen; to detect the point in the story).

(3) *Heilbronner's Picture Test*.—The patient is shown one by one a series of individual drawings of well-known objects (Fig. 2)* made up of more and more completely finished pictures. In each instance the question should be put "what does this represent?" The *lowest* picture number in each series which the patient identifies correctly denotes his certainty of apperception. His ability to account for the missing details in the drawings denotes the compass of his apperception (or attention, not forgetting here possible defective impressions). The test loses in value when inattention is present.

(4) *Binet's Reconstruction Test*.—The patient is to find the meaning in a sentence the text of which is wrongly put together:

(a) We about the (b) of adversity in days for a faithful friend
 early travelled find one will not, that one has done
 in country good regret the
 morning the.

(c) When the ship began to sink the life boats were
 overboard; one of them drowned. of
 the rest on the ship were saved by a steamer.
 The pilot and five sailors could not
 brought from the ship. thereafter the men. but
 be in the boats; but had to jump
 launched. wómen and children were first
 crowded the; one of them sank, All

(Children from twelfth year up. In cases of feeble-mindedness).

*Compare footnote on p. 47.

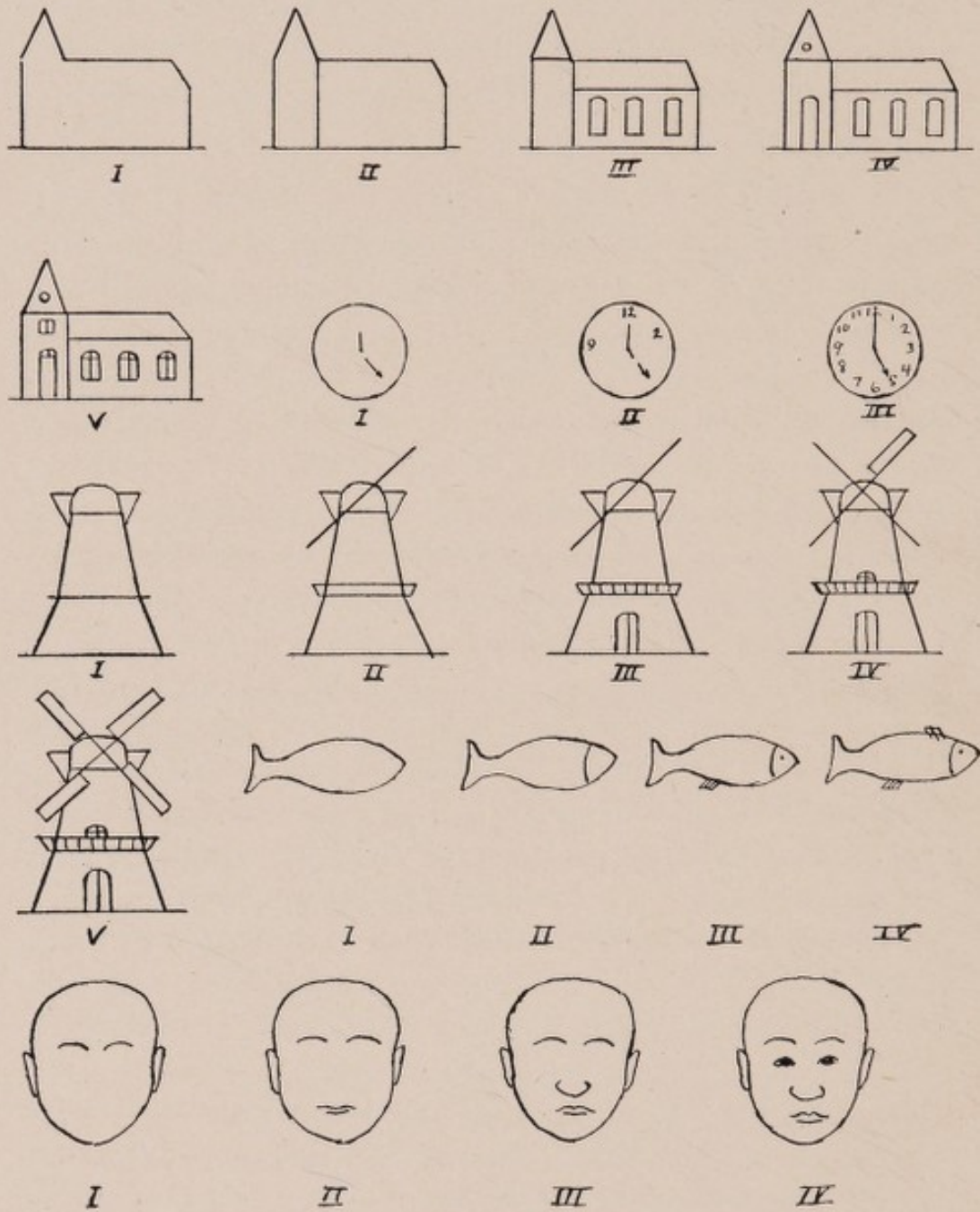


Fig. 2.

V. ORIENTATION

The lucidity of the patient as to time and place, surrounding persons, personal conditions, etc., is of the greatest diagnostic and prognostic importance. Fallacious ideas and hallucinations with full orientation in other directions are often indicative of an incurable disease, while they may be quite transitory in a case of mental confusion, etc. *Information as to orientation must therefore be contained in every clinical history of a mental case.*

The questions in the questionnaire are dealing with information concerning these conditions (p. 31); but the patient may be further asked in what country, in what town he is, and what hour of day it is, the season of the year, etc.

Orientation is most frequently defective as to time, place and surrounding persons (*allopsychic disorientation*), more rarely as to personal conditions ("egoconsciousness"—*autopsychic disorientation*). The patient has forgotten his name, the conditions in his family, his age, etc., thinks he is somebody else, and the like.

Disorientation may be due to: (1) Memory disturbances (*amnestic dementia*, enfeebled recording faculty—p. 69; circumscribed loss of memory p. 69). (2) *Apathy* (mental dullness, certain stupor forms, marked congenital feeble-mindedness). (3) Delusions, fallacious interpretations, etc., (the insane person under arrest takes the physician to be a detective; the paranoic is three months behind the calendar in general use, "because the seasons of the year and the rotation of the earth have been changed by the Catholics").

The condition in which disorientation is most frequently observed is (4) *obscured consciousness*. We dif-

ferentiate between the following *symptom-complexes*, in which the transitions are gradual:

A. *Delirious states*. The train of thought is more or less incoherent, the hallucinations numerous with "fleeting" fallacious ideas. There is usually a self-engrossment sometimes amounting to pseudostupor (p. 24) with changeable or defective apprehension, illusions (p. 55), more or less pronounced "allopsychic" disorientation. We often meet with confused speech-craving and motor restlessness. The mood varies in accordance with the contents of consciousness: Depressive, apprehensive, suspicious, angry, ecstatically happy, etc.

B. *Confusion*. (Amentia.) A marked incoherency of ideas with fragmentary speech is present. It is almost impossible to fix the attention; the power of apprehension is quite defective and illusions, disorientation, disturbances of reproduction (p. 68), hallucinations to a certain extent and fallacious interpretations, etc., are observed. The mood is changeable—apathy, whining, unhappiness, *affects of irresoluteness*, in which latter we find disease-insight present to a certain extent with an uncomprehending, helpless gaze, moping face, disconnected outbursts as: "Well, but is it really—but how—it isn't possible that I—" etc.

C. *Dream states* (twilight state, Dämmerzustand, État onirique): (a) *General*: Essentially psychogenic, now and then following trauma or the effect of alcohol ("pathological intoxication" p. 13). Outwardly the patient's peculiarity is frequently not especially noticeable. He may be somewhat absent-minded, show slight inhibition, irritability, shyness. He may perhaps be able to perform his daily work, is frequently, however, rather awkward and shows noticeable forgetfulness. He may

travel about extensively without attracting attention (poriomania, fugue), may be able to talk quite intelligently about things in general, does not show noticeable defects of apprehension, is not hallucinatory, neither does he reveal fallacious ideas.

More accurate examinations, however, frequently show defects in orientation as to time, amnesic gaps (*lacunæ*) (p. 70), poverty of thought to a certain extent, a clinging to ideas, etc., and especially often a contraction of consciousness (see below).

(b) *Dream states proper*: Dream-like, relaxed or confused train of thought, a greater or lesser degree of exclusiveness from the outer world (*pseudostupor*), marked disorientation (sometimes also *autopsychic*), gross disturbances of apprehension and illusions. Frequently marked apprehensiveness or ecstatic states, reckless excursions in behavior (assaults, crimes, suicides). A general analgesia is often observed.

The dream state is especially profound in epilepsy ("grand mal intellectuel" *Falret*), also in schizophrenic dream states. The *psychogenic* dream states which are often to a certain extent "general" are frequently characterized by the *delirium of reminiscence*; in this we find a *contraction* of the patient's consciousness about a certain group of ideas showing pronounced affects [memories (complexes) wish-fancies], which he *lives over again* in a very vivid manner, showing marked motor reactions with screaming, apprehensiveness or ecstasy—even to the extent of hysterical convulsions. Systematized amnesia (p. 69) leading to disorientation has often been observed. The contents of consciousness may sometimes be distinctly inferred from the patient's conversation. Hysterical stigmata may sometimes be present.

A clinically and forensically important dream state is the so-called *Ganser syndrome*, the chief symptom of which is paralogia: the patient gives a wrong answer or an answer which is so far-fetched that one can detect that the *general idea contents* related to the question have been stirred. This paralogia is noticeable even when asking the simplest questions; as, for instance, "What is 2 plus 2?" "How many fingers on the right hand?" "How many eyes have you?" etc. The patient can not name well-known objects. When asking, for instance, "What is this (leather purse)?" she may say: "Skin and bone," etc. The patient's ignorance seems sometimes more dense than that of a very sluggish case of paresis (*pseudodementia*). There is often a certain stamp of willful or giddy, jesting, meaninglessness in the answer.

The syndrome is usually psychogenic (hysterical stigmata and anamnesis to be looked into), but may also be due to negativism: The paralogia is then as a rule quite meaningless, without being in the least related to the question, and the answers are ambiguous often with here and there an intelligent reply. Even the *maniac* may "talk ambiguously" or "at random" but does it in an intended, willfully elated way. He loses his cue, however, every little while. Finally it must be stated that the *simulator* may now and then barricade himself behind paralogia (more frequently, however, behind mutism), but usually he does it with monstrous, baroque replies, and with a vigilant gaze or concocted sheepish expression. He may also come to forget himself where the questions are especially made to involve affects, when he becomes angry, etc. One should be careful in making the diagnosis "*simulation*," especially where there are sensory disturbances.

VI. SENSE-DECEPTION

We differentiate between illusions and hallucinations, although it may often be difficult to draw the distinction:

1. *An illusion* is a falsified apprehension of external impressions; rust spots in the carpet become blood, the patterns in the wall-paper become faces, an indifferent remark is interpreted as a threat, a fellow patient is recognized as a relative, etc.

Like other disturbances of apprehension, illusions may be due to (a) weak, transitory, indistinct impressions (observed in twilight, at night, etc.); (b) disturbances of attention during pronounced emotions, fear, intense expectancy and the like, and during marked self-engrossment with fallacious ideas (compare p. 63) and complexes of other kinds; (c) disturbances of association, particularly those observed in *obscured consciousness* (mental confusion). Under the last mentioned circumstances the awakening of "identifying" ideas necessary to apprehension is too weak or absents itself altogether (in deep mental confusion), or it leads to the awakening of erroneous ideas (hasty, external resemblance, etc.).

Certain patients see everything in a red color (erythemia), in a brilliant luster; or it is quite colorless, extremely large or extremely small (macropsia, micropsia), or everything shows a lean-to, etc.

2. *Hallucinations*: When a patient states that he hears sounds, voices, sees visions, smells, tastes, feels various things and we are not able to demonstrate the activity of corresponding external sense stimulants, we say that he is hallucinated.

Hallucinations may reveal themselves while filling out the questionnaire, may be expressed in the patient's spontaneous declarations or in his writings. At other

times one may *suspect* them: the patient is markedly self-engrossed (which may be entirely due to fallacious ideas), he stops suddenly in his conversation, his face having an expression as though listening intensely, he gazes into vacancy, takes a frequent peep out of the window, pinches his eyes together, stops up his ears or nose, hides himself, sits about with an ecstatic, blissful expression on his face, shows a sudden explosion of anger, makes violent attacks, shows fits of laughter, etc. One should here be on the lookout for catatonic symptoms, negativistic behavior and the like.

The diagnostic and prognostic *import* of hallucinations is dependent upon whether they occur (1) in connection with *obscured consciousness* ("hallucinatory confusion"), in which case they usually disappear with other psychotic symptoms; (2) where there is *full consciousness* ("lucidity") under which circumstance they often are a bad omen (suggesting a paranoid state, p. 64). There are, however, exceptions such as: Acute or subacute hallucinosis in drunkards and certain degenerative mental diseases where the prognosis is better or quite good.

In connection with hallucinations we must *ascertain* (a) the sense involvement, (b) the contents, (c) the vividness, (d) the localization, (e) the special mode of origin, (f) the disease-insight.

Hallucinations are confined to the domain of one sense or they involve a *combination* of senses (speaking, figures, etc., in which case there may be a gradual transition to delirious experiences). According to sense involvement we meet with:

1. *Visual hallucinations* (visions) which are most frequent where consciousness is obscured, may be plain (gleam of light, smoke, shadows, etc.), plastic or more richly colored (faces, figures, complete sceneries, etc.).

Visions of animals (small, numerous, mobile) are especially characteristic of delirium tremens. Autoscopy is said to be present when the patient sees himself in one situation or another.

2. *Auditory hallucinations*: Elementary (a sound, roar, scream, etc.: akoasmata), complicated (words, sentences and the like: "voices," phonisms).

3. *Hallucinations of smell and taste* (disagreeable odors, vapors, poison in the food, etc.): The patient stops up his nostrils, throws away his food, refuses his food.

4. *Hallucinations of general sensation*: (a) Influences affecting the *sensory organs of the skin* (touch, pressure, pain, temperature). The patient experiences unpleasant contacts, itching, worms in the skin (cocaine habitués), darting pains, sensations of a cold "puff," electric currents. In delirium tremens the patient has a feeling of pieces of money, threads, etc., in his hands, hair in the mouth and the like. (b) *Organic sensations* in the various internal organs: A tightening, an atrophy of the brain, a running out of the spinal cord, a stabbing in the heart, a growing together of the intestines, etc. The sensations are as a rule variously interpreted by the patient (compare hypochondriac delusions). Especially important are the *genital sensations* (unpleasant, painful sensation in the testes, internal sexual organs, sensation of enforced coitus, sexual perverse practices, etc.).

5. *Hallucinations of the muscle sense (kinesthetic hallucinations)* are especially observed in mental confusion and certain forms of paranoia): The bed is moving, shaking; the patient is floating in the air, riding on a train and the like. A peculiar form is the psychomotor hallucination (p. 59).

The *contents* of the hallucinations may be neutral (in-

different words, faces, etc.); usually, however, it is of great importance to the patient and in *intimate connection with his whole train of thought*, especially with the fallacious ideas present. The hallucinations may be hostile, threatening, scolding, upbraiding voices, frightening visions, painful organic sensations, etc. More rarely they are friendly, encouraging, praising, acquitting voices (observed in prisoners, for instance), the voice of God, promises, exalting visions, etc. With regard to contradictory voices, see below.

Especially important are the *imperative* (auditory) hallucinations: challenges to refuse food, to remain immobile, to make certain movements, to escape and to perform violent acts, commit suicide, and the like.

The *vividness* of hallucinations is often but little developed even when the patient is lucid, but when consciousness is obscured, it is with difficulty that one can get an idea of their vividness in any other way than by observing the patient's reaction to the hallucinations. The visions may under such circumstances be indistinct, with uncertain contours; the voices soft, whispering, "in a foreign language" so that the patient does not understand them. At other times he gives a rosy description of them (interpretation?).

Their *localization* (projection into the outer world) varies: the visions may be seen sometimes "within the head, the eye" (some patients admit that they are only living "pictures of fancy"), sometimes in space, or they are seen by the side of, or covering, the real objects. The auditory hallucinations are located "within the head" (said by the patients to be "in reality only thoughts:" inner voices *pseudo-hallucinations*), "in the ears," in space (down in the cellar, outside the window, in the ventilators, etc.).

A peculiar phenomenon is the *hearing of one's own thoughts*. The thoughts of the patient are "repeated," "read," "spoken" (when the patient is thinking, reading, writing, speaking), either in advance of, or as a repetition of his talk which is frequently distorted. Now and again this is associated with motor-sensations in the lips, tongue, throat or "within the chest" and often then combined with precordial anxiety: "Somebody is talking with his tongue" or he feels a craving to express what he thinks (often mean, obscene things): *psychomotor hallucinations*.

Auditory hallucinations may be *unilateral* and may then often be due to organic functional disease of the ears. In cases of *contradictory* (antagonistic) hallucinations we find "good" voices in one ear and "evil" voices in the other.

Sometimes the patient does not know from where the voices come (for instance, in confusion), sometimes not even whether they are human voices or not. Other patients state definitely that they recognize the voices as belonging to men or women, certain persons, etc., admit, however, frequently that the voices seem "distorted," that "they come through a telephone" and the like (for instance, in cases of pronounced paranoid psychoses—paranoid interpretation?).

The chief cause of hallucinations is mental disease. *Factors favoring their development* may, for instance, be the time just prior to falling asleep (hypnagogic hallucinations), the darkness and stillness of night, loneliness (in prison, dark and locked rooms of old style hospitals for insane), external impressions (words heard *in* and *accompanying* the chirping of birds, in the creaking of shoes and the like), peripheral organic disease (oto-

sclerosis, choroiditis, genital diseases, etc.). Hallucinations may also now and then be elicited by organic disease in the particular sensory centers of the cerebral cortex (tumor of the occipital lobe—visions—etc.).

Hallucinations may be developed through *suggestion* in delirium tremens, in some confused hysterical patients (or during hypnosis). The case of delirium tremens will clutch spasmodically to the imaginary whiskey glass that is offered him and empties it with all the signs of evident enjoyment. By pressure on the eyeballs through the closed lids vivid visions are produced (*Liepmann*); from a blank piece of paper he will read a confused array of words. Electric stimulation of the acoustic nerve or tuning forks placed before the ears may give rise to elementary hallucinations of hearing.

The *disease-insight* varies with the degree of intensity of the rest of the psychotic disturbances, especially with that of the obscurity of consciousness and the fallacious ideas present. Marked and more prolonged hallucinations are as a rule apprehended as real, eliciting fear, unhappiness, despair leading to suicide, escape, anger, attacks of frenzy, violent acts, sending for the police, homicidal attacks upon suspected instigators.

VII. FALLACIOUS IDEAS (DELUSIONS)

We should in this connection record the contents, mode of origin, further development and association of the fallacious ideas. With regard to *contents* we have:

1. *Depressive* (melancholic) delusions, usually in connection with depression or depressive inhibition: ideas of unworthiness, self-reproaches, ideas of sinfulness, self-accusations (real or imaginary faults), expectation of future punishment in this life or in the hereafter (with

regard to the patient himself, or his family, the whole world).

In melancholics along in years we often find economic fallacious ideas (the patient has ruined himself, the family, will end in the almshouse, will die from hunger, is to be punished because he can not pay his taxes and the like).

2. *Hypochondriac* delusions (compare organic sensations, p. 57): The patient has cancer, syphilis, consumption, rotten kidneys, withered limbs, does not pass any urine, bowels have not moved in an age, etc. (The delusions are especially baroque in senile cases and in general paresis.)

The forms of delusions under 1 and 2 may lead to the development of *nihilistic* delusions (ideas of negation): the patient is not alive, is a living corpse, is petrified, the entire world is dead, etc.

Depressive and hypochondriac ideas show as a rule marked affects often combined with abnormal motor reaction: wandering about ("melancholia errabunda"), surrendering upon confession of imaginary crimes, refusal of food, attempts at suicide during attacks of "raptus melancholicus" due to fear and the like. A *blankness of affects* is observed where a sluggish mental condition is in the background of the mental state (in dementia precox for instance): the patient rehearses his complaints in a monotonous, jabbering manner with an embarrassed smile, and has new complaints for every day that passes, etc.

3. *Persecutory* ideas (delirium of persecution): The patient thinks himself pursued, spied upon, insulted, treated spitefully, thinks his life and property have been plotted against, etc.

In the *physical form of delirium of persecution* the patient thinks himself subjected to unpleasant bodily influences (organic sensations), which he attributes to his persecutors, who act upon him through electricity, telepathy, x-ray work, etc.

In old people ideas of *having been robbed* are frequent: Neighbors, relatives rob them of their belongings, defraud them of their money, and the like.

4. *Grandiose ideas* (expansive delirium, megalomania). The contents of these ideas varies with the patient's intelligence, degrees of refinement, ideals, wealth of imagination according to the nature of the disease (see below): The patient is exceedingly talented, a universal genius, an Edison, the most powerful man in the world, the leading tenor, multimillionaire, community reformer, prophet, Christ, the bride of God, of noble birth, king, emperor, princess, the most beautiful woman in the world, etc., etc.

Persecutory and grandiose ideas are sometimes described together as *paranoic or paranoid ideas*, which as such are without decided diagnostic significance, this being dependent upon (1) other coexistent psychotic disturbances, (2) the *mode of origin* of the paranoic ideas, and (3) their *further development* or course.

(a) As an accompanying symptom paranoic ideas may be observed (1) in *diseases of a distinct manio-depressive type*: In maniacal excitement we often find *grandiose ideas*, more or less florid. They are as a rule multiple, changeable, quickly abandoned, not connected or much elaborated, often with a distinct impress of half-conscious boastfulness and accurately paralleling the waves in the excitement. The melancholic patient may speak of himself as being "persecuted," but seeks

the cause in his own badness, etc. In degenerative or psychogenic disease of the affects the paranoid ideas (mostly persecutory) are of a more lasting character.

2. In cases where the *consciousness is obscured*, we find them usually multiple, transitory, seldom of more connected or elaborated character (delirious states), commonly with marked involvement of affects, intimately mixed with sense-deceptions.

3. In certain demented or stuporous conditions: The grandiose ideas in the exalted stage of *dementia paralytica* are baroque, beyond all bounds (Planetary president and the like), without sense (the patient's watch runs an hour faster than all other watches in the world), rummaged together with a change from day to day—the patient being leading tenor, emperor of the world, multimillionaire, and laborer making \$26 a week, all at the same time. The delusions are often much more childishly deficient—the patient is “so healthy,” has “lots of money,” “beautiful children,” etc. He is usually extremely generous—makes presents of millions to his physician, appoints him to some high position, and the like. As the dementia progresses, the ideas gradually fade away, leaving at the most a demented euphoria. The somatic signs are of decided importance in making the diagnosis.

In *dementia precox* (paranoides) there is often an incipient depressive, catatonic restless or stuporous phase, or these symptoms develop later on. There is also usually a pronounced “schizophrenic” trend (thought abstraction, mannerisms, stereotypies, negativism, etc.). The fallacious ideas are as a rule multiple, changeable, poorly put together in rhyming fashion (systematized), frequently indicative of baroque physical influences.

After a shorter or longer period, a distinct mental deterioration results with marked disorganized delusions. The paranoid delusions developing at (a) *the presenile age* are usually also quite effaced and changeable, and symptoms of dementia become quickly added to them. In *alcoholic* paranoid states an acute or subacute hallucinatory introductory phase is frequently observed, hallucinations being as a whole the dominating feature of the disease picture. There is but a slight tendency to a rhyming display of delusions and the patient often assumes a noticeable apathetic attitude toward the threatening and scolding voices, etc. In such cases signs of deterioration also quickly set in.

(b) As the chief symptom the paranoid delusions occur in the *paranoid psychoses proper*: (1) In *acute or subacute forms* with now and then a slight obscurity of consciousness ("acute paranoia," delire d' emblee); they are expansive, persecutory in character or a mixture of both (presenting at times a few depressive hypochondriac ideas), well elaborated, up to a certain length of time stabile with marked emotional reaction, frequently accompanied by numerous hallucinations. The patient attempts to a certain extent to make "jingles" of his delusions, to find the cause for his persecution, to find an explanation for the high position, etc., which he thinks he is occupying (often through memory falsifications, p. 70). There are no schizophrenic features about the disease picture. It may terminate in recovery or may leave behind certain uncorrected fallacious ideas (residuary delusions) or its further course may be in the direction of a chronic paranoia.

(2) *Chronic paranoid states* (paraphrenia, paranoia). In rather rare instances (paranoia vs. paraphrenia sys-

tematica) it may take quite a distinct course through different phases: (a) *An incubation period*, during which there is a general feeling of uneasiness with a feeling on the part of the patient that everything refers to him (*reference delirium*) or he interprets all in this direction (*interpreting delirium*). (b) *A period of persecution*: He feels certain that a persecution directed against himself is going on, and begins to weave his various morbid experiences into a web constituting an explanatory *system* in which "anthropomorphosis" prevails: The patient is pursued by a definite person, a conspiracy, for a definite reason. At the same time hallucinations are usually experienced which are predominantly auditory, rarely perceived as due to physical influences (in psychoses of the climacteric period in former alcoholics). In the course of years (c) *the grandiose period* becomes added, which usually embodies a single, well elaborated idea, logically tied up to the persecutory delusions and sometimes combined with expansive hallucinations. After many years we finally have (d) *the terminal stage*: The emotional life grows dim to some extent, there is some disorganization in the system of delusions, but *no typical schizophrenic features*.

The disease picture is often more simple in character: Slowly developed paranoic delusions, which are usually either persecutory or expansive, not especially numerous, prolonged, with fixed systematization, almost without hallucinations, without mental deterioration or schizophrenic features. The condition remains unchanged through decenniums: *paranoia persecutoria* (p. *querulans*, p. *sexualis*, p. *of jealousy*, etc.); *paranoia expansiva* (*inventoria*, *reformatoria sive religiosa*, *erotica*, *originaria*).

The patient quite frequently expresses himself openly

concerning his paranoic delusions, reveals their presence in his letters, in his behavior. At other times he secludes himself in complete silence or *dissimulates*, speaks heretically of his former ideas as "imagination" (the diagnosis "recovery" should be made with caution).

VIII. IMPERATIVE IDEAS (OBSESSIONS)

1. *Imperative ideas* (the imperative thought habit): In the presence of lucidity and without connection with momentary train of thought, ideas turn up, which are *constantly forcing themselves* upon the patient, and which he recognizes to be *morbid* but thinks *his own*, which are accompanied by a more or less pronounced *fear*, and the disappearance of which he usually feels as a *relief*.

The *contents* of the imperative thoughts vary greatly. Several forms are often found present in the same patient. The phenomenon is sometimes *persistent*, constituting the patient's principal mental disturbance ("obsessional psychosis," *maladie du doute*—found usually where there is a psychopathic constitution), sometimes it is remitting-intermittent, now and then it is met with in manio-depressive insanity.

(a) *Imperative brooding* (query-mania) is applied to a condition in which more or less indifferent, baroque, "metaphysic" questions crowd themselves upon the patient, such as: "Why do men exist?" "Why are there two kinds of people?" "Why is man to die?" "Why have we winter and summer?" etc. At other times the patients are imperatively obsessed with words accidentally heard, fragments of melodies and the like. (b) *Insanity of doubt* (*folie du doute*) in which there is a constant doubt as to whether the patient has performed a certain act or not, has done a thing correctly (locked

the door, stamped a letter, written correctly, etc.). (c) *Phobias*: fear connected with doing certain daily duties, such as walking across public squares has been termed agarophobia, remaining in a closed room claustrophobia; making an attack on themselves or upon some other person with sharp objects lying about, as knives, scissors, etc., is aichmophobia (belanophobia); becoming contaminated or infecting other persons by coming in contact with door knobs, seats of toilets or by spitting, is mysophobia, or *delire du toucher*; fearing that other people will detect their blushing is an erythrophobia and the like. (d) Imperative incitements (impulsions): to make use of vulgar or uncouth words is known as coprolalia; to count everything that one comes across, arithmomania; to destroy things belonging to himself or others, elastomania; to set fire to property, pyromania; to steal, kleptomania; to run away, dromomania; to drink, dipsomania and the like. Practically speaking, it is rather uncommon for the patient to yield to the impulse.

2. *Autochthonous ideas*: lucid, imperative *ideas forcing* themselves upon the patient that have no apparent connection with his momentary train of thought, the ideas being felt by him as *foreign* to himself and toward which he assumes a critical attitude—interpreting them as due to external influences and the like. These obsessions are observed in schizophrenia, paranoia and psychopathic states.

3. *Overvalued ideas* (“überwerthigen Ideen”-Wernicke) are constantly obtruding complexes with marked affects (p. 32) which the patient only in part has sought to expel from consciousness, which are not felt by him as being foreign and which often lead to the focusing of the train of thought in a particular direction, for in-

stance in the form of reference ideas, interpreting delirium and the like, as observed in psychopathic individuals.

IX. MEMORY

We differentiate between memory for recent impressions (*recording faculty*) and memory of older impressions (memory in a more restricted sense), and with regard to the latter we again distinguish between the *memory contents* and the *power of its reproduction*.

The three sides to memory may suffer individually or in various combinations.

(a) *The recording faculty* is solely or predominantly suffering, for instance, in certain senile psychoses (presbyophrenia), in alcoholics (the Korsakow syndrome), in traumatic insanity (concussion psychoses), in more pronounced confusions and manias, frequently in neurasthenics and in hysterical patients. In the latter cases there is often simply a "retarded memory," i. e., the remembrance turns up when the patient has had a chance to "pull himself together" or it develops spontaneously hours or even a day afterwards. In catatonic disease pictures we find that in spite of the thought abstractions present the recording faculty remains as a rule intact or "capricious."

(b) *The memory contents* proper becomes diminished in organic brain diseases, now and then leading to the formation of elective lacunæ (arteriosclerotic dementia, compare also aphasia, p. 108), but more frequently producing a diffuse or progressive form to an extreme degree (paresis, senile dementia).

(c) *The power of memory reproduction* often fails in the diseases mentioned under "b," but may also frequently do so where the stock of memory is in itself

intact, as where one is dealing with an inhibition of reproduction (functional loss of memory): (1) This *inhibition* may be diffuse. Such an example may be met with in inhibitory mental depression or stupor, in hysteria, neurasthenia, schizophrenic states with obscured lucidity. Care must, however, be taken not to confound it with schizophrenic block and negativism. In this form it is also observed in nervous diseases following trauma capitis, etc. Inhibition of memory reproduction must not be confounded with dementia.

(2) The form of memory inhibition may be localized, as in amnesia, which is understood to be a loss of memory for chronologically coexisting recollections, for an individual experience, covering a more or less prolonged period of time, during which we find either a normal state of consciousness, as in retrograde amnesia following trauma capitis, convulsions, strangulation, poisonings, etc., or we find psychic disturbances as in "anterograde" amnesia in which there may be confusion, dream states, a markedly impaired recording faculty, etc. We sometimes meet with a combination of symptoms, i. e., retro-antegrade amnesia, in which the loss of memory may be more or less complete (concrete memory), prolonged or only transitory or alternating (see below).

Circumscribed losses of memory occur most frequently in *psychogenic* (hysterical) psychoses and are sometimes systematized: The patient has forgotten all recollections referable to a certain person or thing, remembering on the contrary other things from the same interval of time. In case of *alternating* amnesia ("double individuality") two or more phases of consciousness alternate with the "special memory" incidental to each; i. e., in phases of consciousness A and C we find a memory for phases B and D present, but not the reverse, etc.

Defective memory in the manner of difficulty in keeping track of conditions of time, place, etc., keeping as a whole recollections distinct from one another, are seen in organic brain diseases, epileptic psychoses, certain congenital feeble-mindedness, etc.

Memory falsifications, paramnesia, are either an *illusional* transformation of actual experiences (for instance when associated with affects; compare also interpreting delirium) or *memory hallucinations*, i. e., a production of quite independent imaginary experiences, which to the patient have a stamp of recollections. This is the case for instance in mania, dementia paralytica, where it sometimes makes the impression of an embellishment of past events or of imaginary achievements, in mental depression (self-accusations in relation to imaginary crimes) and in certain forms of paranoia (retrospective interpretations).

Especially vivid and complicated falsifications of memory (*confabulations*) are met with in certain senile psychoses (presbyophrenia), in many parietic patients and in the alcoholic Korsakow psychoses: The patient fills up the loopholes or lacunæ in his memory with the most minute fabrications, doing it spontaneously or when questions that are somewhat suggestive are put to him. He describes all sorts of experiences on the same day, visits about town, various achievements in work, etc.; he relates everything in a matter of fact, plausible manner, frequently without it being to the least extent necessary for him to reflect upon what to say.

Pseudologia phantastica (mytomania) signifies an abnormally intense activity of imagination, observed in many psychopaths, resulting in the production of more or less fairy-tale-like fiction which the patient either

keeps to himself ("day dreams") or of which he makes a great display, in the form of boasts, projects, or more active grandiose ideas. It is observed in swindlers, adventurers and pathologic liars. Fabulizing with malicious denunciations is especially observed among hysterical patients.

Memory Examination Methods

1. The *recording faculty* is tested: (a) By dictating a few numbers in four figures or difficult words which the patient is to repeat after 1, 15, 30, etc., minutes, the patient being diverted by an easy problem in arithmetic; for instance 3×9 or the like. (b) A repetition by the patient of some short newspaper notice, history or the like, which has been read by or to him. (c) Five pictures (portraits, single geometrical figures and the like) are shown the patient for about half a minute after which he is to select them from among a series of 20 other pictures. A still more exact test is (d) *Ranschburg's word-pairing test*: Nine (eighteen, if indicated) pairs of words are read to the patient once or twice (he is to repeat them at once to insure against mistakes in hearing). After 1, 15, 30, 60, minutes, 6-12-24 hours the first word in the pair of words is told to him to which he should normally be able to add the other word in the pair. My word pairs are:

Cow-meadow; street-people; old-crutch; forest-deer; song-joy; table-cloth; window-sun; carriage-dance; letter-greeting; house-small; flower-red; room-large; gown-beautiful; chair-heavy; tree-tall; garden-old; horse-white; street-broad.

2. The *memory contents* must always be examined with regard to the individual conditions: the patient's sex, age, social surroundings, the nature and duration of the

school education, the time directly following this, the special training or experience, interests, etc. Of greatest importance is (a) memory of *personal experiences*, about which information should already have been secured, in making anamnestic inquiries of the patient. In addition to this he may be questioned concerning: place of birth, details about parents (whether living, age, deceased, when), sisters and brothers (number, name, age, lot in life); attendance at school (place, how far along, standing); interest in religion, occupation, position occupied (place, time and duration); whether he has moved about the country much, where and how long; whether married, when, the whereabouts of wife and children, the number and ages of the latter, important events in the domestic life, etc. He should also be questioned as to *historical events experienced*: catastrophes of nature, important social or political events, the world war, great personalities living and dead, particularly within his special sphere of interests, etc. It is sometimes of value, where possible, to have the patient give a biography of his life. The questions should in every case be formulated in accordance with what the patient may be expected to present in memory content. This is still more true with regard to inquiry into his general knowledge.

(b) *General Knowledge*. We should ask ourselves: Is the patient possessed of such information as would be in keeping with his daily work and special education? A waiter in a restaurant should be able to do simple figuring, remember prices of dishes in a general way, remember the names of certain restaurants or hotels in the town from which he comes, etc. The mechanic should be able to name places where he has worked, people under whom he has worked, the wages he has been

getting, the facts concerning unions, strikes, and non-union strikers, etc. The farm hands should give facts about agriculture in the way of preparing the soil for crops, etc.; the clerk, the names of various classes of goods he has been dealing with, prices, firms, etc.; the book-keeper, facts about accounts, bookkeeping, firms, etc.; the college man, information concerning his particular profession. The housekeeper should be able to answer questions concerning housekeeping duties, cooking, etc.

In making inquiries as to schooling it is always more or less difficult to draw the line. Normally we find a great deal has been forgotten, especially where the patient later in life has paid very little attention to book knowledge. The school instruction may have been defective, the patient, in spite of natural talents and good memory, indolent, etc. The retention of a certain minimum average should, however, be required.

The memory questionnaires in general use (Sommer's, Ziehen's, Kraepelin's) may be found of practical value, especially in congenital feeble-mindedness and advanced forms of dementia. The answers are noted down in the patient's own language.

1. Arithmetic. Easy and more difficult examples in the four methods of computation in written or mental arithmetic. This is of special importance in the case of patients who, prior to their illness, had much to do with figures, computations, etc. Many people devoting much of their time to reading in general, especially women, are habitually poor in arithmetic.

2. Which is the largest city in the United States? In the world? What is the population of each? What is the capital of France, Germany, England, etc.? What country lies north of the United States? What country

south? How can you get from Chicago to New Orleans? What are the chief signs on the compass? What causes the changes from day to night and the seasons of the year?

3. Into what chief classes are the animals divided? What animals lay eggs? To what chief class does the bat, the whale, belong? What kind of metals do you know? What different kinds of money are in use in our country? Of what denominations? Mention the different denominations in weight used in the United States; the different measures. How many seconds in a minute, minutes in an hour, hours in a day, days in a month, in a year? How many days in a leap year? Give the names of the days in a week, names of the months, give serial names backwards.

4. Who was George Washington? Abraham Lincoln? General Grant? Roosevelt? What was the war in 1861-1865? When was the Revolutionary War? Who was Napoleon? Shakespeare? Walter Scott? Mark Twain? How do laws, statutes come into existence? What is Congress? What does the President do? What are the names of the chief political parties in this country? Why do we pay taxes? How can one manage to borrow money in a bank? What different creeds or religions are there? Why do we make use of bread and butter, wool, etc.? What is a thermometer? Of what use is it?

X. INDIVIDUAL MENTAL WORK (AUTOPSYCHIC ACTIVITY)

The patient's train of thought, power of apprehension and the accuracy and wealth of his memory reflect his "intelligence," looking at it from one point of view. They are at least the essential presuppositions to his *individual mental activity*, his power of judgment, discrimination, foresight, mental independence and initiative,

his perseverance, ability to combine, and his imagination, etc. However, despondent inhibition may be associated with intact independent mental action. The "slow learner" is in fact able to judge clearly and independently—a qualitatively sound mental contribution may be made with a small memory-fund at one's disposal, etc. Our last problem in the examination of the function of consciousness of the insane, is therefore to test this independent mental activity of the patient.

It is self-evident that scheduled tests which are always very elementary can in this instance but inadequately meet the numerous individual possibilities of variation. We must therefore first and foremost draw conclusions:

1. From the patient's *general behavior throughout life*: The results of his life's work, his conduct when facing certain situations which make demands upon his power of judgment, foresight, psychic independence, initiative, ethic principles, etc. His purely *external achievements*, such as results at examinations or his promotions, economic productions, etc., give us an opportunity when considered in connection with preexisting influences (original environments, education, chances, etc.) to form an estimate of his individual method of mental work. We finally may also obtain information concerning these circumstances from his relatives. In this connection it must, however, be remembered that *variations in feeling-tone* (maniacal or melancholic phases) will not infrequently detract from the results of mental achievements, otherwise considered good, such as frequent change of position, failure at examinations, etc.

2. Through *conversation* with the patient on different subjects (general and personal), by *observation* of

his daily doings, his conduct toward surroundings, his reactions, interests, occupations, amusements, etc.

3. From objective inquiry into his activity, during which we make use of the patient's reactions to certain questions and tests (his replies should be stated word for word), which experience has taught us may often reveal intellectual defects, but naturally only in the connection in which the test is applicable (many, otherwise intelligent people, find it difficult to understand witticisms, etc.). The tests in general use are:

(a) *Questions in definition* (Ziehen, Binet): What is a bird? a butterfly? a thunderstorm? a table? a chair? a horse? a martyr? a swindler? a judge? Superficial or automatic replies may here be considered satisfactory. What is sympathy? envy? mischievousness? self-denial? gratitude? simplicity? uprightness? Here we are making use of the abstract. The normal child dealing with concrete ideas will often "illustrate" with certain instances, usually self-experienced, likewise many "uneducated" adults—especially women. The congenital feeble-minded will not infrequently "define" in many words, but in reality miss the essential. The same is sometimes true of the demented.

(b) *Questions in differentiation*: What is the difference between a cow and a horse, a child and a dwarf, a lake and a river, water and ice, mice and bats, a bird and butterfly (these are in part "questions of knowledge"); covetousness and economy, mistake and falsehood, belief and knowledge, sympathy and importunity, confidence and conceit?

(c) *Catch questions*: Which is the heaviest—a pound of iron or a pound of lead? and like jokes (Ganter): Gentleman, "When downtown this morning I ran across Mr. Miller." Lady friend, "What, you didn't hurt

him?" Both forms of the test are, however, of very limited value (see remark above).

(d) *Henneberg's picture test*: The patient is shown a continuous series of pictures (humoristic, a dangerous situation). He is expected to be able to interpret them.

(e) *The renarrative method* (p. 49), the reconstruction test (p. 49) may also give one an opinion of the patient's autopsychic activity.

(f) *Questions concerning conduct* (Binet) especially applicable to children (compare p. 81), congenital feeble-minded, demented: What would you do, supposing that you are on the way to the train and should find you are too late for it? When you have happened to destroy something that belongs to someone else? What should one do before he acts? What are you going to say in case people ask your opinion concerning a person that you do not know very well?

(g) *Masselon's test*: A sentence expressing good sense is to be formed from three words; such as, hunter—rabbit—gun; dog—man—ruffian; bicycle—wagon—boy; child—window—hospital; hunger—rich—children, etc. The test is also adapted for the illustration of disturbances in the course of idea associations (flight, inhibition, divertibility).

(h) Of special importance is *the completion test of Ebbinghaus*: Different words or, less satisfactorily, parts of words are omitted in a coherent narrative and the patient is then told to fill out the lacunæ correctly, i.e., in such a manner that the intended meaning is brought out. It is best to make use of several texts:

1. An innkeeper had four —. One of these was a poodle. On winter evenings — used to come into the room and lie down near the — stove. One evening the poodle came —, and the other — had occupied all the — places by the —, whereupon — ran for the door

and began a loud —. When the other dogs — it, they — ran to the door and began — with the poodle. The — then returned and occupied the — place by the —.

2. My friend in S had returned home and I — to surprise — with a — visit. I secured a heavy — as the weather was —, and — to the railroad station. But when I — there the — had just —; my — was found to be half an hour —. I then decided to — to S. It had been snowing — and —times I got — in snow —. I finally — S and my friend's —. He was very — to — me, shook my — heartily, gave me dry — to put on and seated — by the warm — to enjoy a — drink. After enjoying a hearty —, a pleasant — and a game of —, I went to — and — soundly until the following — when my friend had to — me in time for —.

(i) *Ethic concepts*, ideals and the like. Upon questioning in this direction we are of course only able to get information concerning the patient's theoretic knowledge. Whether this has a corresponding influence upon his behavior must be ascertained in some other way (see above). Sometimes the patient will answer the questions propounded in the next paragraph by quoting an instance in illustration or by citing a personal experience in which case one often gets a good insight into his moral habitus:

To whom do we owe love, respect, gratitude, sympathy? Why does one love his parents? Whom do you love the most, and why? Why should one not lie, steal, set fire to one's own house? What would you do in case any one should by accident strike you, if he should do you harm, injustice or cause you grief? Which is worse, to slander a person or give him a whipping? What would tend to make you most sad or most happy? What would you do supposing you had found a purse containing \$100? If you should draw the big prize in a lottery? What kind of an occupation would you prefer? What

person would you like to resemble, and why? Whom do you admire most and why?

In making all "tests of intelligence," one should not only give consideration to conditions in general (page 72) but we should also consider (1) the greater or less ability on the part of the patient to express his thoughts and feelings in words; i.e., to think abstractly; and (2) one should also note the presence or absence of "examination excitement," disturbances of attention, of thought inhibition, or "thought block."

RECOGNITION OF CONGENITAL FEEBLE-MINDEDNESS

In order to recognize this, it is of importance to know the conditions of mental development of the normal child.

Binet and Simon* (Prof. Alfred Binet, Paris, 1905) have grouped a series of tests for the purpose of ascertaining whether the development of the child to be examined corresponds to what is considered normal for its particular age (third to fifteenth year):

Imbeciles

MENTAL AGE

3rd year:

1. Point to your nose, your eye, your mouth.
2. Listen well and repeat what I say: 3-7; 6-4; 2-5. Full credit given for one exact repetition.
3. Place picture before the child and ask: "What do you see in this picture?" (recognition and understanding of detail).
4. What is your name — and your other name? Surname required.
5. Listen well and say what I shall say: "It is warm, I am thirsty." (There should be no mistake.)

*According to Dr. Oerum's version in *Bibl. f. Laeger*, 1914, p. 288.

4th year:

1. Are you a boy or a girl?
2. What have I in my hand (key, knife, coin)?
Name of three objects required.
3. Repeat these figures: 7-9-4; 2-1-6; 5-3-9.
(One of these series must be repeated correctly.)
4. Which is the longest of these two lines?
(The test must be solved correctly.)

5th year:

1. Tell me which is the heavier; differentiating between weight (boxes weighing 3 and 12, and 6 and 15 grams).
2. Copy the drawing of a square 3 to 4 centimeters in diameter (with pen and ink).
3. Listen well and repeat what I say: "My name is Conrad, pugh, the nasty dog."
4. Place four pennies in a row and let the child tell how many there are. No error allowed.
5. Putting together of a visiting card cut diagonally after showing the child an intact card like the one cut.

6th year:

1. Is it morning or evening now (forenoon or noon)?
2. What is a fork, a table, a chair, a horse, a 'mamma'?
(Three replies should be correct.)
3. Let the child copy the drawing of a rhombus with pen and ink.
4. Adding together of 13 pennies: Count these pennies for me, pointing to each one as you count it.
5. Three beautiful and three ugly faces are shown alternately: Which is the prettiest of the faces, etc?
(No error allowed.)

7th year:

1. Hold up your right hand—point to your left ear.
(No error allowed.)
2. Description of picture (as to contents, and the "point").

3. Execution of complicated problems (in proper consecutive order).
4. Adding together of three one cent pieces and three two cent pieces (no mistakes after 10 seconds).
5. Naming of four colors (red, yellow, blue, green—no mistake after six seconds).

Morons

8th year:

1. The difference between a bird and a butterfly, between glass and wood, paper and cardboard (the exact difference in two of the tests must be satisfactory).
2. Counting backwards from 20 to 0 (not more than one mistake in twenty seconds).
3. Discovery of errors in four defective drawings of persons (p. 47), at least three satisfactory replies.
4. Statement of day of the week, the month, the year (no mistake).
5. Repetition of figures: 7-9-5-9-2; 9-1-3-5-7; 2-5-8-1-3. One group at a time (one response must be correct).

9th year:

1. Easy subtractions: 20-16, etc., or going through a game of storekeeping.
2. Definition of fork, table, chair, horse, mother (three good definitions).
3. The coins in general use must be recognized looking at obverse side.
4. Enumeration in order of the months of the year (twenty seconds, only one error or transposal allowed).
5. Questions concerning conduct or behavior: If one should arrive too late for the railroad train, what is he to do? If a companion should strike you by accident without intending to do so, what would you do? When one has destroyed a thing belonging to someone else, what should he do about it? (Two good responses required.)

10th year:

1. Five paper boxes of unequal weight (3, 6, 9, 12, 15 grams) are to be arranged according to weight (three trials made, the boxes mixed after each. Two successes in three are required).
2. Copy the drawing of figures shown (prism, *a la greque*; 10 seconds). Full credit given if the whole of one drawing and half of the other is reproduced exactly.
3. Dictation of sentences having no sense to test appreciation of absurdities: (a) I have three brothers, John, Willie and myself. (b) Yesterday I visited my deaf and dumb aunt, who is always talking about her youth. (c) A railroad accident occurred yesterday which was not very serious, only 30 people were killed. (d) A bicycle rider fell and crushed his head. He died immediately. They fear in the hospital that he can not live. (e) I met a fine gentleman with his hands in his pockets. He went along swinging his cane (correct solution of three of five tests required).
4. Questions concerning conduct: (a) When it is time to go to school and you fear that you might be late, what are you to do? (b) What should one do before taking part in something of importance? (c) What would you say if one should ask you if you like a person you do not know? (d) Why should one judge a person by his acts rather than by his words? (Twenty seconds for each reply; three satisfactory replies.)
5. Masselon's test: Write the words, "hunter, rabbit, gun" and tell the child to write a sentence containing all three words (one minute allowed for the test; allowing two sentences having sense).

12th year:

1. Differentiation between lines of unequal length.
2. Masselon's test (the words must be embodied in one sentence).
3. Calling out as quickly as possible of nouns (for instance, table, bread, house, wagon, etc.—at least 60 in three minutes to be accepted).

4. What is charity, justice, kindness (should give two satisfactory explanations)?

5. Reconstruction test:

(a) we into	(b) to ask translation	(c) a defends and
early went	my I have	dog good his
in the country	teacher	master courageous.
the morning.	correct my.	

(One minute to be allowed for fixing words properly in each sentence: two must be satisfactory.)

15th year:

1. Repetition of figures: 9-6-4-0-5-1-8; 7-3-8-4-2-6-1; 5-9-2-8-0-3-7. (One success in three required.)
2. Three words to rime on a given word (man, old, etc.)—must be found in one minute.
3. Listen carefully and repeat exactly what I say: "The other day I saw on the street, a pretty yellow dog. Little Morris has soiled his nice new apron."
4. Test with pictures. Interpretation required showing logic and imagination.
5. (a) "A man, who was walking through a park, stopped suddenly greatly frightened and ran to the nearest police station to report that he had seen — in a tree." After a pause ask, "A what?"
(b) "Different persons have been seen going into my neighbor's house, one after another, a physician, an attorney and finally a priest. What is going on at my neighbor's house?"
(Both problems must be answered satisfactorily.)

One should begin with a test, which corresponds to the age of the child. If that is not solved, one should try the test corresponding to the age just below this, etc. Normal children may sometimes be able to solve tests corresponding to those of an age above them.

Defects amounting to an inferiority of one year need not indicate mental enfeeblement. The idiot does not exceed the third year level. The extreme or medium degree imbecile does not reach above the fourth or fifth

year level. The mild degree of imbecility is on a level with the seventh year of a normal child. The moron (debile imbecile) is on a level with the ten-to-twelve-year-old child. The schedule is especially adapted to mass examination of children (in the public schools, schools for backward children, schools for defectives, etc.), or where it is desirable to follow the intellectual development of children extending over years.

The Binet test has been improved and revised by Dr. H. H. Goddard (Vineland, New Jersey, 1911). The tests in Goddard's intelligence "scale" are divided into a number of groups. Each group consists of four or five tests and represents a year. The person examined is said to have a certain "mental age," which is considered equal to the highest year, which he is able to pass in all the four or five tests of the group, plus a fraction representing the number of tests expressed as fifths of a year, that he passes in the groups beyond the one in which he passed all the tests.

"The point scale" (Yerkes, Bridges and others of Boston—Warwick and York of Baltimore, 1915) presents a series of tests arranged in the order of their difficulty, and is not, like the Binet test, based upon a subdivision according to age groups. It is designed to test the various mental functions, such as auditory memory, motor coordination, ideation, kinesthetic discrimination, logical judgment, etc. A certain value expressed in points is attached to each of these tests. The sum of these values, i.e., the score attainable by the completion of all tests, is 100 and the score attained by the individual examined, is the sum of the points assigned to the tests in which he is successful. Part credit is given for partial completion of the tests, thus abandoning the

“all-or-none” principle of the Binet system. The final score is then compared with the norm, which should be made a variable quantity, allowance being made for favorable or unfavorable circumstances of environment and training. The setting of the norm may also be determined by a direct comparison of the scores of individuals belonging to the same group and need not be left to the personal judgment of the examiner.

The war department of the United States Government has recently introduced “a scale of performance” test, which is especially adapted to group studies of adults. The test can be applied to 60 to 80 men at a time and completed in 45 minutes.

CHAPTER III

THE SOMATIC STATE

Scheme of Examination

1. External habitus. State of nutrition, color of skin, telangiectases, perspiration, edema, scars, bruises. General bodily development. Cranial deformities.

2. Internal organs. Heart, blood vessels (arteriosclerosis), pulse; lungs, respiratory disturbances (Cheyne-Stokes); difficulty in swallowing, vomiting; abdominal organs. Examination of urine; bodily temperature.

3. Organs of sense. Exophthalmus, ptosis, conjugate deviation, nystagmus, ocular movements. Pupils: size, reaction to light. Visual power, field of vision, ophthalmoscopy. Sense of hearing, smell, taste.

4. Speech. Dysarthria, scanning, syllable-stumbling, aphonia, mutism, paraphasia, word-deafness, alexia, agraphia, agnosia, apraxia.

5. Motor disturbances. (a) Spasms. Clonic, tonic, hysterical; local, unilateral, general. Chorea: athetosis; tremor (at rest, intentional). (b) Paralyses: topography (peripheral, monoplegic, paraplegic, hemiplegic); degree; muscular tonus (contracture); state of nutrition (electric reaction).

6. Reflexes. (a) Skin reflexes: plantar reflex (Babinski reflex); abdominal, cremaster reflex. (b) Tendon reflexes: patellar, ankle reflex (clonus); arm reflexes. (c) Vesicorectal functions.

7. Sensory disturbances. (a) Subjective: pains paresthesias, vertigo. (b) Objective: touch, pain, tem-

perature, muscle sense; degree, kind (dissociation), topography (compare under 5b).

8. Disturbances of coordination. (a) Spinal ataxia (finger-nose, knee-heel test; Romberg's test; gait). (b) Cerebellar ataxia (astasia-abasia; adiadokokinesis). Vestibular tests.

9. Blood tests for Wassermann reaction.

10. Spinal fluid. Cell count; albumin determination; Wassermann reaction.

During the initial examination it will as a rule be sufficient to note down (1) the external habitus; (2) the examination of heart, blood vessels, pulse, lungs, urine, temperature; noting (3) the reaction of the pupils to light, a possible conjugate deviation of the eyes, visible ocular paralysis and the like; recording (4) any noticeable speech disturbances (dysarthria, paraphasia); observing (5) any possible spasms or paralysis; examine (6) the plantar and patellar reflexes; and (7) the possible presence of ataxia in arms and legs (disturbances of gait).

1. External Habitus

(a) A noticeable *loss in flesh* with paleness is seen in certain confusional mental states (inanition deliria), often in cases of melancholia. A marked general "fading away" is seen for instance in paresis, while a plethoric habitus is sometimes met with in cerebral hemorrhage and the like. A striking *plumpness* with an otherwise healthy appearance is common in chronic demented states, in the climacterium, etc. Chronic *alcoholism* may reveal itself in plumpness, flaccid features, telangiectases on the nose and cheeks, congested eyelids and the like. An *early aging* in general appearance should make one suspicious of arteriosclerotic or presenile brain disease.

An abnormal blood distribution in the skin is often

seen: Cyanosis, especially of hands and feet with diminished temperature of the skin (in motor paralysis and stupor), spontaneous vasomotor spasms (with pallor and often very slight hemorrhage upon pricking of skin), congestion around pricked area; forceful streaking with the handle of the percussion hammer may cause dermatographia, urticaria factitia with formation of vesicles, but may however also be seen in healthy persons. Increased perspiration (in neurasthenia, morbus basedowii, stuporous states) or hyperhidrosis (myxedema, see below). One should also look for scars in the skin, leucoderma colli, ecchymoses (which may be incurred during convulsive seizures or in apoplexy) edema, ulcerations (gangrene, decubitus), herpes vesicles.

(b) *General Bodily Development*.—Disturbances in internal secretions give rise to various anomalies in growth: (1) *Dwarfism*, (“nanism”) diminished, often defective bodily proportions (too large a head, deformed facial features, long arms and short legs, kyphosis), defective development of genitals, undeveloped secondary sex signs (growth of hair in the axilla, pubes, growth of beard, undeveloped mammæ, childish voice and the like), infantile slenderness or abnormal deposits of fat (“dystrophia adiposogenitalis”), etc. Myxedematous dwarfism (idiopathic; endemic cretinism, then combined with struma); as myxedema in adults, it is characterized by the peculiar change in the skin which becomes pale, dry, slightly desquamating (“mealy”), mellow skin, in coarse folds amounting to bagginess (wrinkles of the forehead), falling out of the hair, hyperhidrosis. (2) *Giantism*: sometimes developed in certain parts (acromegaly disease of the hypophysis): abnormally large hands, feet, bony parts of the face (lower jaw, nose), development of kyphosis.

Sometimes it is more general with or without disturbance of proportions.

When the patient presents secondary sex characteristics of the opposite sex, as regards general bodily build, especially of the chest, hips, mammæ, hair development and voice characteristics, we speak of feminism or viraginity. As physical signs of degeneration (*stigmata hereditatis*) a series of malformations was formally enumerated. A number of them occur in "endocrine" disturbances. They are without importance when occurring singly; when several of them occur together, they may point toward inherited neuropathy.

(c) *Deformities of the Cranium.*—The greatest horizontal circumference of the cranium amounts to 55 to 57 cm. in an adult normal individual, the greatest longitudinal diameter is 17 to 21 cm. and the greatest transverse diameter 14 to 16 cm. In microcephaly the horizontal circumference is below 49 cm., the skull cap is small in proportion to the bony skeleton of the face, the forehead is often low and slants abruptly backwards, the eyes are set close together, etc.

In hydrocephaly the skull cap is abnormally large, forehead broad, projecting beyond the relatively small bony skeleton of the face, the eyes appear to be forced downwards, etc. Rachitis gives rise to a square or bulged ("pear-shaped") skull cap with projecting tubera parietalia et frontalia, flattened occiput, etc.

Other external stigmata of degeneration are: Facial asymmetry. This may be to a certain extent physiologic and must not be confounded with facial paralysis (p. 127), prominent cheek bones, protrusion of the upper or lower jaw, harelip, cleft palate, dental anomalies as to number and emplacement of teeth (which should not be con-

founded with rachitic or hereditary luetic malformations); deformities of the outer ear (Morel's ear, sessile ear lobe, "Darwinian ear");* coalescent eyebrows, "Mongolian eyes," congenital cataract and the like. Malproportioned arms or legs; missing, supernumerary, fusion of toes (polydactylism, syndactylism—often hereditary); malformation of the genitals (cryptorchism, epispadias, hypospadias, atresia vaginæ, etc.).

Concerning unilateral aplasia of the osseous system, see p. 135.

2. Internal Organs

The examination is to be made in conformity with the rules of internal medicine. Heart, blood vessels, lungs, urine are always to be examined, especially in cases of loss of consciousness, mental confusion or paralyzes.

Demonstration of valvular disease, dilatation, myocardial degeneration may become diagnostically of great importance (cerebral hemorrhage, cerebral embolism and the like). Aortic aneurism is very suspicious of syphilitic infection and is often met with in cases of paresis.

The pulse should be taken *repeatedly* as the patients are easily affected. A *slow* pulse often occurs in mental depression, stupor, in organic brain-diseases (tumor cerebri, hydrocephalus; in meningitis even in spite of fever, etc.). A *quick* pulse is seen in anxious states, febrile deliria, neurasthenia, hysteria, Basedow's disease (in the latter case it is quite continuous). *Paroxysmal tachycardia* ("vagus neurosis") is observed, for instance, in hysteria and neurasthenia (not overlooking the presence of myocarditis and arteriosclerosis). *Increase in blood pressure* (blood pressure to be measured

*Othematoma among the insane (paresis, arteriosclerosis, usually from contusions) is a subperichondral hemorrhage, which when absorbed leaves an atrophied cartilage and crumpled ears.

regularly) observed possibly in combination with hard, serpentine blood vessels may make one think of an arteriosclerotic brain disease or atrophic kidney disease.

An examination of the blood (eosinophilia, hyperleucocytosis, leucopenia and the like) is not as yet of a definite diagnostic value in psychiatry. *The Wassermann test* should be made (see p. 172).

Changes in respiration of a purely nervous origin are frequently met with: *Simple tachypnea* (in conditions of fear), *hysterical dyspnea* ("asthmatic," inspiratory or expiratory, without cyanosis or action of the auxiliary muscles, without expectoration or cough, disappearing when patient rests on his back or at night). In coma we often find deep, labored, "rattling" respiration or respiration of the Cheyne-Stokes type: passing gradually from easy superficial inspirations to wheezing, labored ones, and changing back again to superficial respiration, terminating finally in cessation of all respiration for some seconds. The *hysterical cough* is paroxysmal, influenced by external conditions (the rounds of physicians, the humor of the patient); it is "barking," without expectoration or other pulmonary symptoms (an eventual "hemoptysis" always originating from the gums or the retropharyngeal space), and ceases during the night.

With regard to "*aphonia*," see p. 108.

When alcoholism is suspected, the liver and hepatic functions must always be looked into, ascertaining the liver dullness, looking for a palpable edge of the liver, biliary pigment in the urine.

The gastrointestinal canal: Attacks of *vomiting* are frequent in brain diseases (meningitis, tumor, especially of the cerebellum), in tabes (gastric crises), in uremia, in attacks of headache, hysteria, and also in stuporous

cases of mental disease where it may be due to insufficient mastication and overeating.

Vomitus matutinus (principally mucus; associated with extreme nausea) is observed in alcoholists. Vomiting of *cerebral* origin is not usually associated with nausea, is copious and not closely connected with taking of food. The *hysterical* vomiting is capricious (not observed after dishes of which they are especially fond), is frequent in connection with emotional excitement (anoyances) and without visible signs of nausea or exhaustion. The *tabetic* "*crises gastriques*" consist of paroxysmal pains radiating in all directions in the region of the epigastrium where one often finds segmentary sensory disturbances (p. 155). They sometimes lead to prolonged vomiting and dangerous emaciation.

Difficulty in swallowing by reason of paralysis of pharyngeal muscles occurs in organic brain disease (bulbar paralysis) and in postdiphtheritic paralysis. Based upon *spasm of the esophagus* we find it in hysteria where it is paroxysmal, and usually associated with fear.

Very loud ructus is often heard in hysteria, sometimes in connection with swallowing of air which may cause meteorism (see below). *Rumination* is principally seen in congenital feeble-mindedness of a low grade or in demented cases.

Anorexia amounting to refusal of all food may be observed in mental depression, stupor and hysteria. It is often of long duration and leads to extreme emaciation. *Bulimia* (voracious appetite of a paroxysmal type) and *polyphagia* in which the feeling of satiation is often lacking, occurs among the insane (in maniacal and demented states), now and then in hysteria and neurasthenia. Sometimes associated with these or occurring by itself one may observe *polydipsia* (*cave diabetes mellitus*).

Meteorism is seen in demented and hysterical patients, often originating from swallowing of air and sometimes causing local intestinal spasm and "phantom tumors" which must not make one overlook the possibility of a *distended urinary bladder* in demented, stuporous cases or during coma.

Painful rectal crises with constant bearing down pains (evacuating finally only mucus and blood) are met with in tabes. Neurasthenics often complain of a prolonged sensation of fullness in the rectum with craving for bowel evacuations (*but without results*).

The genitourinary system: Polyuria or diabetes insipidus may follow head trauma and occur in brain tumors especially at the base, in hysteria and in Basedow's disease. *Glycosuria* is now and then observed in organic brain diseases, apoplexy, epileptic convulsions and concussions of the brain. *Albuminuria* may be found in meningitis, following epileptic convulsions (p. 118) and in apoplectic attacks. A careful examination of the urine should be made in all these cases (chemical, microscopic, etc.) to exclude the possible presence of nephritis or diabetes mellitus.

Priapism (prolonged erection of the penis with or without libido) may be met with in certain diseases of the spinal cord and in certain mental diseases when it is often combined with masturbation. *Penis* and *clitoris crises* (paroxysmal pains with libido and secretion) occur in tabes dorsalis.

Impotentia coeundi (erectionis, ejaculationis) with or without loss of libido, now and then with emissions and spermatorrhea may occur in mental disease (alcoholic, presenile, parietic, etc.), in chronic morphinism, neurasthenia, tabes, disease of the spinal genital centers and the like.

The *body temperature* should always be taken. We find *subnormal* temperature for instance in severe or comatose alcoholic intoxications and in marked stuporous paretics, etc. An *increase* in temperature is seen in infectious psychoses, meningitis, abscess of the brain (in the latter intermissions are frequent), in tetanus, epileptic and paretic convulsions (especially in "status epilepticus"), and in certain apoplexies, particularly during the preagonal stage, etc.

3. The Organs of Sense

(A) The *examination of the eyes*. The *external eye muscles* with the exception of the external rectus (N. abducens) and the superior oblique (N. trochlearis) are innervated by N. oculomotorius. The nuclei of the oculomotorius nerve are situated below the corpora quadrigemina on each side of the aqueduct of Sylvius. Somewhat more posteriorly is the nucleus of the trochlear nerve, while the nucleus of the abducens lies in the medulla oblongata below the fourth ventricle close to the nucleus of the facialis. The innervation is decussated and in part bilateral, the nuclei being in intimate and mutual connection through fibers passing along fasciculus longitudinalis posterior. In the cortex there are several centers for associated ocular movements in a frontal, parietal and occipital direction. Concerning connections with the cerebellum, or the vestibular apparatus see p. 162.

The *internal eye muscles* (sphincter and dilator pupillæ) are innervated through ganglion ciliare—the dilator from the sympathetic, the sphincter from the inner branch of the oculomotorius. When the *pupil reacts to light* (p. 97) the nerve impulse takes the following path

(see Fig. 3): (1) from the retina through centripetal fibers in the optic nerve with partial decussation in the chiasm through the tractus to "the primary visual center" (the anterior corpora quadrigemina, thalamus); (2) by connecting fibers from here to the nuclei of the oculomotorius; (3) by centrifugal fibers in the oculo-

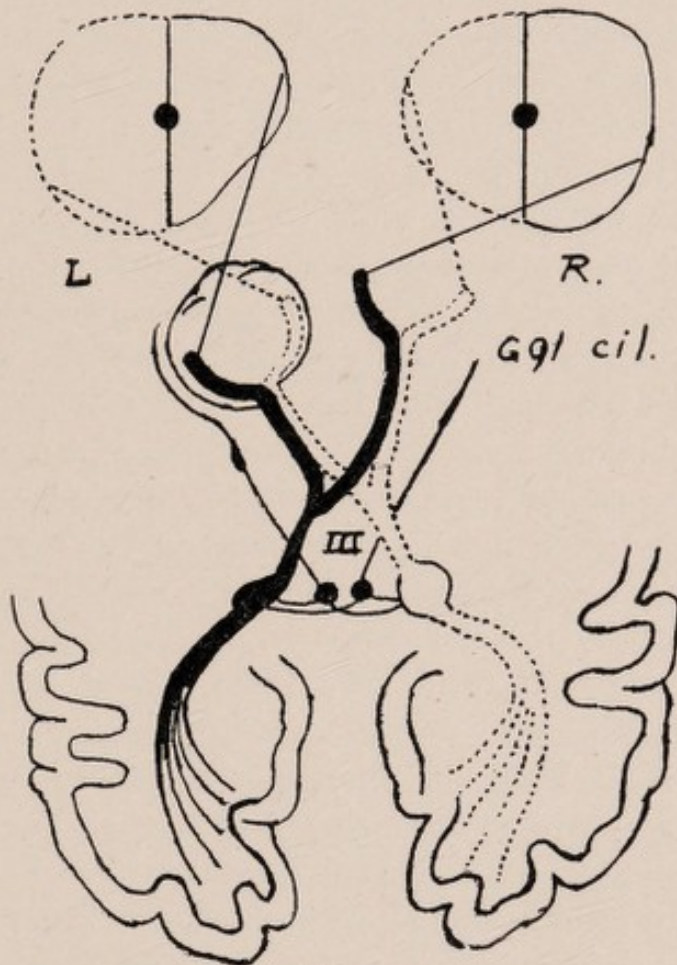


Fig. 3.

motor nerve to the sphincter muscle of the iris. The dilator muscle has a centrum ciliospinale in the 4th to 7th segment of the cervical part of the cord, the nerve fibers leaving the cord with the first to second anterior nerve root.

The fibers of the optic nerve decussate in part in the

chiasm (Fig. 3) so that each tract contains (a) fibers from the *temporal* half of the eye of the *same side* and (b) fibers from the *nasal* half of the eye of the *opposite side*. These fibers terminate in the primary visual center (see above): The secondary neurone (the optic rays, bundle of Gratiolet) extends from thalamus posteriorly through the occipital lobe, terminating around cells in the cortex, especially about fissura calcarina. As the lens projects the object as an *inverted* picture on the retina, the decussation of the fibers in the chiasm causes the visual impression from the *right* half of the visual field to be conducted through the *left* optic tract and vice versa.

The eye examination leads directly to the observation of possible *ptosis* (p. 99), of a possible *exophthalmus*—a regular protrusion of the eyeball so that one sees more of the sclera than ordinarily—(observed in intraorbital tumors; it is bilateral in Basedow's disease), of a possible *blepharospasm*—a tonic spasm of the orbicularis (N. facialis) seen in painful diseases of the eyes and in hysteria (not to be confounded with negativistic pinching together of the eyelids).

The Corneal Reflex.—When the cornea is carefully touched (with a hair or the head of a pin) a normal retraction of the bulbus results together with powerful reaction of the orbicularis (the reflex taking place through N. trigeminus). The reflex is diminished or absent during coma and narcosis, in diseases of ganglion Gasseri or of the trigeminus in the posterior cerebral fossa (tumor of the acoustic). It is less frequently so in hydrocephalus, hysteria, etc.

The pupils have an average diameter of 3-5 mm. A prolonged contraction (*myosis*) is seen, for instance, in iritis, in morphinism and the opium habit, following application of eserin, in luetic diseases (tabes, paresis, etc.),

in meningitis, senility and diseases of centrum cilio-spinale (dilator paralysis). Abnormally large pupils (*mydriasis*) are present in states of fear, during pain, convulsions (epileptic), in coma and in oculomotorius paralysis (p. 99). Inequality of the pupils (*anisocoria*) exceeding 1-2 mm. may now and then be seen in health. It is transient in migraine, neurasthenia, and catatonia, and may be permanently present in paresis, tabes, etc. *Angular* or distorted pupils are observed in local diseases of the eye (synechiæ, glaucoma) and in paresis, tabes, lues cerebri, etc.

Pupillary Reactions.—

(a) *Reaction to light:* When light is thrown into the eye the pupils contract (1) in the illuminated eye (*direct reaction to light*) and (2) in the eye not illuminated (*consensual reaction*).

(b) *Reaction to accommodation:* Strong convergent movement of the eyeballs (fixation of the finger immediately in front of the patient's nose) results normally in marked *pupillary contraction* in both eyes (joint motion, not reflex).

Examination of the reaction to light may be done in bright daylight. The patient is placed facing the light and made to accommodate for "distance." One eye is tested at a time, the other being kept covered and observed for consensual reaction, while the illuminated eye during the examination is alternately covered and uncovered. One should carefully exclude all movements of accommodation. When there is the least doubt as to results the examination should be made in a dark room with reflected light and the use of a head mirror. *This latter mode of examination should, in reality, be the only method in use, as it is the only one to be depended upon, and because the reaction of the pupils to light is of so*

decided a diagnostic importance in mental and nervous diseases.

*Absent reaction to light may be due to: (1) Disease of the centripetal pupillary fibers in the optic nerve. In such cases where there is a monolateral lesion there is (a) an absence of direct reaction to light in the sick eye (on the affected side); (b) loss of consensual reaction in the well eye (on the opposite side); (c) reaction to the convergence in both eyes, " Amaurotic pupillary rigidity" (until after a lapse of time). (2) Disease of the centrifugal pupillary fibers ("ophthalmoplegia interna," due to lesion of the oculomotorius and its nuclei, and is seen in basal meningitis, tumors, etc.). In this instance there is (a) an absence of direct and consensual reaction to light as well as reaction to accommodation in the sick eye ("absolute pupillary rigidity"); (b) in the well eye all the reactions are intact. Mydriasis is usually present in the sick eye. (3) Disease of the nerve tracts connecting the centripetal and centrifugal parts of the reflex arc (p. 94, anatomic relations not well understood) causes "reflex pupillary rigidity" (the Argyll Robertson pupil): *The pupil of the affected eye is not contracted by either direct or consensual illumination, but contraction results upon accommodation and convergence.* With regard to the first form of lesion resulting in "amaurotic pupillary rigidity" the determination should depend upon the condition of the consensual reflex, the nature of the visual impairment and the results of the ophthalmoscopy.*

Reflex pupillary rigidity is always an evidence of organic nervous disease, most frequently tabes or paresis. It is very seldom seen in chronic alcoholism, syphilis and following head trauma (unilateral).

In lues cerebri we usually see ophthalmoplegia interna;

also in morphine-, atropine-, hyoscine-intoxications, in arteriosclerosis, during epileptic (and hysterical) convulsions, in coma (?) and in catatonic stupor.

As an *important premonitory* symptom of pupillary rigidity one often sees extending over a considerable time a *pupillary sluggishness* (unilateral or bilateral) when the eye is subjected to light.

Eye muscle (ocular) paralyses: When *all* eye muscles are paralyzed, there is a divergence outwards of the eyeball; it is immovable and there is a drooping of the upper eyelid. Whenever the *external* branch of *nervus oculomotorius* is paralyzed we find: drooping of the upper eyelid (*ptosis*) with overactivity of the corresponding musculus frontalis (pronounced wrinkles of the forehead), a slight extension backwards of the head, a deviation of bulbus outwards and downwards or doing away with the turning of the eye inwards, upwards, and downwards. Whenever this is complicated with disease of the internal branch we find "ophthalmoplegia interna" (paralysis of the sphincter and absolute pupillary rigidity) with mydriasis. *Paralysis of the abducens* results in a doing away with the outward movement of the eyeball, while paralysis of *nervus trochlearis* results in defective movement downward-inward and defective rotary movement of the eyeball when looking downward-outward.

The symptoms of these paralyses are besides *strabismus* (convergens, divergens) as a rule *diplopia* (seeing double pictures in different directions according to the muscles affected), which disappears whenever the eye on the paralyzed side is covered. The diplopia may be combined with complaints of dizziness, etc.

In cases of *psychogenic* (hysterical) ptosis there is no overactivity of musculus frontalis. In hysteria one may

also come across *monocular diplopia* (polyopia). The patient sees the physician's finger double with one eye, etc. (usually best at a distance of 10-15 cm.).

In *ocular paralysis* (diseases of the pons—the "ocular center" is located near the nucleus of the abducens) the associated movement of *both* eyes (outwards, inwards, etc.) is done away with entirely or becomes defective.

In Basedow's disease we find besides exophthalmus (see above) at the same time often (1) *Graefe's* symptom: When looking from above downwards the upper eyelid does not fully accompany the movement of the eyeball or plane of vision but falls more slowly or even jerks back—the supracorneal sclera continues therefore to be visible. (2) The symptom of *Moebius*: Defective or arrested convergent movement (i. e., one eye turns out) when the patient fixes his eyes on an object on the level of his eyes and near them, while at the same time the individual recti muscles act normally when each is innervated by itself. (3) *Stellwag's* symptom: Staring with diminished frequency of reflex or involuntary winking movements of the eyelids, which may be entirely missing.

Conjugate deviation of the eyes: Both eyeballs are continuously directed toward one side, being at the same time usually directed a little upward, and can not voluntarily be directed toward the opposite side (observed in organic brain disease,—apoplectic-coma). There is at the same time often an *imperative turning* of the head in the same direction.

Nystagmus is a regular oscillation of the eyeballs when at rest (spontaneously) or not until intentional movements are made, i. e., when the eyes are turned laterally. The movements are pendulum-like (the swinging back

and forth being of equal length) or jerking: A quick excursion in one direction (right-sided or left-sided nystagmus) with a retarded return swing.

Nystagmus may be congenital (sometimes unilateral). It is an important symptom in disseminated sclerosis, in *Friedreich's ataxia*, luetic brain disease, disease of the cerebellum, or vestibular apparatus (see p. 164). It can not be psychogenic.

The *visual power* is tested by means of Snellen's test table, by counting fingers, etc. The *color-sense* is tested by means of color tables, worsted or tufts of wool of different tints, etc. The presence of any possible anomalies of refraction must first be corrected.

An enfeeblement of vision (*amblyopia*) and blindness (*amaurosis*) are met with in the various organic diseases of the visual apparatus or optic tracts. *Psychogenic amaurosis* (possibly bilateral, often following convulsive attacks) seldom eliminates altogether the patient's power of orientation in space; the pupillary reaction to light is intact (or sluggish?). When this type of amaurosis is unilateral, the "blind" eye perceives in the stereoscope or when aided with a prism.

Defects of color vision (*acromatopsia*) are seen in organic as well as in functional diseases. In the latter forms, mixed colors resulting from the primary colors, which the patient claims are not seen by him, are often perceived. Achromatopsia must not be confounded with *deficient color apperception*, i.e., defective designation (especially in imbeciles). Concerning macropsia, etc., see p. 55.

The field of vision is tested by means of the perimeter or more superficially by the hand test: The examiner places himself and the patient upon two chairs opposite each other at a distance of about a quarter of a meter,

the heads being on the same level. Each eye is to be examined by itself. One eye being covered, the patient is to fix his other eye upon the forehead of the examiner (and the latter is to fix his eyes upon the forehead of the patient). The hand or finger of the physician is then moved from without and midway between the two eyes into the visual field of the patient, the patient being required to state when he can faintly see the hand. If these statements (from the temporal, the nasal side, from above and below) in the main correspond with the physician's perception of the movements of the hand or finger, the patient's field of vision is considered normal.

The anomalies are: (1) *concentric contraction of the visual field* in (a) organic diseases, choked disk, optic atrophy, disseminated sclerosis, in (b) psychogenic states, in which it often varies in degree in the two eyes. It is as a rule greater on the side where there are other stigmata. A peculiar form is the *tubular type of visual field*; i. e., that the visual field remains the same for near and distant objects, while it should normally increase with the increase in the distance of the test object from the eye. It is observed in neurasthenia and hysteria. Sometimes there is a *gradual* contraction of the visual field in the last-mentioned diseases as the examination progresses due to fatigue.

Hemianopsia (respectively hemiamblyopia); blindness or impairment of one-half of the field of vision, either the temporal, nasal, upper or lower portion. A central zone of both optic centers representing the region of the macula is usually intact except in disease of the optic tracts. Hemianopsia is usually homonymous, i. e., the two right halves or the two left halves of the visual fields are blind or lost in both eyes (*hemianopsia dextra, sinistra*).

Hemianopsia may be due (a) to lesions in the optic tract in which case we now and then find *hemianoptic pupillary rigidity*, i. e., contraction of the pupils only upon illumination of intact portions of the retina. Sometimes the sparing of the macular region is absent; (b) to lesions in the bundle of Gratiolet (often in the neighborhood of the optic thalamus or in capsula interna, for which reason it is often associated with hemiplegic disturbances of the corresponding side of the body); (c) to lesions in the occipital visual center (*emollitiones cerebri*, etc.), being now and then accompanied by agnostic disturbances (p. 112). The patient is sometimes not aware of his own blindness. When two hemianopsias become combined, one after another, a total blindness may result.

In cases of hemianopsia the patient either does not complain at all or he says that he has lost his vision in *one eye*, which is the eye in which the greater half of the visual field has been lost. Cases of hemianopsia are examined by means of the perimeter, superficially by the hand test; in confused or demented patients by making threatening motions in the direction of the eyes from different sides or by observing the patient's behavior towards persons, things, etc., in the two visual fields, etc.

Heteronymous hemianopsia is usually temporal (called sometimes "blinders" hemianopsia). It occurs in lesions of the chiasm, luetic meningitis, tumor of the hypophysis. Unilateral or bilateral *sector-formed* defects may also be seen in diseases of the optic nerve and occipital lobes. *Isle-like* defects of peripheral or more frequently of central location in the visual fields (*scotomata*), which sometimes involve color vision only (red, green) are seen in retinitis and neuritis retrobulbaris (alcohol, nicotine, disseminated sclerosis). *Scintillating scotomata* are luminous points in the field of vision which occur in paroxys-

mal attacks and spread to form surfaces which often have "fortification-like" outlines and leave behind temporary isle-like visual defects sometimes amounting to hemianopsia. They are usually unilateral, occurring in epilepsy, hysteria, neurasthenia and migraine.

Ophthalmoscopic examinations should be made (1) in all visual disturbances, (2) where there are prolonged general or focal brain symptoms, (3) in all cases of loss of consciousness, (4) where there is a continuous complaint of headache or vertigo (see later).

With regard to the different ophthalmoscopic pictures of diagnostic importance to the neurologist (neuritis optica, choked disc, optic atrophy, retinal hemorrhages, retinitis albuminurica, etc.), reference must be made to the ophthalmologic text books. Optic *neuritis* is especially found in abscess of the brain, meningitis (tuberculous and luetic) and disseminated sclerosis; however, the neuritis is more apt to be of the "retrobulbar" type as stated above. The cause of *choked disc* is quite predominantly tumor of the brain, possibly hydrocephalus, lues cerebri, etc. *Primary optic nerve atrophy* is seen for instance in tabes, lues cerebri, rarely in arteriosclerosis. As a pallor of the temporal side of the disc it is observed in disseminated sclerosis, and as a *secondary atrophy* it occurs following choked disc. Retinal hemorrhages and retinitis albuminurica may point to the existence of an arteriosclerosis or disease of the kidneys.

(B) The examination of the ear may be divided into two parts: The examination of the *auditory function* (cochlear branch of the acoustic nerve) and that of the *static function* (vestibular branch; see p. 164).

A detailed examination will not as a rule be undertaken unless it be apparent that (1) the patient hears poorly or the clinical history gives information to this

effect, (2) that we are dealing with prolonged attacks of vertigo, disturbances of gait, nystagmus or symptoms pointing to intracranial disease, (3) in patients presenting aphasic symptoms (p. 108) or (4) in cases where there are unilateral auditory hallucinations.

The *function of hearing* is tested for ordinary voice and whispering (especially of figures; such as 35, 78, etc.). *Weber's test*: A sounding tuning fork applied to the vertex of the skull should normally be heard equally well in both ears. If in a case of unilateral deafness it is heard best on the *deaf* side, one is dealing with a disease of the *sound-conducting* peripheral auditory apparatus. If it is heard best on the *well* side, the lesion is located in the *central sound-perceiving apparatus*.

Rinné's test: A sounding tuning fork held against the mastoid process until it is no longer heard and then held in front of the opening of the outer ear, should *normally* be heard for still some seconds (to be read off on a stop watch). The result should be the same in lesions of the *sound-perceiving* part of the ear ("positive Rinné").

In lesions of the *sound-conducting* part of the ear, the air conduction is heard a shorter time than the bone-conduction ("negative Rinné").

(C) The examination of the *function of smell and taste* will as a rule only be of interest in cases where one suspects disease of the base of the brain (tumor, lues, fracture of the base of the skull).

Taste: The anterior two-thirds of the mucosa of the tongue is innervated by the lingual branch of the trigeminus while the posterior one-third receives branches of the glosso-pharyngeal nerve, for which reason special tests of the two regions of the tongue and their halves must be made separately. For this purpose

differently tasting substances (sour, sweet, bitter, salt) are applied with a brush, the patient by nodding indicating whether it tastes sour, etc.

Smell (nervus olfactorius): Each nostril is tested by itself—the other being closed—with differently smelling substances (small vials are best for this purpose). Smell as well as taste are sometimes individually but little developed, frequently much weakened in veteran smokers, alcoholists, imbeciles, demented patients, etc.

4. Speech—Writing—Agnosia—Apraxia

(A) *Speech disturbances* may be due to, first, errors in the *pronunciation* (articulation), in the formation and in the combination of speech sounds: *dysarthria*, *anarthria*, which are met with in diseases of the speech muscle apparatus (palate, tongue, lips), or in the nerves and centers of this apparatus in the medulla oblongata. Secondly, the disturbances may be caused by the patient not being able to find words or he is not able to understand the spoken word: *aphasia*, which is observed in diseases of the cerebral speech centers.

1. *Dysarthria* occurs in various forms: (a) *Stammering*, often congenital, is due to spasms of the muscles of speech. The patient becomes stuck in the first syllable or syllables of the word, repeats this time and again, finally succeeding after making use of considerable force in getting the rest of the word or the whole word pronounced. Spasmodic, conjoint movements in the facial muscles (grimacing) are often observed at the same time. The disturbances will be found increased when the patient is under psychic influence, and they are less severe or found absent when he sings or whispers.

Hysterical stammering is very capricious, increasing

often until it amounts to muteness (see below). The words are cut to pieces in a most baroque manner and now and then letters and parts of words become added which are quite out of place. The psychic irritability of the patient is usually very great.

(b) *Scanning*: The individual syllables are separated by pauses: Dem-o-crat-ic sim-ple-i-ty. It is typical of disseminated sclerosis, and is often at the same time combined with a slow pronunciation (*bradylalia*).

Logoclonia is a mixture of scanning and perseveration: Can I get a va-va-va-cation? (seen in paresis and *Alzheimer's disease*).

(c) *Bulbar dysarthria* is most frequently observed in cases of disease of the nuclei of the medulla oblongata (bulbar paralysis, etc.). The speech is slow, troublesome, "veiled," shows indistinct pronunciation of letters as if the patient spoke with food or the like in his mouth. Special forms are (1) *labial effacement*, in cases where labial muscles are paretic: The consonants *b, p, f, v, m*, and the vowel "u" are especially affected. (2) *Effacement of the nasal character* of the consonants *g, k, j, r*, which occurs in paretic conditions of the soft palate (diphtheria). (3) Paretic state of the tongue affecting the same consonants just mentioned with the addition of *d, t, s, l, r*. The dysarthria is, however, as a rule mixed; in extreme cases it results in unintelligible muttering (*anarthria*).

(d) *Syllable stumbling* (literal ataxia) is, practically speaking, characteristic of general paresis and should perhaps rather be considered a paraphasia (p. 110). The association of the verbal sounds is disturbed: Doubling of words or syllables, omissions, transpositions of letters or parts of words, perseverations, contractions, hesitations, are frequently also combined with a moderate

bulbar veiling. *This symptom should be looked for in all demented and confusional states, especially in patients forty to fifty years of age.* One should make use of words that are difficult to pronounce: Constantinopolitan, third royal dragoon regiment, methodist episcopal, expropriation-commission, and the like. The patient will repeat for instance "expo-pro-pami-commission," etc.

Aphonia may be due to paralysis or spasm of the vocal cords (N. recurrens vagi), but may also be met with in hysteria, in which case it usually begins suddenly and disappears suddenly and occurs in connection with emotional excitement. It sometimes becomes absolute when the patient is only able to whisper, although she may now and then be able to sing. The laryngoscopic picture varies. *Mutism*, when the patient is neither able to talk nor whisper, is observed in psychoses and hysteria.

(B) *Aphasia* is a loss of the power to give expression to ideas in words or in writing (*agraphia*) or to apprehend the word heard or the written character or print (*alexia*) seen, so as to awaken the corresponding ideas. Aphasia is caused by a lesion in the left hemisphere, in the speech centers or the connecting nerve tracts (Fig. 4).

The motor speech center (Broca's convolution) is located at the base of the left third frontal convolution. When this is the seat of lesion the patient can not pronounce words (*motor aphasia*). *The sensory speech center* for "word pictures" is located in the left first temporal gyrus (*Wernicke's center*). When destruction of this has taken place the patient does not understand what is said to him (*sensory aphasia, word-deafness*).

The existence of a definite center for writing or reading has not with certainty been demonstrated. The writing or reading is supposed to be brought about

through connecting tracts between the speech centers above mentioned and the visual center in the occipital lobe or between the former and the motor centers for the right hand. From Broca's center the nerve impulses pass to the bulbar speech centers.

There are, furthermore, connecting nerve tracts between the speech centers and other parts of the cerebral cortex, where the various intellectual processes are supposed to take place. This has been diagrammatically represented in Fig. 5 as the center of ideation, "B."

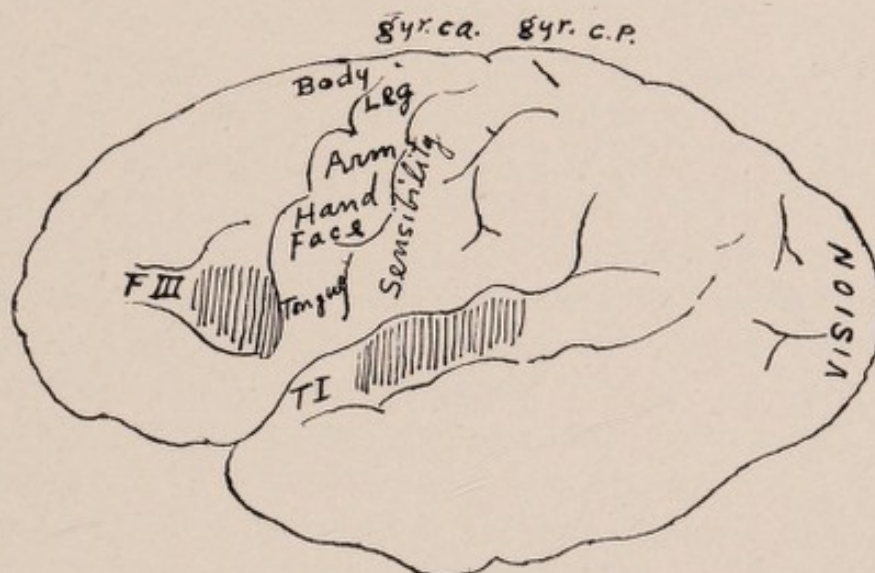


Fig. 4.

Lesions located in the different centers and association tracts occasion the following forms of aphasia:

1. *Cortical motor aphasia* (lesion of Broca's convolution: *b* in Fig. 5). This causes (a) *voluntary speech* to be done away with or makes it markedly defective (except short words, swear words, exclamations, especially during excitement). The patient *can not repeat* words heard, *can not read aloud* (alexia), *can not write*

(agraphia) voluntarily or when dictated to, but he can copy text placed before him. (b) Apprehension of spoken words is intact; understanding of written language is absent or defective.

2. *Sensory aphasia, word deafness* (lesion of Wernicke's center at *a*, Fig. 5). In this form of aphasia (a) the *understanding of spoken or written words is lost*; (b) the patient can *speak spontaneously*, but *verbal par-*

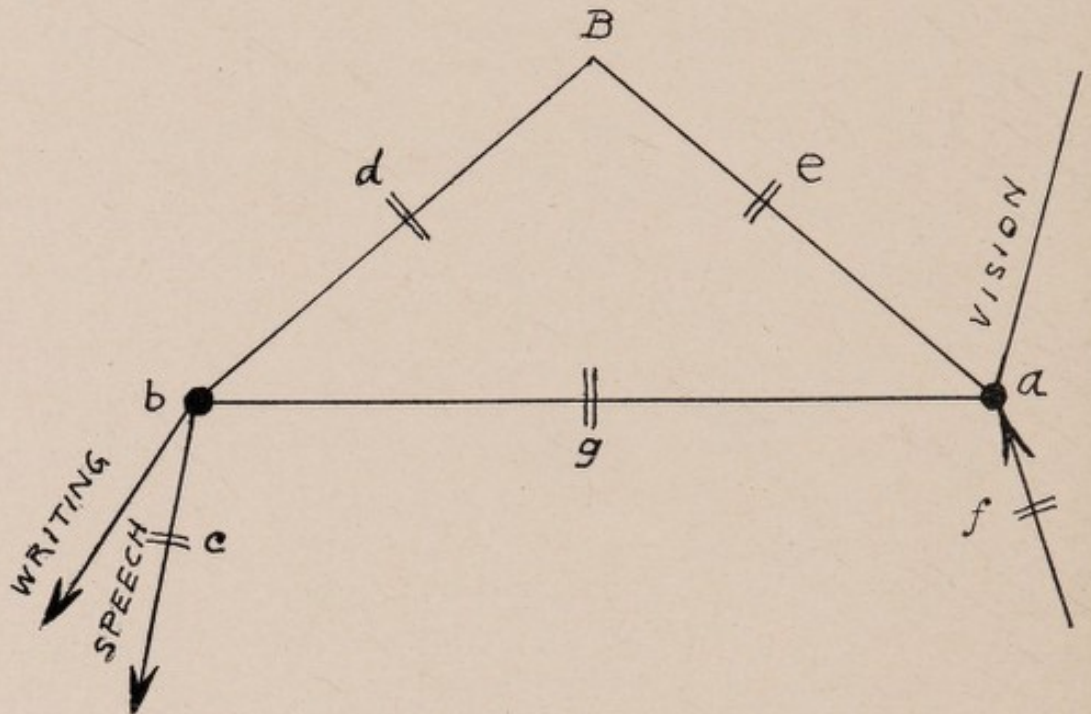


Fig. 5.

aphasia is usually present; i. e., he makes use of wrong, deformed or self-made words. *Perseveration* (p. 40) or reiteration is often observed; (c) he *can not, however, repeat* dictated words, he *can not read aloud, can not write spontaneously* or from dictation, but he is able to *copy*.

3. *Pure (subcortical) motor aphasia* (lesion at *c*) shows (a) *lost*: spontaneous speech, repetition of spoken words,

reading aloud; (b) *intact*: understanding of words and written characters, writing spontaneously, from dictation, and copying.

4. *Transcortical motor aphasia* (lesion at *d*) shows (a) *lost*: spontaneous speech and writing; (b) *intact*: all other functions.

5. *Pure* (subcortical) *sensory aphasia* (lesion at *f*) shows (a) *lost*: understanding of words, repetition of spoken words, writing from dictation; (b) *intact*: all other functions.

6. *Transcortical sensory aphasia* (lesion at *e*) shows (a) *lost*: understanding of words and written characters; *intact*: all other functions, but *paraphasia* and *paragrammia* are often present, and repetition of spoken words, reading aloud, writing from dictation and copying are performed *without being understood*.

Association-tract aphasia (lesion at *g*; the existence of this is doubted) shows *intact*: understanding of speech and written characters but with *paraphasia* and *paragrammia* (except when copying).

Pure forms are rare, combinations are usually experienced. *Total aphasia* (motor and sensory) is especially observed immediately after the beginning of an aphasia but it rarely continues to this extent.

In *amnesic aphasia* which is usually an accompanying symptom in the before-mentioned forms, the patient has a difficulty in "hitting upon" what he wants to say, to name, etc., but he recognizes the word at once when it is told to him and he can also repeat it. The disturbance affects especially nouns and plays a role in the paraphasic interchange of words.

In *optic aphasia*, which is not generally recognized, the patient can not name objects shown to him but can describe them, explain how they are used and name them

after handling and touching them all over and the like.

(C) *The agnosias* are related to the aphasias. We differentiate between the following:

1. *Tactile agnosia* or *astereognosis* ("handling" paralysis, Wernicke): This term is applied when the patient can not with eyes covered recognize or name objects (key, comb, penknife, pen, coins, etc.) put into his hand. Sometimes the patient can not even state the form, etc., of the object. The symptom may not only be explained by the slight sensory disturbances, motor paralysis, etc., which are often present, but it is usually an indication of disease in the association tracts of the cortex (posterior to gyrus centralis), and is now and then also present in lesions of the optic thalamus.

2. *Optic agnosia* (soul blindness). The patient can in this condition with difficulty or *not at all* recognize ordinary objects, the things surrounding him, etc. He can not name them, state what they are used for, etc. The things, therefore, often seem "strange" to him and he is to a certain extent disoriented as regards the outer world, etc. There is frequently also seen a certain degree of alexia combined with effaced or defective optic memories. It is met with in lesions of *both* occipital lobes.

(D) *Apraxia* is loss of ability to perform purposeful (complicated) movements or acts in the absence of paralysis or ataxia, and in spite of the patient's understanding of what he wishes to do, of what he is asked to do, etc. Apraxia is most easily demonstrated in the hands (as regards tests, see p. 115).

Defective reactions show themselves in part by *individual* movements (for instance, greetings, threatening gestures, etc.) becoming inadequate, awkward and baroque: The patient lifts the hand to make a greeting, but instead of doing so makes quite meaningless move-

ments with it in the air. He picks up a key, taking hold of it in the right manner, but can not manage to insert it into the keyhole, etc. More *complicated* movements are in part and especially interfered with: The patient takes hold of a lead pencil in the right manner, but may then suddenly dip it into the inkstand, as if it were a pen, and the like.

Apractic disturbances are always due to lesions in the supracapsular nerve tracts (p. 123), usually diseases in the left hemisphere, posterior to gyrus centralis or in corpus callosum. The left hemisphere does not only regulate the purposeful movements ("eupraxia") of the right hand but also those of the *left*. We therefore frequently find the combination of a paralysis of the right hand with apraxia ("dyspraxia") of the left. Apractic disturbances are also observed in more diffuse diseases of the brain (arteriosclerotic forms and Alzheimer's disease).

Investigation into a possible presence of aphasia (agnosia, apraxia) should be made (1) when the clinical history shows the presence of speech disturbances, etc., (2) when the patient's speech vocabulary seems perceptibly reduced, and when paraphasia or defective understanding is present, (3) in all patients who speak or act in a markedly "confused" manner, especially *elderly* patients, (4) in right-sided hemiplegic or monoplegic paralyzes.

1. *Speech Ability*.—(a) The patient is made to describe his symptoms, tell the story of his past life, and the like. (b) He is asked to name objects shown him. *Defective reaction* may be (1) that he does not "understand" the object (optic agnosia); (2) that he simply *can not name it* (optic aphasia). He should, therefore, be asked what the object is used for, suggesting a number of incorrect names and finally the right one, and

the like. (3) He is made to *repeat* words, series of words (days of the week, months of the year, etc.), series of figures, sentences.

2. *Speech Understanding*.—The patient is asked a number of questions, and remarks more or less “personal” are directed to him; he is called upon to do a number of things, some of them simple (shutting of the eyes, shaking hands, putting out the tongue) and some more complicated (take all the money out of that purse and give me all the twenty-five-cent pieces in it, and the like). Defective reactions may in this connection be due to apraxia (see below).

3. *Ability to Read*.—The patient is made to read aloud from a newspaper or the like, taking notice whether he reads correctly or inserts wrong words (*paralexia*, which is frequently observed in paresis), or whether he is not able to read at all. The question as to whether he has an *understanding* of what he has read is then to be taken up, by asking him to repeat the contents of what he has read, which may possibly reveal amnesic disturbances, and by giving him certain problems in writing (as previously stated). Alectic patients are as a rule still able to read their own names and very common words. The alexia is sometimes elective, confined to certain letters, for instance, or it is only perceptible when long words are used, or the patient is able to read the individual letters, but can not put them into words.

4. *Ability to Write*.—The following tests are used: (a) Writing from *dictation*. Errors here may be due to agraphia, paragraphia (erroneously written, transposed letters and words, etc., often seen in paresis or in impairment of memory, the patient forgetting what has been

dictated). (b) *Spontaneous writing* (letters, narratives, series of figures), which may also give information concerning defective memory of words. (c) *Copying* of written or printed manuscripts. If the patient copies correctly one should test whether he can "transpose," from print to ordinary handwriting and vice versa.

Dysgraphia corresponds to dysarthria; i.e., it is due to disturbances of the writing movements themselves as seen in, (1) tremor (senility, paresis), (2) ataxia or intentional tremor, observed in tabes and disseminated sclerosis, (3) *writers' cramp* (mogigraphia): Spasmodic muscular contractions in the arm or hand whenever an attempt is made to write, observed in neurasthenia, hysteria. In general paresis the handwriting shows coarse tremor, paragraphia, omission of letters and words, and additions out of place.

5. *Astereognosis* (see p. 112); optic agnosia (mentioned under Speech Activity).

6. *Apraxia*.—Each hand should be tested by itself as to (a) *elementary movements*—snapping of the fingers, pill-rolling movements, rolling paper into cornucopias, etc.; (b) *expressive gestures*—greeting, beckoning, threatening with clenched fist, making long nose; (c) *purposeful movements*—use of comb, brush, match box, sponge, soap, bowl, cigars and match box. The patient is asked to remove all the coins from a purse, arrange them on the table according to value, give to the physician all the half-dollar pieces with the left hand, put the remainder back into the purse, put the purse into his own pocket, etc. (d) *Imitation of movements* made by the physician (scratching one's head, rubbing hands, etc.). The apraxia often distinctly reveals itself in the patient's daily behavior (at the toilet, when eating, sewing, knitting, etc.).

5. Motor Disturbances

A. ABNORMAL MUSCULAR MOVEMENTS

1. *Spasms* are involuntarily developed muscular contractions, and are either (a) *clonic*—rhythmically alternating contractions and relaxations—or (b) *tonic*—continuous muscular contractions of shorter or longer duration.

The spasms may be confined to a single nerve-muscle region, they may extend over an entire extremity, over one-half of the body or they may become *general* (convulsions).

(a) Spasms in a single nerve-muscle territory are the following: *Blepharospasm* (p. 96); *masseteric spasm* (in tonic form occurs in trismus, tetanus, epileptic seizures, meningitis, etc.; in clonic form it is seen in the chattering and grinding of teeth observed in meningitis, demented states, etc.); *facial spasm* (tic convulsif) may be clonic, as a jerky pinching together of the eyelids, frowning of the forehead, drawing up of the angle of the mouth, etc. It is usually unilateral, idiopathic, and may be the only evidence of cortical irritation, or may occur as a partial symptom in general convulsions; tonic facial spasm is seen as contracture following attacks of facial paralysis (p. 128) and in hysteria (p. 137); *clonic spasm of the neck muscles* is sometimes circumscribed, but is usually found to extend over several muscles even to the shoulders, (*maladie des ties*), so that the motor effect results in a steady unrest of the head, face and shoulders. It is seen as a rule in the form of a stereotypic mannerism or defense-movement ("memory-spasms"), and usually results from psychic influence or a local annoying ailment, etc. Its course is in part dependent upon psychic circumstances being often combined with imperative ideas, echolalia, coprolalia, etc.,

or may occur during intermittent psychotic attacks—predominantly in nervous (hysterical) people.

(b) Spasms involving a single extremity are usually a sign of cortical disease (p. 118) but may sometimes be observed in hysteria, then usually in the form of a contracture (p. 139).

(c) *General spasms* usually show themselves as intermittent *convulsive attacks*:

1. *Epileptic (Epileptiform) Spasms*.—(a) An external cause is rarely demonstrable.

(b) *Aura* is frequent (lasting seconds, minutes): Slight local spasms, parasthesias; endotic sounds, play of colors, sparks before the eyes, outbreak of perspiration, etc.

(c) The *paroxysm* consists of the patient's *falling down suddenly*, becoming *completely unconscious* and frequently contracting some injury from the fall. (a) *The tonic state* lasts seconds up to a half a minute and comprises a general muscular tension with spasm of arms and legs in a state of extension, a distorted face, head bent backwards, eyes turned upwards and outwards, trismus; cessation of respiration (frequently ushered in with an *outcry*) and cyanosis of the face. (β) During the state of *clonic spasms* we find short, rhythmic jerks passing through the entire body of the patient, *he may bite his tongue*, may show bloody froth at the mouth, a noisy labored respiration, the color of the skin becoming more natural as this stage progresses and it may be combined with an outbreak of perspiration and frequently with an *involuntary* passing of urine and feces. Duration one half to a few minutes.

(d) *Sopor*ous (somnolent) *terminal* state: The patient lies in a completely relaxed condition, is almost without reaction, the respiration is often labored and snoring. *The reaction of the pupils to light, the corneal and skin*

reflexes (p. 141) are absent, sometimes also the tendon reflexes. *Bilateral Babinski reflex is frequent* (p. 141). Occasionally there is a slight rise in temperature. *Duration* as much as several hours. Upon awakening, the patient shows amnesia, muscular fatigue, sometimes slight ecchymoses about the forehead and conjunctiva and quite frequently postparoxysmal albuminuria. Sometimes there may be temporary paralysis in the extremities.

Of the first rank in *etiology* is a "spasmophile diathesis" in the genuine form of epilepsy in children (eclampsia infantilis). Other causes are chronic alcoholism in alcoholic eclampsia, epilepsy, uremia, eclampsia gravidarum, arteriosclerotic and paretic brain disease, tumor cerebri, etc.

Epileptic spasms occurring in series without free intervals, i. e., without return of consciousness, have been described as *status epilepticus*. This is often accompanied by rises in temperature and may end fatally.

2. *Jacksonian Spasms*.—These are usually unilateral or confined to half of the face, to an arm, etc. They are altogether clonic in character, without loss of consciousness, or this may not develop until later during the attack. They frequently start locally and show further extension corresponding to the locality of the cortical motor centers (Fig. 4) and sometimes finally become general. A postparoxysmal muscular paresis (monoplegic or hemiplegic) is often observed or the spasms may show themselves in the extremities that were previously paretic. The duration of the spasms varies according to the etiology, a cortical irritation usually playing the chief role. They occur in tumor, in arteriosclerotic or paretic disease of the cortex, luetic meningitis, and the like.

3. *Psychogenic or Hysterical Spasms*.—(a) These show themselves very frequently in association with emotional

disturbances, eventually as a more or less delayed effect, following a previous mental depression, etc.

(b) They are frequently ushered in by an anxiousness, a globus hystericus feeling, and the like.

(c) The *paroxysms* seldom begin suddenly and the fall is not abrupt. It is more frequently a collapse without injuries. The paroxysm should be interpreted as a *crisis in the affects*. The movements are extreme, absurdly exaggerated affect-movements, which often continue to be the same in each attack: gross, violent, twisted movements of the body (contortions), an excessive bending backwards of the body (*arc de cercle*), a wild combat with arms and legs so that it becomes difficult to hold the patient (contrary to what is observed in epilepsy), a convulsive laughter or weeping, passionate, plastic emotional movements and postures ("*attitudes passionelles*" of fear, anger, ecstasy, libidinousness, etc.), or a clown-like tumbling about with frequent shouting, screaming, singing, which finally may become a complete "muscular frenzy." The respiration is sometimes dyspneic, sometimes tachypneic, the color of the skin being, as a rule, normal. *Biting of the tongue and incontinence of urine are rarely observed.* The pupillary reaction to light is usually intact while the corneal and skin reflexes may be absent, but the tendon reflexes are never so. *The Babinski reflex is never present.* The state of consciousness varies: The patient may frequently be able "to hear but not to speak or see," although one sees more frequently mild defects of consciousness with delirious experiences (involving the initiating emotional excitement, memory-complex, visual or aural hallucinations), delirious speech-craving, in other words, a transition to the "delirium of reminiscence" (p. 53).

During the attack there is often a certain tendency to

being influenced by external conditions, such as remarks made, pressure upon spasmodic zones, etc. (p. 149). The attacks often occur *coram publico* (before an audience), hardly ever when the patient is alone.

(d) The *duration* of the attack varies greatly—lasting minutes, hours, half days. There is no postparoxysmal sopor, only fatigue, drowsiness, normal sleep. There is no postclamptic albuminuria, the memory of the attack varies, hysterical paresis sometimes follows attacks.

Psychogenic spasms without free intervals, no matter how frequently recurring, do not occasion a rise in temperature or a fatal termination.

Other abnormal muscular movements are:

4. *Choreiform Movements*.—These are usually continuous, involuntary, purposeless muscular movements or general muscular restlessness. They are often rapid and with “sudden outburst” reminding one of movements of embarrassment (grimacing, rolling of the eyes, tossing of the head, flinging or fumbling of the arms, kicking with the feet, twisting of the body, etc.). The motor restlessness is *increased* by intentional movements and when the patient thinks herself observed; it *disappears* as a rule during sleep. The movements are usually bilateral (in chorea minor and the Huntington chorea, the latter a hereditary disease leading to a progressively developing dementia in which the movements are less rapid and are diminished by intentional movements), less frequently unilateral (in hemichorea posthemiplegica, especially following the cerebral form of infantile paralysis).

5. *Athetoid movements* are usually observed only in fingers and toes and are slow extensions and flexions, adductions and abductions, varying in nature and tempo

in the different fingers and toes, which cause them to be placed in mutually odd positions. The motor restlessness usually continues during sleep, and is most frequently observed as a hemiathetosis hemiplegica.

Joint movements are spoken of in connection with hemiplegia.

6. *Tremor* shows itself as fine, involuntary, frequent, oscillatory movements with slight motor excursion of a part of the body, especially of the fingers. It is seen normally during emotional excitement (fear, nervous tension, violent anger, etc.), extreme fatigue or exposure to cold. Where the tremor is *pathologic*, we speak of (a) tremor when *at rest*, which may be (a) frequent (8 to 12 oscillations per second), and observed in chronic alcoholism, especially in delirium tremens, chronic lead and mercurial poisoning, morbus basedowii, neurasthenia, etc.; or (β) slow (5 to 6 oscillations per second), observed as tremor senilis which becomes increased when voluntary movements are undertaken, and in paralysis agitans in which intentional movements lead to a diminution of the tremor.

Psychogenic (hysterical) tremor may resemble several of these forms.

(b) Tremor only observed *when muscles are active*, which is known as intentional tremor. This is observed in disseminated sclerosis and in Friedreich's ataxia in connection with voluntary movements such as bringing the glass to the mouth, the index finger to the nose, while writing, etc. (p. 115), when the part of the body in action makes quite regular pendulum-like vibrations, which swing in both directions from the line of motion, the motor excursion increasing as the goal is reached.

Concerning "ataxia," see p. 160.

B. PARALYSES

A complete loss of the voluntary control of the muscular power is known as paralysis; when the loss is partial, it is termed paresis.

The corticospinal muscular tract (Fig. 6) for the psychomotor ("voluntary") impulses consists of two parts ("neurons"):

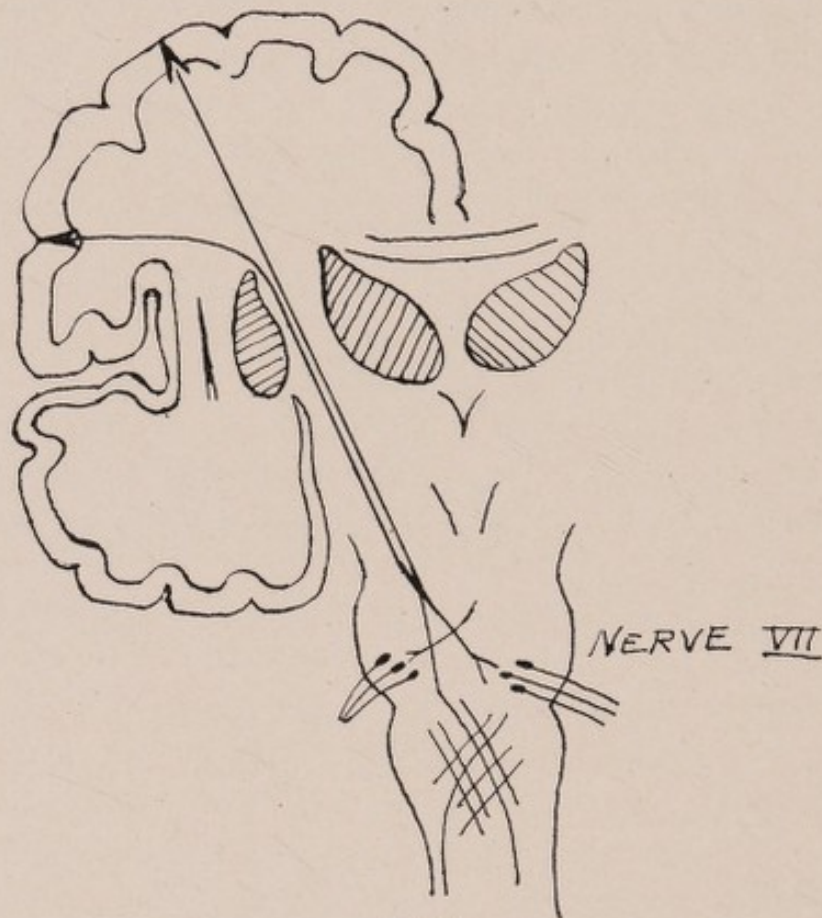


Fig. 6.

1. The pyramidal tract, the fibers of which take their origin from ganglion cells in the *psychomotor centers* of the anterior central gyrus (concerning the location of the different centers see Fig. 4). The pyramidal tracts pass from here convergently through the medullary

substance of the cerebrum to the internal capsule (between the thalamus and the lenticular body), and are in this locality situated within narrow limits and close to the sensory tracts, (p. 152), and the optic radiations, (p. 96). These tracts give off fibers as they pass the nuclei of the cranial nerves (see below) and continue their course through the base of *crus cerebri*, in the center of the pons to the anterior part of the medulla oblongata where the *pyramidal decussation* takes place. The greater part of the fibers cross in the median line in this locality and are continued downward through the lateral column of the *opposite* side of the spinal cord, as the *lateral pyramidal tracts*, which farther along in their course send fibers into the *anterior horn of the same side*, where they split up around the "giant ganglion cells" of the anterior horns.

From these emanate the fibers of (2) *the primary motor neuron* which, after passing through the *anterior spinal nerve roots* and the peripheral nerves, finally reach the muscles. A small portion of the pyramidal fibers pass *undecussated* through the anterior columns of the spinal cord as "direct pyramidal fibers" to the anterior horns of the same side (?).

Even before the decussation takes place in the medulla oblongata *crossed fibers* are given off to the group of nuclei of the cranial nerves (Fig. 6).

The innervation of the voluntary muscles is therefore quite predominantly crossed; i. e., the innervation takes its origin from the opposite cerebral hemisphere. Only certain muscle groups (eye muscles, facial muscles about the forehead and orbital region and the musculature of the trunk of the body) also receive an undecussated or bilateral innervation (see pontine-paralyses p. 133).

Innervation from the cerebral cortex is of special importance to the more complicated, *synergic muscular movements*, particularly of the hands and fingers. The "coarse" muscular power, the gross movements (principal movements, gait, etc.) are innervated to a large extent by lower lying infracortical, bulbar and spinal nerve centers.

All the muscles of the body are normally in a mild state of tension. This tonus is probably due to continuous innervation coming from the motor cells of the anterior horns, elicited by sensory impulses emanating from the skin, muscles, tendons, periosteum, and joint surfaces. Another important component influence is the cerebellar impulse (p. 163). The muscle tonus becomes *diminished* or may be entirely absent (*hypotony, atony*) during complete narcosis and other profound unconscious states, in the diseases of the cerebellum and in lesions of the sensory tracts. The tonus is *increased* (*hypertony, spasticity, rigidity*) where there is lesion of the pyramidal tracts. *Disturbances in tonus generally run parallel with disturbances of the tendon reflexes* (p. 144).

Muscular rigidity is seen as a predominating symptom (without real paresis) in *paralysis agitans* leading to the development of a peculiar outward habitus: A leaning forward of the trunk of the body with the head more or less bent upon the chest, a mask-like facial expression, the arms "glued" to the body, with slightly bent elbows, the fingers in writer's position and with a characteristic tremor like a "rolling of pills" (p. 121). All the movements are small and slow, the gait is tripping. If the patient is shoved he can not stop but runs forwards or stumbles backwards until he falls, (*propulsion-retropulsion*). In Thompson's *myotonia con-*

genita we find a tonic rigidity, (tetanic contraction) in the muscles involved in an intentional muscular movement: the patient can not let go the cup he wishes to put down, etc., which may last seconds or minutes. A similar tetanic contraction is observed as the result of faradic muscular stimulation of short duration (myotonic reaction).

The state of *nutrition of the muscles* is dependent upon influence coming from the giant cells of the anterior horns. When these or their connection with muscles through the anterior nerve roots and the peripheral nerves become diseased, we frequently find that *muscular atrophy* results.

This latter is, according to causation, either *localized, elective* (certain muscles), *systematized* (functionally related muscles, see p. 132) or *diffuse* (extending over an entire extremity, etc.). Diffuse muscular atrophy may, for instance, also be observed following joint diseases (arthrogenic atrophy), or long-continued immobilizations, (inactivity atrophy). Muscular atrophy is *detected* (a) by inspection (disappearance of the normal muscle outlines, detection of folds in the overlying skin, etc.); (b) by palpation (volume, consistency, tension). Where the atrophy is unilateral one should always make a careful *comparison with the muscles on the sound side* (measurements). (c) Muscular atrophy may furthermore be demonstrated by means of the electric reaction of the muscle.

ELECTRODIAGNOSIS

If a *galvanic* current of medium strength (a few ma.), is made to pass through a muscle by direct stimulation or through its nerve (indirect stimulation) the muscle contracts with *lightning-like* rapidity as the electric current

is being opened or closed. The contraction is most powerful when the negative pole (the cathode) is applied to the muscle; the other pole (the anode) being placed on the chest or back. With the poles thus applied it is more powerful upon *closing* of the cathode than by closing of the anode, the results being expressed by the formula: Ca C C > An C C. *Faradic stimulation* of the muscle or its nerve gives a tetanic contraction as long as the current is passing. Where there is an affection of the primary motor neuron (ganglion cells of the anterior horn, anterior nerve root, peripheral motor nerve fiber) there may be found (a) *quantitative changes* in the irritability, usually as a *diminution* of the strength of current necessary to bring about muscular contraction; (b) *qualitative changes* especially seen as a *degeneration reaction* (DeR): (1) *sluggish, worm-like contractions upon direct galvanic stimulation of the muscle*, (2) *a possible reversal of the reaction formula*, i. e., a change to An C C > Ca C C. The indirect galvanic and the direct and indirect faradic irritability is sometimes intact, sometimes diminished or absent.

The muscular atrophy may occasionally be hidden by deposits of fat in and about the muscles (lipomatous muscular atrophy, pseudohypertrophy), which sometimes may lead to much accentuated muscle outlines, although this is as a rule only partially so. Palpation will often reveal distinct relaxation, a "cotton-feel," etc. Concerning fibrillation see p. 132.

Examination as to the presence of *paralysis* may be made by observing the patient (his facial expression, spontaneous movements, postures), by letting him perform movements upon request, with or without resistance, (p. 130).

In every case of paralysis we should determine: *To-*

pography (muscle, muscle groups or limbs involved), *degree* (paralysis, paresis), *tonus*, *state of nutrition* of the muscles (electric reactions).

1. *Paralysis of Cranial Nerves*.—Concerning eye muscle paralysees see p. 99.

(a) *Facial paralysis*. Here it is important to differentiate between the peripheral (infranuclear, nuclear) and the central (supranuclear) type. (1) The *peripheral type* (the nucleus—ganglion geniculatum—and the peripheral nerve trunk). The place of lesion is often in the fallopian canal and may then be caused by an otitis—"rheumatic"—or it may be at the base of the brain from meningitis, tumor, and the like. *All three branches* may be affected, in which case we find relaxation of the particular side of the facial musculature, obliteration of the facial wrinkles, voluntary movements done away with or diminished, such as wrinkling of the forehead, complete closing of the eye, elevation of the mouth. The eye stays wide open, (*lagophthalmus*): when the patient attempts to close it the eyeball will frequently roll upwards under the upper eyelid (Bell's symptom). The corner of the mouth droops, the mouth is frequently drawn in a crooked way toward the sound side. When he speaks it often has the appearance of an "exclamation point," the point being formed by the paretic, closely approximated halves of the lips. The patient can not whistle, blow out a candle light when it is held in front of the paretic side. Liquid food and saliva will often ooze out on this side. Now and then there is difficulty in chewing, i.e., interference with the function of the buccinators. The speech is frequently labial (p. 107). The paresis is often made still more apparent by attempts at speaking or laughing.

The electric reaction of the muscles varies, degenera-

tion reaction is often present (p. 126). In cases that have not recovered we frequently find secondary *contracture* in the paralyzed muscles, which tends to some extent to hide the disfigurement, but the paralyzed side of the face remains equally immobile.

When lesion of the facial nerve occurs in that part of its course where it conveys fibers from the chorda tympani, we may find *defects of taste* on the anterior two-thirds of the tongue.

The nuclear form of facial paralysis (disease of the pons) may sometimes leave the facial innervation of the forehead intact and is often complicated by disease of other cranial nerves (abducens, compare p. 99), etc.

(2) *The central type of facial paralysis* (observed in cases of capsular hemiplegia, see p. 133, and cortical diseases: meningitis, emollitio cerebri, paresis, etc.) is usually seen as a paresis. The picture is in a general way like that of the peripheral type, *the facial innervation of the forehead is, however, intact* (bilateral innervation).

There is no degeneration reaction. Where it is of cortical origin we frequently find chronic spasms in the paretic half of the face.

One should take care not to confound facial paralysis with facial asymmetry or *congenital* difference in innervation of the two halves of the face (anamnesis, earlier photographs of the patient).

(b) *Paralysis of the masseters* is upon the whole a rare symptom even in undoubted cases of trigeminus disease. It is tested by palpation, by letting the patient close the mouth against resistance, by letting him bite into a stick of wood, etc.

(c) *Paralysis of the tongue* (nervus hypoglossus) may

be (1) *bilateral* (seen as a nuclear disease, especially in bulbar paralysis): The tongue then lies motionless within the mouth, is broadly flattened, and can not be protruded. There is dysarthria (p. 107) and difficulty in swallowing. Usually *atrophy* (relaxed state, wrinkles and folds especially shown along lateral edges with fibrillation), is present. (2) It may be *unilateral*: The tongue is protruded obliquely pointing toward the *paralyzed* side due to overaction of the sound genioglossus muscle.

(d) *Paralysis of the soft palate* (nervus trig.; glosso-pharyngeus?) may be (1) *bilateral*: the soft palate hangs down, remains motionless during phonation (making the "ah" sound). The speech is nasal, there is difficulty in swallowing (regurgitation of fluids through the nose). It is observed in postdiphtheritic paralysis, bulbar paralysis, etc. (2) It may be *unilateral* (frequently in hemiplegia): During phonation the palate is not equally arched or remains motionless on the sick side. An oblique position of the uvula is an uncertain sign.

When the uvula is touched the palate is normally raised with a shortening of the uvula, this constituting the *palate-reflex* which, however, is rather an inconstant sign. It is absent in the before-mentioned diseases, and also in hysteria and during the administration of bromides.

(e) *Dysphagia* is a paralysis particularly observed in bulbar diseases. The difficulty especially encountered is swallowing solid food, which often "goes down the wrong way," i.e., into the incompletely closed larynx (aspiration-pneumonia). Similar symptoms, which may or may not be due to real paralysis or sluggishness, may sometimes be observed in patients showing deep stupor.

(f) *Paralysis of the laryngeal muscles* (museles of

phonation, nervus vagus) is not of material significance in psychiatry. The examination requires laryngoscopic expertness (text books referred to).

2. *Paralysis of the Extremities.*—Paralyzed limbs are often seen lying limp and helpless with effaced muscle contours, sometimes in uncomfortable positions in which the patient seems inclined to let them remain. If they are passively lifted, they will fall heavily to the bed.

Paresis may be suspected if the patient of himself makes use of his arm or leg to an unusually slight extent, especially is this true when the right arm is referred to and he is not left-handed. He may be more carefully tested (a) by directing him to make voluntary movements *without offering resistance* (noting the extent, rapidity and regularity of the same), and (b) when *resistance is made* to the movement. The hand grip is tested by means of a *dynamometer*, the muscular power of one side being compared with that of the other, provided, of course, the paralysis is not bilateral.

Paralysis confined to an arm and leg may be (a) *peripheral* (lesion of one or more peripheral nerves, caused by traumatism, neuritis, etc.). The degree of the paralysis varies, and its extent corresponds to the anatomic region innervated by the affected nerves. The paralysis is *flaccid* (atonic), often *atrophic*, according to the seat of lesion peripherally to the cells in the anterior horn. The tendon reflexes (p. 142) are diminished or absent. Sometimes there may be sensory disturbances, and the *degeneration reaction* is often found to be present in the muscles affected.

Among peripheral forms of motor paralysis which may be met with in the insane may be mentioned (1) radial paralysis, for instance in alcoholists who may have been sleeping off a debauch with the head resting

upon the arm, or may have suffered the grip of a policeman when arrested, etc.; (2) neuritic paralysis of the lower extremities in alcoholists (polyneuritis alcoholica), which is usually bilateral, occurring as a paraparesis (p. 132), with tenderness of muscles and nerves (in the region of the Achilles tendon, the head of the fibula, in the popliteal space and about the anterior crural nerve below Poupart's ligament). We find a flaccid condition of the muscles, frequently slight atrophy (in the peroneal region and in that of the vastus muscles), an absence of tendon reflexes (Areflexia, p. 143), more or less extensive sensory disturbances, sometimes ataxia, frequently characteristic disturbances in gait: "high-stepping," i. e., the anterior part of the foot showing a tendency to hang and slightly turn inward, being incapable of pronation, due to paresis of the peroneus. The tip of the foot is apt to catch while walking, which is the reason the patient forcibly lifts the leg by bending the knee and hip joint ("strutting").

(b) *Spinal paralysis.* Through its peripheral nerve, every muscle receives fibers from *several segments of the spinal cord* lying one above the other, the innervation being plurisegmental (from one main cell and several accessory cells; i. e., anterior horn cells). The spinal centers therefore represent to a certain degree complicated muscle actions ("synergies"). Lesion of the anterior horns will, in accordance with this, result in *combined paresis* (rarely paralysis) of muscles having different peripheral innervation. Lesion of the 5th and 6th cervical segment for instance causes paresis of the deltoid, biceps, brachialis internus and supinator longus. Inasmuch as the intermingling of the nerve fibers does not take place until the plexus is reached, lesion in the *an-*

terior roots may in part give rise to a similar "segmental paralysis."

Text books in neurology are referred to for sketches illustrating the segmental innervation.

The principal types of spinal paralysis are:

(*a*) *The anterior horn type*. The immediate result (for instance in poliomyelitis) is paralysis, *later on paresis*, which is limited to *individual synergically related muscle-groups*, corresponding to the cell-segments which have undergone definite destruction. The paralysis is *atonic* and *atrophic*, frequently showing partial or total degeneration reaction (DeR). Fibrillation is frequently seen in the atrophic muscles and consists of fine fibrillary, quick contractions without motor results. *Areflexia* of all or of certain reflexes, according to the seat of lesion, is as a rule met with. No sensory disturbances.

(*β*) *The lateral column type* (with lesion of the lateral pyramidal tracts): *Usually paresis*, which, however, is evenly distributed over *all* of the muscles located below the seat of the lesion. *Hypertony*, increased tendon reflexes (p. 144), is frequently combined with clonus. Babinski reflex (p. 141) is as a rule present, but no muscular atrophy or DeR. *Etiology*: Independent systemic disease ("spastic spinal paralysis"), meningitic processes, tumor, etc.

(*γ*) *The mixed type* (seen in systemic diseases, amyotrophic spinal paralysis, etc., and in transverse lesions of the spinal cord) shows varying pictures with symptoms from *a* and *β* and symptoms referable to the posterior columns (sensory disturbances, possibly ataxia), etc.

The paradigm of spinal paralysis is the *spinal paraplegia*, respectively *paraparesis* (diffuse diseases of the dorsal part of the cord, compression myelitis, etc.): bi-

lateral paralysis of the lower extremities with hypertony, hyperreflexia (increased reflexes) and bilateral Babinski reflex. Sensory disturbances are as a rule found in both lower extremities and in the abdomen as high up as the level of the seat of the lesion in the spinal cord, and there is a disturbance in the functions of the vesical and rectal sphincters (p. 146). The gait is either paretic-dragging or spastic; the patient does not quite succeed in getting the plantar surfaces off the ground and is not able to bend sufficiently the knee and hip joint.

Concerning spinal hemiplegia see p. 156.

(c) *Bulbar or pontine-paralyses* are predominantly characterized by evidence of lesion of certain cranial nerves: of the facial, hypoglossal and trigeminus (p. 158), together with paralysis of ocular muscles. The paralyses are of the nuclear type. There is also often a combination with lesion of the pyramidal tracts (the *opposite* arm and leg) resulting in an *alternating* paralysis: i.e., on one side central paralysis of the facial nerve, on the other side paralysis of the arm and leg (*Millard-Gubler's* paralysis; lesion in the posterior part of the pons *behind* the decussation of the nerve fibers of the facial, see Fig. 6). Concerning hemiplegia sensitiva alternans, see p. 158.

Lesion in crus cerebri may produce hemiplegia oculomotoria alternans (*Weber's paralysis*).

(d) *Cerebral paralyses* are either *hemiplegic* (arm, leg, and side of face of the same side), or they are *monoplegic*.

(a) *Cerebral hemiplegia* is usually *capsular* (the pyramidal fibers being most compact in the capsula interna (p. 122), and are therefore most easily affected *in toto* in this locality). It may be caused by hemorrhage, embolism, luetic processes, general paresis, tumor, etc.

Its mode of development is most frequently sudden as a *cerebral apoplexy*: the patient falls suddenly to the ground in an unconscious state, becoming afterwards more or less completely comatose. Even at this time there are often signs of hemiplegia: drooping of one corner of the mouth, ballooning out of one cheek during respiration, oozing of the saliva over one side of the face, the extremities seemingly more inanimate on one side than on the other, *unilateral* absence of the skin reflexes (p. 141).

Of decided importance is a *unilateral Babinski reflex*, although a bilateral B.R. may also be met with, for instance, in postepileptic coma. With the gradual subsidence of the coma the hemiplegia becomes more noticeable: absent spontaneous movements in arm or leg of one side and diminished reaction to pain in one half of the body. When the patient has completely recovered consciousness, we find (1) central facial paresis and paralysis of the hypoglossal nerve, (2) paresis of arm and leg on the same side and (3) frequently hemianesthesia, sometimes hemianopsia and motor aphasia.

The hemiplegia may, if it does not completely disappear, as it for instance often does in general paresis, remain the same for some time (possibly atonic, now and then with areflexia). After some time, however, it usually *subsides*: certain coarse muscular action (principal movements) is restored, while *finer* movements (synergie) have suffered much, especially as far as the hand is concerned. The hemiplegia which originally was flaccid becomes *spastic* with hypertonic muscles and increased tendon reflexes (p. 142) and the muscles stiffen more and more as *contractures* are being developed: The arm becomes fixed in adduction and slight inward rotation, the forearm in more or less marked flexion, the hand in flex-

ion, the fingers more or less tightly clenched into the hand. The leg is fixed in extension at the hip-knee joint, the foot remains in a more or less pronounced talipes position. The muscles involved in contractures may show some diffuse emaciation, but a *real muscle atrophy is rare, and there is never DeR or fibrillation.*

The *disturbances in the gait* of the hemiplegic are often characteristic, revealing the hemiplegia at first sight: In order to swing the stiff leg forward the patient elevates the limb at the hip and makes an outward semicircular, "scythe-like" movement (circumduction), the tip of the foot being, however, usually audibly dragged along the floor. Other hemiplegics simply walk in a stiff-legged, dragging manner, moving forward the well leg and then bringing the paralyzed one up to it, etc.

The picture of cerebral hemiplegia varies some in accordance with the etiology: in hemiplegia following the *cerebral form of infantile paralysis* we often find but slight affection of the facial nerve, the arm is often contracted in a position of extension, or there is considerable degree of pronation so that the palm of the hand is turned toward the side or backward with the fingers more or less flexed. Hemiathetosis is frequently present as well as *joint movements* (the paralyzed hand is doubled up whenever the patient makes a fist with the well one and so forth). *Hemiaplusia*, i.e., an inhibition of growth in length and circumference of the paralyzed arm and leg is frequently observed.

Cerebral spastic diplegia (sometimes seen in arteriosclerotic encephalomyelomalacia, but more frequently as a congenital paralysis in *Little's disease*) usually leaves the face and in part the arms intact, while the spastic paraparesis is pronounced, being frequently associated with contracture in abduction at the hip joints, and

contractures in flexion at the knee joints, so that the legs "scissor" and the patient has to shove himself forward by jerky, wriggling movements.

In cerebral arteriosclerosis, general paresis, more rarely in lues cerebri and tumors (vascular gliomata) one may meet with abortive *hemipareses*, which develop suddenly without full loss of consciousness and as quickly subside.

(β) Cerebral monoplegia or monoparesis is most frequently seen in diseases of the cortex involving the respective brain centers (Fig. 4), such as tumor, meningitis, emollitions, traumata, general paresis, lues cerebri, etc. They show themselves either as apoplectiform attacks, but without pronounced loss of consciousness, or they develop gradually and are either faciolingual, brachial or crural, or a combination of these types: the faciobrachial, for instance, developing with the spreading of the pathologic process into a final attack of "cortical hemiplegia." The paralysis is usually (1) a paresis (possibly only of a part of an extremity, the fingers, thumb, etc.), (2) it is of an atonic type without muscular atrophy, (3) the tendon reflexes are increased, and when it involves the lower extremities the Babinski reflex may now and then be present. (4) Where there are sensory disturbances these often involve the deeper sensibility joint sense, see p. 154 or astereognosis p. 112. (5) Paroxysmal or continuous clonic spasms may frequently occur in the paretic extremity, occasionally developing into a Jacksonian convulsive attack. The spasms are often followed by an exacerbation of the paresis. (6) When the monoparesis is of the faciolingual type one may sometimes find motor aphasic disturbances.

Concerning "cerebellar hemiplegia" see p. 162.

3. *Psychogenic* (hysterical) *paralyses* may to a cer-

tain extent resemble several of the before-mentioned types of paralysis. They may be recognized by (a) their often *sudden development* (now and then with fainting spells preliminary to convulsive attacks) in association with psychic or mechanical trauma; (b) by the *variation in severity of the paralysis or paresis* which varies with the patient's emotional state, degree of attention and the effects of external influences. Sometimes the paralyzed extremity is more "dead" than the most pronounced organic paralysis. The dynamometer does not give the least intimation of voluntary innervation. In making passive movements one may detect action of the antagonists; if the arm which is being flexed against directed resistance is let go, it does not as under normal circumstances continue on a step further in direction of flexion, but is extended, etc. When emotionally aroused it is sometimes found that pricking, faradic stimulation (these "unmasking tests" should be applied with great care) will result somewhat in the manner of a reflex in a violent movement of the paralyzed extremity. (c) The paralysis is sometimes flaccid, sometimes spastic (i. e., contracture results, see below); but there is *never unilateral hyperreflexia, never genuine clonus, never Babinski reflex, almost never muscular atrophy, fibrillation or DeR.* (d) *The paralyzed portion of the body usually shows sensory disturbances of a characteristic nature* (see p. 159).

The topography varies: *The hysterical hemiplegia leaves the facialis and tongue intact*; sometimes a paresis may be *simulated by contracture of the opposite side* of face and tongue (hemispasmus glosso-labialis). The unilateral sensory disturbance which often is present embraces, as a rule, the face as well as the mucosa of the tongue and nose. If there be a unilateral visual disturbance this is *never a hemianopsia*, but a concentric contraction

of the visual field. *The gait* shows either the dragging of the foot (or of the entire leg) as a dead mass, a load which does not concern the patient, or it is pulled along under loud puffing and moaning, or it is carried about as stiffly as a stilt but without the patient's attempting to get around his "elongated" leg by circumduction, etc., like the organic hemiplegic patient.

If one should bend the fingers of a patient into his hand and then flex the hand upon the forearm, while using the other hand of the examiner in the fixation of the forearm ("rolling up of fingers and hand"), there would normally occur a gradual increase in the flexion of the elbow (with visible contraction of the biceps). This, *Leri's reflex*, is usually *absent* in organic hemiplegias, *never in the psychogenic form*.

The *psychogenic paraparesis* is differentiated from the organic (p. 132) by the state of the skin and tendon reflexes, by the intactness of the vesicorectal sphincters (at the most there is only retention of urine), by the sensory disturbances seldom corresponding in area involved to what is to be expected in any anatomical lesion of the spinal cord, finally by the character of the disturbances in gait, which usually are an abasia. This abasia is either a complete flaccid collapse which results as soon as the patient is put on her feet, or it is a tripping-jerky movement with enforced tumbling from side to side but without falling (as when one attempts to simulate the gait of a drunken person); or it is an irregular swaying of the body, stepping across with dance steps, etc., the whole display being to a great extent easily influenced psychically. Sometimes it is found that the patient is able to crawl on her knees, to hop along, make goose-steps, etc. Abasia may also be met with in

an isolated form; i. e., without paralysis of the lower extremities, when lying on the back.

The *hysterical contractures* may vary in form and topography: (1) they often develop suddenly (compare with the condition found in organic paralysis); (2) they often reflect postures characteristic of affects; (3) they may be psychically influenced (by diversion, surprise, etc.); but (4) are often increased by attempts at correction; (5) they disappear during narcosis but not always during sleep; (6) they are often attended in the region of the contracture by characteristic sensory disturbances; (7) there is no muscular atrophy, no hyperreflexia or clonus, never Babinski reflex. They often develop in regions of psychogenic joint neuralgias where the tenderness is usually located altogether in the soft parts, varying with the emotional state of the patient and where there are no objective signs of joint disease (no redness, swelling, crepitation or rise of temperature and free mobility of joint during narcosis).

A peculiar "*fatigue paralysis*" is found in *myasthenia*: no prolonged paresis, but the muscular movements (speech, eating, looking upwards, handshake, gait, etc.) become rapidly more and more retarded, require more and more exertion on the part of the patient and finally become quite impossible. When faradic or galvanic stimulation is applied to such a muscle we see a corresponding diminution or cessation of the muscular contractions. There are no symptoms of lesion of the pyramidal fibers, no muscular atrophy, as a rule normal sensibility and coördination is intact.

In cases of arteriosclerosis of the vessels of the extremities, most commonly when occurring in the legs (endarteritis obliterans), we may find *intermittent limping*

(claudication). After walking or making use of the hand for some time (five to fifteen minutes), severe pains set in with a "feeling" of paresis. After resting for a shorter or longer time the muscular power has returned to normal and there is no pain. Upon making new attempts at movements the same disturbances repeat themselves, etc. There is no prolonged paresis, the tendon reflexes are normal, there is no myasthenic muscular reaction, but the *peripheral arteries are pulseless* and the x-ray pictures will show arteriosclerosis.

6. Reflexes

A reflex is an involuntary contraction of a single or a few functionally conjointly acting muscles whenever a certain region of the skin ("reflexogenic zone") or the tendon of the muscle is mechanically stimulated. The reflex is brought about through the spinal cord (*the reflex center*): The sensory impulse passes through the peripheral nerve and the posterior spinal roots into the posterior horn on the same side of the cord, from which part it is deviated along a "reflex-collateral" through the gray substance to a cell of the anterior horn of the same side of the cord, around which arborization takes place. From the cell of the anterior horn the centrifugal fibers of the reflex are pass through the anterior nerve roots and the peripheral motor nerve to the particular muscle or muscles in question. Concerning "inhibition" of reflexes through the pyramidal tracts see p. 124.

We differentiate between the *superficial*, skin and mucosa reflexes, the *deep* or tendon reflexes and the *vegetative* reflexes of the bladder, rectum, and genitals.

(a) *The Superficial Reflexes*.—Of these we should al-

ways make tests for the plantar reflex, and the abdominal and cremaster reflexes. Concerning the corneal reflex, see p. 96; the palate reflex, see p. 129.

1. The *plantar reflex*, the center of which is located in the first to second sacral segment of the cord, may be tested by making a light, even stroke on the sole of the foot from before backwards with the finger nail or a blunt point, while the patient is resting on his back or side with the muscles of the leg relaxed. It results normally in a more or less sudden, *associated plantar reflex of all toes* (usually most marked in those on the fibular side). The reflex is a flexion reflex and is combined at the same time with a visible contraction of tensor fasciæ latæ.

Irregular, *evasive movements* result if the stroke, tickling or pricking is *too severe*. They are of no diagnostic importance.

The *plantar reflex* (flexion reflex) is *absent* (a) in conditions of complete unconsciousness; (b) often in hysteria without unconsciousness (sometimes in spite of existing plantar hyperesthesia with evasive movements); (c) where there is lesion of the reflex arc itself.

Where there is a *lesion of the pyramidal tracts* the type of the reflex is found changed: A *minimal blunt* stroke of the plantar surface which is best made along the lateral edge of the foot will result in an *isolated sluggish dorsal flexion of the big toe* known as the *Babinski reflex*, the toes on the fibular side either remaining quiet or being flexed toward the plantar or the dorsal surfaces of the foot. In individuals over two to three years of age, the Babinski reflex is *always* an indication of an *organic nerve disease* (in the absence of coma or narcosis). The reflexogenic zone is sometimes more extended so that a stroke or a pressure on the anterior surface of

the tibia will elicit a Babinski reflex (*Oppenheim's* reflex).

2. *The cremasteric reflex* (first to second lumbar segment) may be elicited by a pinch or a stroke of the inner part of the thigh which causes the testicle of the same side to be drawn upward. It is frequently absent in the aged and under the same conditions as spoken of in connection with the plantar reflex.

3. *The abdominal reflex* (eighth to twelfth dorsal segment). Cross strokes (blunt or with a pin) in the epigastrium on a level with the umbilicus or in the hypogastrium will cause a quick contraction of the abdominal muscles with a movement of the umbilicus in the direction of the stimulated part. It may be absent in fleshy people and otherwise as spoken of in connection with the other reflexes.

Concerning the bulbocavernous and anal reflexes, see p. 147.

(b) *The Tendon Reflexes*.—Of the greatest importance are those of the lower extremities, of which one *should always examine*:

1. *The patellar reflex* (second to fifth lumbar segment): A single blow upon the ligamentum patellæ inferius causes quick contraction of the quadriceps muscle with an extension of the leg.

The *examination* may be made while the patient is sitting with knees crossed or both legs slightly extended so that the soles of the feet are wholly resting on the floor, or the patient may be resting on his back with one leg across the other or slightly flexed over the left hand of the examiner placed in the crook of the knee. With a *heavy* percussion hammer (a machinists' chipping hammer is especially adapted), which is to fall partly by its own

weight, one should make a limp, elastic, (not hard-hitting) blow upon the tendon. *A complete relaxation of the quadriceps is essential.* If the patient can not bring this about himself, (a) the *Jendrassik* reinforcement should be tried: The patient is requested to hook the fingers of one hand into those of the other, and on calling to him "now" he is to pull as if he wished with all his strength to tear one hand from the other. While this is going on the tendon is to be percussed. (b) What is still better and more convenient to the patient and physician is to place the *patient on his side* so that he rests with his entire body (there should be no pillow under the head), keeping the hip and knee in passive flexion. In this position one may at the same time be able to detect the contraction of the quadriceps with the left hand.

When examining women it should be remembered that a too rigorous uncovering of the lower extremities or the genitals may sometimes be the cause of tension in the muscles of the legs. This is easily overcome by a proper covering of the body.

2. *The Achilles reflex* (ankle jerk; fifth lumbar, first to second sacral segment) is best examined when the patient rests on the side which should always be done in doubtful cases by pressing the plantar surface upwards. The tendon is to be put on a stretch to a certain extent so that the position of the foot makes an angle of a little less than 90° with the anterior line of the tibia. Percussion of the tendon should then give a plantar flexion of the foot.

The patellar reflexes (knee jerks) should not, practically speaking, vary during health, and ankle jerks are almost constantly present, even in infants. *Absence of tendon reflexes* (areflexia) in the lower extremities is

therefore as good as always due to an organic nervous disease, but *temporary areflexia* may be found in coma, epileptic convulsions, in recent apoplexies, in recent cases of total transverse lesions of the spinal cord (caused by trauma or hemorrhage) located above the reflex centers. *Prolonged areflexia* is seen in (a) lesions of the reflex arc involving the posterior roots in tabes; the sensory nerves in sciatica (ankle jerks): the reflex center itself in poliomyelitis, diffuse myelitis, traumata, hematomyelia; the motor nerve trunks in neurites, etc. (b) Sometimes where there is a greatly increased brain pressure; in certain cerebellar diseases (p. 163). (c) Frequently in diabetes (peripheral neuritis ?).

Increased tendon reflexes (hyperreflexia) of the lower extremities are found: (a) in strychnia poisoning and tetanus; (b) sometimes in inflammatory diseases of the sensory portion of the reflex arc (neuritis, spinal meningitis); (c) *lesion of the pyramidal tracts* above the reflex centers, occurring therefore usually with hypertonia in systematic diseases of the spinal cord, myelitis in the upper sections of the cord and in cerebral paralyses, etc.; (d) in certain neuroses (epilepsy, hysteria, neurasthenia), and psychoses (catatonia, p. 22), and sometimes after severe hemorrhages, acute infectious diseases, etc.

Unilateral hyperreflexia is nearly always a sign of organic nerve disease.

The hyperreflexia shows itself often as a clonus, (a) *the patellar clonus*. By quick, hard, somewhat elastic jerks of the patella downwards, maintaining this in a fixed position we get a series of rhythmic, rather quick, even contractions of the quadriceps. (b) *Ankle clonus*: By making a quick dorsal flexion, and fixation of the foot (often best done by keeping the knee flexed at an

angle of about 90°) one may get similar rhythmic movements of the Achilles tendon which frequently last as long as the foot is kept fixed in dorsal flexion.

In cases of functional hyperreflexia *pseudoclonus* may be observed. It consists of a series of relatively few, quick, uneven contractions, the excursions of which gradually diminish. It disappears when the muscles become fully relaxed.

3. *The tendon reflexes of the upper extremities* are also quite constantly present. Those tested are *the triceps reflex* (sixth to seventh cervical segment), *the biceps reflex* (fifth to sixth cervical segment), *the supinator reflex* (same segments), *the handflexor reflex* (seventh cervical segment).

The *examination* is made while the patient is lying on his back with the arms placed across the chest, or while he is sitting up with the hands in his lap. The triceps tendon is directly percussed; the biceps is best percussed on the finger laid across the tendon in *plica cubiti*; the supinator tendon is reached on the radial edge of the forearm a couple of centimeters above the line of the wrist (it is often necessary to search quite a little for the reflexogenic zone). The flexor reflex may be observed by hooking one's fingers into those of the patient, lifting the hand; i.e., dorsally extending it a little, and then percussing the tendons.

The *causes* for absence or increase of the arm reflexes are the same as those for the reflexes of the lower extremities. The condition of the arm reflexes is of great importance to the diagnosis and the determination of the level of diseases of the spinal cord, which by predilection are located in the cervical intumescence (amyotrophies, syringomyelia, poliomyelitis, etc.)

(c) *The Vegetative (Excitomotor) Reflexes.*—The

evacuation of *bladder* and *rectum* during the first year of childhood takes place *altogether* by reflex action (automatic intermittent); later on it is to a certain extent under the control of the will power. The evacuation reflex is elicited by stimulation of sensory fibers of the mucosa, (at a certain degree of distention). The motor impulse going to the detrusor muscle passes through *nervus pudendus*. The retention of urine and feces, ("continence"), is controlled by tonic tension of the sphincters. *The reflex-centers* for bladder and rectum are (1) *sympathetic* (ganglion mesentericum inferius, hypogastricum, hemorrhoidale), and (2) *spinal* (located in third to fourth sacral segment) regulating the external sphincters (?). The action of the detrusor is followed by a relaxation of the sphincters and vice versa, the evacuation being *voluntarily* inhibited through the external sphincter muscles or assisted by abdominal pressure or action of *musculus bulbo cavernosus*.

The disturbances of evacuation may be (1) *retention* due to (a) recent lesions above the reflex centers (transverse lesion of the spinal cord, cerebral apoplexy); (b) *tabes* (anesthesia of the bladder mucosa?); (c) purely psychic disturbances (coma, catatonic stupor, dementia, hysteria). (2) In cases of spinal cord lesions above the reflex center there is a gradual development of *automatic intermittent incontinence*: the bladder empties itself quite as though by reflex action at a certain degree of distention, the patient frequently not experiencing any calls of nature to urinate or defecate, or sensations of the passage of the excretions. (3) Lesion of the *reflex centers* themselves should theoretically cause a real vesical and rectal paralysis, i.e., there should be a *constant escape* of urine or feces (dripping of urine—over-

flow); but even here we often see an automatic intermittent evacuation.

In cases of incontinence of the bladder we often find residual urine, cystitis, etc. In *hysteria* we never see real incontinence.

Enuresis (reflex evacuation of the bladder without organic disease of its nervous apparatus), is seen as a stigma of degeneration ("reflex infantilism"), for instance, from fright and during sleep (rarely in the day time). *Strangury* is met with in neurasthenia and hysteria. *Bladder crises* (severe pains in the bladder with tenesmus) are observed in tabes. Frequent interruptions of voluntary micturition ("urinary stammering") occur in incomplete disease of the spinal cord, hysteria and neurasthenia.

The centers of *erection* and *ejaculation* are sympathetic (ganglion hypogastricum), and spinal (associated with the vesicorectal centers). When these centers are affected *priapism* results. In diseases of the sacral portion of the spinal cord, in tabes, in psychoses and neurasthenia we meet with *impotence* (p. 93).

The bulbocavernous reflex. Light stroking of the glans penis gives contraction of musculus bulbocavernosus or a slight erection. Stimulation of the skin surface or mucosa about the anus causes contraction of the sphincter and a retraction of the anus (*anal reflex*), which is often absent in lesion of the conus of the cord.

Vaginismus (painful tonic contraction of constrictor cunni, for instance during coitus) is especially seen in hysteria.

In several of these vegetative reflex disturbances one should think of causes of a *nonnervous* character, such as reno-vesical concretions, cystitis, phimosis (in chil-

dren suffering from enuresis), prostatitis, glycosuria, diseases of internal genitals, etc.

7. Sensation

(a) *Subjective Sensory Disturbances.*—Pains are frequently complained of in organic and functional nerve diseases. Their reality can rarely be established by objective observation, (mimicry, congestion, pulse disturbances, *Mannkopf-Rumpf's* symptom, p. 150). Special forms are:

Headache (cephalalgia) is felt within the head, in the galea and in the skin (a cutaneous hyperalgesia is often noted). It may be diffuse or localized. The causes may be (1) intracranial diseases (*tumor, meningitis, abscess, arteriosclerosis*); (2) diseases of the cranial bones (*tumor, osteomyelitis, lues, in which there are frequently nocturnal exacerbations, and results of traumatic lesions*), or of the integuments ("infiltrations" in the galea and muscles of the neck, etc.); (3) general diseases (*fever, uremia, anemia or chlorosis, gout, alcoholism, nicotine poisoning, pregnancy, etc.*); (4) neurasthenia, hysteria and psychoses. Vomiting and vertigo are frequently found associated with headache.

Neurasthenic headache is often felt as a "tight band" around the head. *Hysterical* headache is usually located at the vertex and felt as a "boring" pain (*clavus*); it is frequently capricious in character, divertible, and combined with cutaneous hypersensitiveness. *Vasomotor* headache is often aggravated by movement of the head (bending forward, looking upwards), by noise, heat, oppressive air (arteriosclerosis, traumatic brain disease, more rarely ordinary neurasthenia).

Migraine (hemicrania, sick headache), is usually con-

stitutional, directly inherited, from infancy, with occasionally a pause at puberty. It is *paroxysmal* (may for instance be connected with the menses), recurring as a rule on the same side, the pain rapidly increasing with vomiting, photophobia, congestion or paleness of the same side of the face, frequently initiated by the characteristic visual signs of scintillating scotomata (p. 103), now and then combined with temporary aphasia, oculomotor paralysis, etc. The pains seldom last more than from a few hours to a day, usually disappearing after a sleep.

In cases of *prolonged headache* one should always examine (1) the urine, (2) the fundus of the eye (neuritis, choked disc), (3) the blood (hemoglobin, Wassermann reaction).

Increased sensibility of the skin to tactile impressions (*hyperesthesia*), to pain (*hyperalgesia*), occurs (1) in organic diseases (with circumscribed topography), (2) in functional disturbances as *topalgias*—clavus (see above), “*ovarie*” (tenderness to pressure in the iliac fossa), tenderness in the inframammary region, etc. The tender points are often *spasmogenic*: pressure elicits wailing, moaning, gasping after breath, tachypnea, quick pulse, congestion, twisting of the body, *arc de cercle*, fear and sometimes hysterical convulsions. The tenderness often disappears when the attention is diverted, etc.

Neuralgias occur as attacks of more or less serial darting pains extending along the course of a peripheral nerve (trigeminal, sciatic nerve, etc.). There is often tenderness upon pressure of the nerve where it lies beneath the skin or upon the bone (*Valleix's points*), or tenderness may be evoked by overstretching of a limb: If the lower extremity is flexed in the hip-knee joint to 90° and a full extension is then attempted at the knee,

pain is developed in the sciatic nerve and there is an interfering muscular tension of the hamstring flexors (the *Lasègue-Kernig* symptom, observed in sciatica, and meningitis). The *rigidity* of the neck in meningitis is an analogous symptom.

In diseases of the posterior spinal nerve root (tabes, meningitis, tumor), there are frequently *radicular pains* with *segmental* distribution (p. 155). The pains occur often in paroxysms, at other times they are more continuous, usually accompanied by objective sensory disturbances: pains may be experienced in *hemiplegic* extremities, especially in diseases of the thalamus.

In cases where pains are *simulated* it has been claimed that Mannkopf's symptom (increased frequency of pulse and blood pressure by pressing the painful place) is absent. This test has been found very unreliable.

Paresthesias (formication, burning, numbness, etc.), are met with in functional and organic nerve diseases. Where the topography is constant (segmental for instance) they may be of some diagnostic importance.

The term *dizziness* or *vertigo* is sometimes among other things applied by patients to, (1) attacks of a moderate degree of fear, to a muddled condition, to having things turn black before the eyes (vertigo after falling from some height, for instance), to abortive fainting spells, a sudden feeling of general languor, fatuity of thought, etc; (2) to real *disturbances of balance*, (swinging sensation, as though the earth were sinking beneath one, as though one were reeling, etc., or were rotating about the longitudinal axis of the body, or as though one's surroundings—persons or objects—were rotating before one's eyes). In such cases there frequently is a *visible* reeling when in an upright position and walking, Romberg's symptom (p. 161). Certain forms

of dizziness are especially present during movements of the head (forward or backward, see p. 164), when exposed to heat, etc. Hysterical and neurasthenic dizziness may sometimes not show any outward symptoms or the patient may show a baroque reeling when Romberg's test is being made (p. 161).

Prolonged dizziness (especially when it is objective), calls for an examination (1) of the eyes (ocular paralysis, ophthalmoscopic examination), (2) of the ears (p. 164), (3) tests having a bearing upon the presence of an organic brain disease, (tumor, arteriosclerosis) or other organic diseases (anemia, nephritis, etc.).

(b) *Objective Sensory Disturbances*.—We distinguish between the *superficial* (cutaneous) sensibility, (touch, pain, heat and cold sensations) and the *deep* sensibility (particularly the "articular sense").

The sensory fibers from the terminal organs of the skin, muscles, tendons and joint surface pass through the peripheral nerve trunks and posterior roots into the spinal cord. Here they become divided up as follows (see Fig. 7): (1) the fibers for the passage of *pain* and *temperature* impressions (γ, δ) which become split up about the cells in the *posterior horns*. Fibers constituting the secondary (lower) neuron take their origin in the posterior horns and pass in an ascending course through the third to fourth spinal segments on the same side, from which they cross through the gray commissure to the opposite half of the spinal cord, where they form the spinothalamic tract (ϵ, η), which passes upwards through the lateral column. (2) The fibers for the *sense of touch and joint sensation* (coming from the muscles, tendons, joint surfaces, α, β) pass virtually *undecussated* upwards through the posterior columns of the spinal

cord (the columns of Goll and Burdach) to cells in the medulla oblongata, around which they subdivide and from which another neuron (ζ) takes its origin and passes centripetally and *decussating to the other side* (the decus-

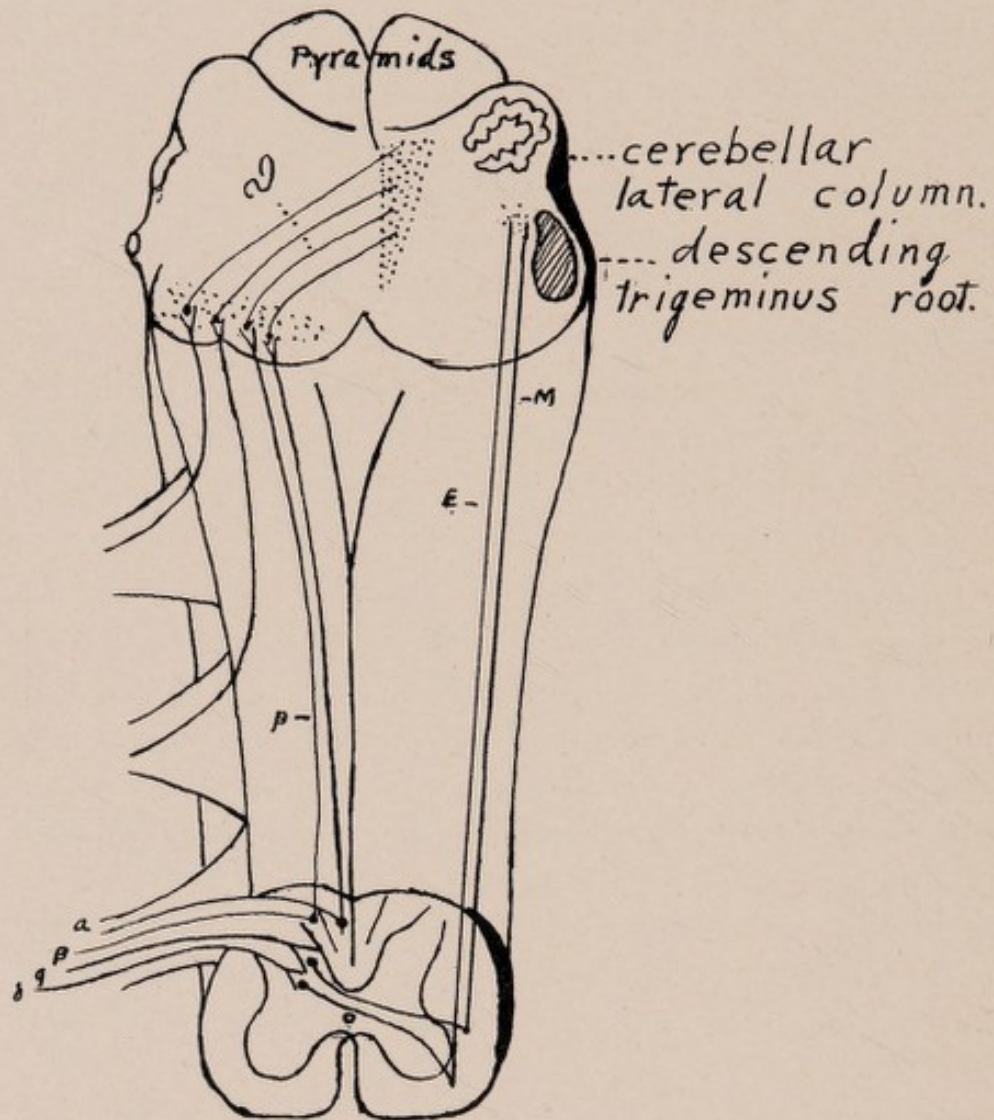


Fig. 7.

sation of the median fillet). From here all the fully decussated sensory fibers pass in a *bundle* through the pons and crura cerebri to nuclei in the *thalamus opticus*,

from which a final sensory neuron conducts the sensory impression to the *cerebral cortex* in the region of the posterior central gyrus and a part of the parietal lobe.

In making the various *sensory tests* we are to a great extent dependent upon the attention, intelligence and good will of the patient. The examination therefore becomes uncertain or has to be abandoned in confused stuporous and demented patients and in conditions of pronounced loss of consciousness (see, however, remarks made below).

1. *The sense of touch (tactile sense)*. The patient's eyes are to be kept covered; a soft camel's-hair brush or tuft of cotton should be used as irritant (single contact, not tickling). The patient is to signal the sensation perceived with a "yes" or the like, or he is informed in advance of the touch to be made with a "now," fooling him from time to time by not letting the expected touch materialize. Aphasic patients may be made to signal with a nod or the like.

One may also let the patient *count* aloud the contacts (at the same time counting to one's self). In case of simulation (and in hysteria) the feigner may after a series of nonsignaled contacts get too high a number, the patient having (unconsciously) counted the nonperceived contacts. Sometimes one will wish to have the patient locate the *point* of touch ("power of localization").

When the disturbances of sensibility are unilateral the sense of touch should always be compared with that of the opposite side of the body.

Diminished sense of touch is known as *hypesthesia*, absent as *anesthesia*.

2. *The sense of pain* is tested with a not-too-pointed needle furnished with handle (etching needle), which

should so far as possible be used with the same force. The perception should be corroborated (a) by the patient's statements, (b) by his mimicry and his evasive or defensive movements (especially when dealing with the before-mentioned disturbances of consciousness and in suspected simulation).

The summation method may sometimes be used to advantage: A series of prickings following quickly upon one another is made in the same spot, making note of the number in the series at which a reaction to pain (verbally, by mimicry, etc.) sets in, after which the test is repeated in the region which is claimed to be anesthetic. (A similar summation effect *by touch* has at times been observed in tabes on anesthetic cutaneous areas.)

Diminution of the sense of pain is termed *hypalgesia*; complete loss, *analgesia*. Sometimes the patient will say that he only feels "something pointed," or that he can not differentiate between "pointed" and "blunt."

3. *The temperature sense* is tested separately for heat and cold effects by means of test tubes filled with water having a temperature of about 50° and at, or slightly above, the freezing point. The test tubes are pressed lightly against the skin for at the most a couple of seconds, the patient being requested to call "warm" or "cold." Sometimes every sensation of temperature is completely absent (*thermanalgesia*), at other times either the sensation of warmth or that of cold is absent; or both "cold" and "warm" are perceived as "cold" respectively "warm" (*thermoperversion*).

4. *The joint or articular sense*: (1) The sense of *muscular movement*: Passive movements of the joints of the body, even of the small ones, and the direction of the movement are normally perceived. The distal bone of the par-

ticular joint is grasped with an even pressure from all sides, in the case of fingers and toes the bone is pressed from side to side, and one is then to make slight, relatively slow movements of the joint, the patient's eyes being covered up. (2) The *posture sense*: If the extremities, fingers or toes are passively placed in certain postures, the normal person should, with eyes closed, be able to describe the particular positions in which they remain, or imitate them with the opposite extremity (fingers or toes).

Disturbances of sensibility are differentiated (1) as to their *nature*, (2) their *intensity*, (3) their *topography*. All qualities of sensation may suffer or only certain ones; the others remaining intact (*dissociation*). Concerning hyperesthesias see p. 149.

The topography and to a certain extent the nature of the sensory disturbances are of decided diagnostic importance.

(a) In lesions of *peripheral nerves* (to a certain extent also of a plexus), the topography of the sensory disturbances corresponds to the territory of the cutaneous innervation of the particular nerve or nerves diseased. When the nerve disease is extensively disseminated (as in polyneuritis) the sensory disturbance is, however, often more diffuse, involving the entire leg or arm, etc., but with irregular ("toothed") outlines. Peripheral sensory disturbances are almost never dissociated. This relates particularly to the cutaneous sensibility.

(b) An isolated lesion of the *posterior horns* (hematomyelia, syringomyelia) usually shows (1) *dissociated sensory disturbances*, affecting pain and temperature sense with complete exemption of the sense of touch and the joint sense (compare Fig. 7); (2) *segmental* (ra-

dicular) *topography*. The different skin territories are not innervated by one spinal cord segment but by *several* segments placed one above the other (plurisegmental innervation; see under Spinal Paralysis p. (131). An affection of one spinal cord segment or of a posterior horn therefore involves sensory disturbances in the domain of *several* peripheral nerves. (With regard to the cutaneous sensory projection of the spinal cord segments see Fig. 8*).

(c) An affection of the *posterior roots* (tabes, luetic meningitis, tumor, etc.) usually also shows (1) a *segmental topography* (in cases of tabes for instance, it frequently corresponds to the upper dorsal segments—a *girdle anesthesia* passing through mammae, anesthetic ulnar bands along the arms, etc.); (2) *the sense of touch and the joint sense* show as a rule earlier and more intense symptoms than the sense of pain; (3) complication with sensory disturbances is frequent in disease of the long nerve tracts of the spinal cord (see below); (4) *pains in the nerve roots* are frequent.

(d) Disease of the *long nerve tracts* in the spinal cord may give rise to a peculiar topography: lesions (tumor, for instance), that are located high up and are on one side may by compression of the spinothalamic tract (p. 152) cause *unilateral dissociated sensory disturbances* (arm, leg, trunk); or they may, as the real unilateral lesions (the traumatic, luetic, etc.), give rise to the Brown-Sequard paralysis: (1) *motor paralysis* (leg or leg-arm) on the side of the lesion with disturbances of the *sense of touch (girdle)* and the *joint sense* in the same area (with ataxia, p. 160); (2) disturbances of the pain and *temperature sense on the opposite side* (see Fig. 7).

(e) *Diffuse diseases of the spinal cord* (transverse

*This figure shows the "lines of direction;" the segmental zones are located on both sides of these lines.

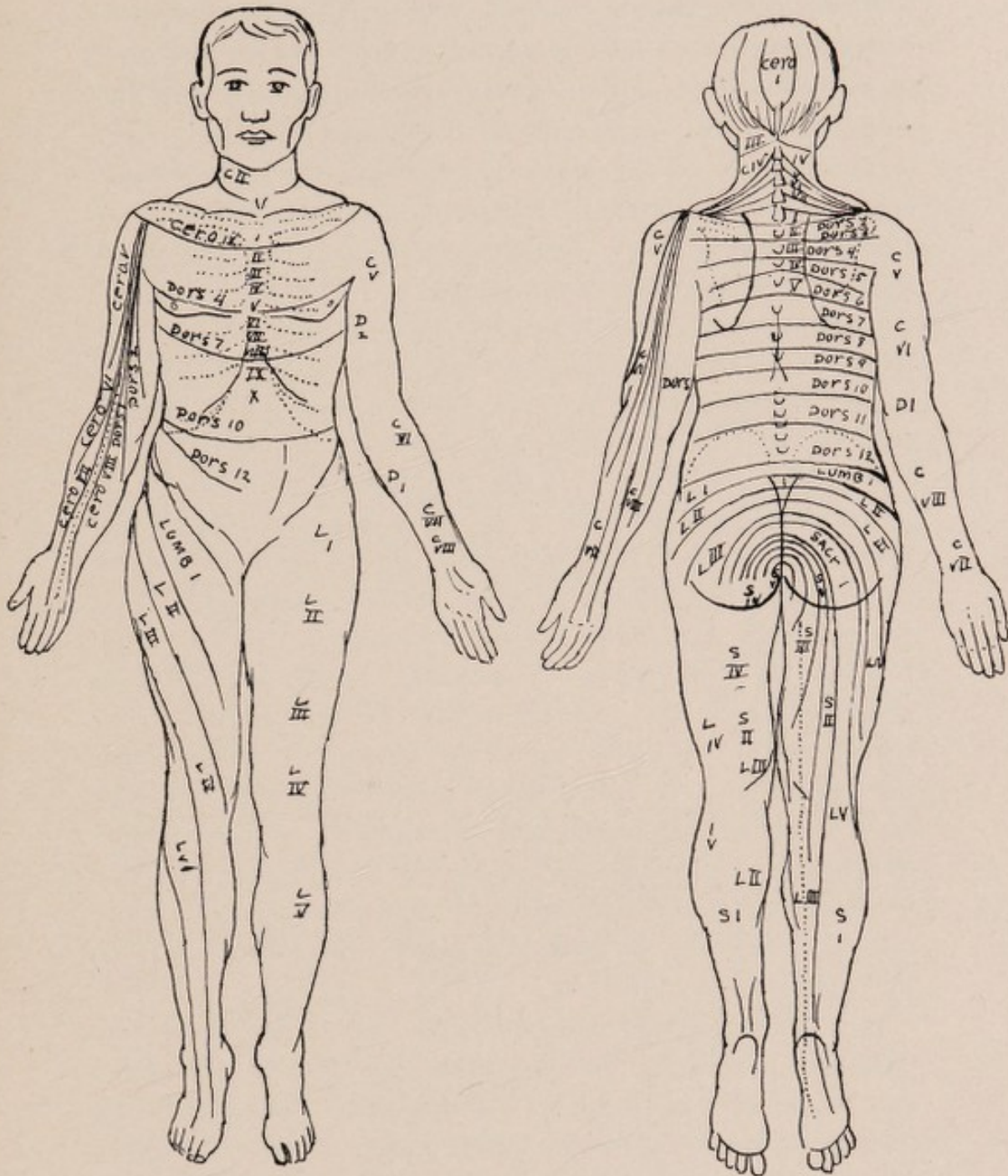


Fig. 8.

myelitis, etc.), give rise to *paraparetic topography*, i.e., sensory disturbances in both legs and on the trunk of the body, the extension corresponding to a point just below the seat of the spinal cord lesion, with circular borders but as a rule without dissociation.

(f) Lesions of the sensory tracts in the *medulla oblongata or pons* may by reason of the special course of the trigeminus fibers (see below) give rise to an *alternating sensory hemiplegia*: sensory disturbances covering one side of the face and the *opposite* extremities. There is at the same time usually a diseased condition of other bulbar cranial nerves (causing dysarthria, paralysis of the palate, difficulty in swallowing and the like).

The sensory fibers of the trigeminus emanate from ganglion Gasseri and enter the pons in a compact bundle from which they, for the most part, pass downward through the medulla oblongata *on the same side*, and through the first two cervical segments (descending root of the fifth nerve) constantly giving off fibers to neighboring cells. A new neuron takes its origin in this locality and *crosses* to the opposite bulbar half, passing from here upwards in association with the sensory tracts of the trunk and extremities. A lateral bulbar lesion may therefore cause an interruption of (1) the undecussated fibers of the trigeminus on the *same side* and (2) the decussated sensory tracts coming from the opposite extremities.

(g) *Lesions in the internal capsule* and the inner and posterior portion of the thalamus give rise to *unilateral and contralateral sensory disturbances* (hemianesthesia and hemianalgesia), which are more or less complete (frequently most marked in the distal parts of the extremities), usually extending slightly across the median line of the trunk of the body and showing as a rule par-

ticular involvement of the sense of touch and pain. There is at the same time usually present a hemiplegia, a hemianopsia, possibly a hemichorea or hemiathetosis and an astereognosis, especially in lesions of the thalamus where we also often find hemiataxia (p. 162), and hemiplegic pains (p. 150).

(h) *Cortical lesions* frequently lead to sensory disturbances limited to an arm, leg and one side of the face. This is as a rule of less intensity (hypesthesia, hypalgesia). An isolated affection of the *joint sense* with *astereognosis* is also frequently present. The diagnosis is corroborated by the monoparetic or monospastic symptoms present, etc.

(i) *Psychogenic* (hysterical) *sensory disturbances* may simulate several of the above-mentioned organic diseases, but will then often show (1) a sudden development following psychic or mechanical trauma with symptoms on the same side or at the seat of the mechanical lesion ("local hysteria"), and the existence of a prolonged psychic susceptibility; (2) an affection as a rule only of the *superficial* sensibility, but often to an extreme degree; (3) a *closely circumscribed territory*; (4) frequently a *peculiar topography* not in keeping with that of the organic disturbances: the hemianesthesia ceases abruptly in the median line, the areas of sensory disturbances on arms and legs show distinct limitation by circular "amputation-lines," making "gloves" "sleeves," "socks," "trouser-legs," etc., or they may be irregularly distributed as "islands" all over the body; (5) they are almost *never dissociated* as is the case, for instance, in syringomyelia.

One should always remember that *hysterical sensory disturbances are not infrequently associated with organic*:

(j) Sensory disturbances met with in mental diseases are usually general in character, most frequently

in the form of hypalgesia or analgesia (observed in confusion, catatonic stupor and profound mental states).

8. Disturbances of Coordination

The effective performance of a muscular movement presupposes a harmonious cooperation of *several* muscles, a constant adaptation of the motor impulses as to force and consecutive order. The ensemble of the muscular mechanism (coordination) is brought about (1) by anatomicophysiological trained synergies (a mutual operation of two muscles with a motor excursion in the direction of the resultant force; for instance, the iliopsoas and tensor fasciæ latae), by concert of action between *agonists* and *antagonists* (resulting in a gradation of the movement); or by muscular cooperative action for the fixation of a joint by movement of its distal parts (for instance of the wrist by clenching of the fist). (2) Further anatomicophysiological coordination takes place through *the centers of coordination in the spinal cord* (?) and particularly in *the cerebral cortex*. (3) *During the muscular movement* there is a constant regulation (a) through *centripetal sensory impulses* coming from the parts moved (joint sense, p. 154); (b) through *centrifugal impulses, coming from the cerebellum* or the static apparatus of the ear (p. 163); (c) through the *sense of vision*. Abolition of *a* or *b* gives rise to *incoordination* (ataxia, asynergia). The principal forms of incoordination are:

(1) *Spinal ataxia* due to affection of the centripetal sensory impulses (tabes, Friedreich's ataxia, etc.). The ataxia shows itself during (a) voluntary movements of the extremities, (b) when the patient is in a standing posture and (c) in the gait.

Tests of Ataxia.—(1) *The upper extremities:* The patient is asked to bring his index finger as far as possible away from the body and thereafter to carry it not too quickly to the tip of the nose (*the finger-nose test*); or he is asked to stretch out both arms to the sides and then bring the points of the two index fingers together (*the finger-finger tests*). In ataxia the movements are unsteady and fumbling with irregular deviation from direction of the movements, often missing the mark, etc. (2) *The lower extremities:* The patient lying on his back is asked to lift up one leg and place its heel down upon the knee of the other leg (*the knee-heel test*). When ataxia is present, the leg will be lifted unnecessarily high, is swung about in the air and is thumped with needless force upon the knee-pan or by the side of it, etc. *The tests are first to be made by the patient with eyes open, afterwards with closed eyes, which will tend to increase the ataxia or perhaps not until then make it apparent.*

(3) *Romberg's symptom:* The patient is required to stand upright with heels and toes together and look straight forward, when, if ataxia is present, swaying of the body and a slight balancing upon the heels may be observed. *When the eyes are covered,* the swaying will become more noticeable or he may tumble about. The test may also be made by having the patient stand on one leg with open and closed eyes (*Fournier's test*; often difficult for a perfectly healthy person).

In a case of hysterical, or simulated "Romberg" the disturbances of the equilibrium are often quite grotesque and the attempts of the ataxic patient to make balancing movements with the feet are frequently lacking.

(4) *Ataxic disturbances in gait*: The patient walks unsteadily, places his feet on the ground with unnecessary vehemence, knocks his heels into the floor, lifts the swinging leg needlessly high, carries it with flinging movements, is unable to walk along a straight line on the floor and tries very carefully to control his walk with his eyes. When the disturbances in gait are slight the ataxia is sometimes most apparent when the patient is to turn around quickly; he then reels and has to step heavily about. The gait is still more defective with eyes closed.

The spinal ataxic gait (in tabes and Friedreich's tabes), is furthermore characterized by (a) *an absence of paresis* of the legs, (b) by *a marked hypotonia* of the musculature: During the walk the legs become knock-kneed, and are capable when fully extended of being flexed upon the abdomen to an abnormal extent, etc.

In cases of *diffuse neuritis* in the lower extremities, for instance in the alcoholic form, the gait may resemble very much the tabetic ("pseudotabes alcoholica"), but one usually then finds (a) a certain degree of paraparesis (ataxic-paretic gait); (b) never pupillary rigidity, and extremely seldom incontinence of bladder and rectum.

In *diseases of the cortex* (in the anterior central convolution and frontal lobes) and in affections of the thalamus we now and then see hemiataxic disturbances.

(2) *Cerebellar and vestibular disturbances of coordination* show themselves especially as disturbances (a) of the upright position (b) of the gait, and (c) of the arm-leg movements, particularly of those that lead to defective fixation of the *large joints*.

The coordination apparatus in question are: (1) *Centripetal* tracts (to the cerebellum), receiving peripheral

sensory (but unconscious) impulses through (a) posterior spinal roots, posterior horns with arborization around the cells of *Clark's columns*, from which another ascending neuron passes via the "direct cerebellar tract"—*all in the same half of the spinal cord* through *crus cerebelli inferior* to the cerebellum; (b) *the vestibular tract* receiving peripheral stimulation from the semicircular canals (by means of the movements of the endolymph), through the vestibular nerve to cell groups in the medulla oblongata (especially *nucleus Deiteri*), where another neuron originates, bringing fibers in part to all the *ocular muscles* (including also those of the opposite side), in part to the *cerebellum*. The vestibular tract is likewise *undecussated*. (2) *Centrifugal tracts* sending fibers from the cerebellum to Deiter's nucleus, from where they pass undecussated through the spinal cord as *tractus vestibulospinalis* with distribution of fibers to cells in the anterior horns of the *same side*. Aside from this, the cerebellum has centrifugal and centripetal connections with the cerebrum, especially with the frontal lobes, the basal ganglia, etc., and is also connected with the vagus, (vomiting spells).

The cerebellar or vestibular symptom-complex consists of: (1) *Atony* (usually hemiatony), muscular flaccidity, which must not be confounded with paresis, and sometimes *areflexia*. The atony is due to loss of centrifugal tone, affecting impulses passing through the vestibulospinal tract. (2) *Astasia*: The equilibrium in the upright position is unsteady, the patient staggers so it is often necessary for him to straddle to prevent falling. The disturbances of equilibrium are *not* increased by closing the eyes. (3) *Abasia*: The patient walks like a drunken person, with feet wide apart, is dangling, tumbling and frequently experiences falls, often in a certain

direction (see below). The individual movements of the lower extremities are, as a rule, not incoordinatory as in spinal ataxia. (4) *Asynergies* are present in other intentional muscular movements: if the patient lies prostrate on his back with crossed arms and he then attempts to get up, the legs will not as normally rest in complete contact with the underlying surface, but are lifted into the air, so that the patient "rocks" on his nates. When quick successive pronation and supination are made with the hand (as in fencing), the muscular excursions on the diseased side will become slow, jerky, irregular, arrhythmic, awkward, (*adiadokokinesis*—the experiment is normally always more defectively performed with the left hand). (5) *Nystagmus* is frequently present (see below).

In unilateral cerebellar lesions the disturbances are always on the same side as the lesion.

Examination of the vestibular functions (Bárány).

(1) *The caloric test:* By irrigating (the irrigator being but slightly elevated and the nozzle being directed against the anterior wall of the ear duct) the external ear duct for about 20 seconds with water of a temperature of 68° to 86° F., a *vestibular nystagmus* in the direction of the unirrigated ear will be produced if the vestibular function is normal, the nystagmus lasting one-half to two minutes. The reaction is absent or diminished in diseases of the labyrinth and the vestibular nerve trunk (tumor involving the acoustic nerve and the like). (2) *The rotary test:* If the patient is rotated rapidly on a special "revolving chair" about ten times in twenty seconds and in the same direction, a nystagmus in a direction *opposite* to that of the revolving movement will be developed by sudden stoppage of the rotation (best observed through smoked glasses). (3)

The pointing test: If the patient presenting spontaneous or experimental nystagmus is asked with uncovered eyes to touch an object, for instance, a certain line on an immovable graduated rod, and he is then asked to repeat this with *closed eyes*, he will point *past the mark in a direction opposite* to that of the nystagmus. (4) *The fall test:* In a case of vestibular nystagmus the patient will fall to the *side opposite* to that of the nystagmus when he stands erect looking straight forward. *If the head is turned 90 degrees* in the direction of that of the nystagmus, he will fall *forward*. If the head is turned 90 degrees in a direction *opposite* to that of the nystagmus, he will fall *backwards* ("as if the fixation of the head decided the direction of the fall").

9. Examination of the Cerebrospinal Fluid

The liquor cerebrospinalis is found in the subarachnoidal space surrounding the brain and the spinal cord and filling the cerebral ventricles. Normally it measures in amount 60 to 100 c.c.; the pressure is equal to 60 to 125 mm. of water when the individual is lying down and up to 410 when he is sitting up. It is clear like water, feebly alkaline, contains but few cells and embodies colloidal substances such as albumins, as stated below.

The pressure of the cerebrospinal fluid and its contents of formed elements and albuminous substances are subject to change in a series of *organic* mental and nervous diseases. *Examination of the spinal fluid is required* in: (1) all *meningitic diseases*, (2) when there is a *history of lues*, (3) in all conditions of dementia or in psychotic or neuropathologic disease pictures in persons of 40 to 50 years of age in whom the diagnosis is uncer-

tain, (4) in cases in whom there is reason for suspicion or a probability of *luetie disease of the nervous system* (paresis, tabes, and cerebrospinal lues), and (5) sometimes in *conditions of unconsciousness* when urinary examination gives no information.

The spinal fluid for the examination is obtained by *lumbar puncture* (Quinke), which is, generally speaking, a procedure free from danger and which is contra-indicated only when there are signs of, or a probability of, *tumor of the brain*, especially in the posterior cerebral fossa.

Technic of the Puncture.—The conus medullaris of the cord extends to a point opposite the first or second lumbar vertebra (in children sometimes to the third). The *puncture* is made in the *third* (or the *fourth*) *lumbar space*, that is, in the space which is just above an imaginary line connecting the two superior posterior spines of the ilei. The patient is to *lie* either on his side with knees bent on the chest and with back bent forward as much as possible, or he is to *sit* on a small table, leaning well forward and bending the back with buttocks placed as close as possible to the farther edge of the table. Especially made needles on the principle of the hypodermic (nickel or platinoiridium), 4 to 10 cm. long, 1 to 3 mm. thick, with slanting and carefully fitting stilet and *most carefully sterilized* are used. The physician making the puncture should have sterile hands and the skin of the back where the puncture is to be made, should be painted with *iodine*. Sometimes local anesthesia (ethyl chloride) is employed. *Children* should be punctured exactly in the median line, *adults* $\frac{1}{2}$ to 1 cm. on either side of the median line, with the direction of the needle slightly toward the median line and slightly upwards. It is sometimes necessary to feel your way between the

arches of the spinal column. In children the puncture is made to the depth of about a centimeter, in adults 5 cm., in very fat persons sometimes double this distance.

The spinal fluid flows in a stream where there is hypertension or in drops in the average case, and is collected in a sterilized tube. When the fluid is bloody, it should be allowed to run until it becomes clear and then be collected in 2 or 3 separate tubes. *The rule is to secure about 5 c.c. of the fluid.* The needle is quickly removed, the skin opening compressed with sterile cotton, dried, and covered with cotton collodion. The patient is to remain 24 hours in bed, preferably on the back without support of a pillow.

Examination of the Spinal Fluid.—This comprises:

(1) The *Wassermann reaction*, (2) the *cell count*, (3) the *albumin determination*, and sometimes (4) the *bacteriologic examination*.

(1) About 1 c.c. of fluid is sent in the usual way for the *Wassermann test*.

(2) The *Cell Count* (for this about $\frac{1}{2}$ c.c. of fluid is required) is made with *Fuchs-Rosenthal's counting chamber*.

The surface of the chamber is divided into 16 squares of 1 mm. each. Thus the entire area of the chamber is 4 x 4 mm. or 16 sq. mm. The chamber is 0.2 mm. deep, and its cubic contents are therefore $0.2 \times 16 = 3.2$ c. mm. The spinal fluid having been mixed in the pipette with diluting solution in the ratio of 1 to 9 (1 part diluting solution and 9 parts of spinal fluid) we have the following calculation:

$$x = \frac{c \times 10}{3.2 \times 9} = \frac{10c}{28.8} = \text{about } \frac{c}{3} = \text{number of cells in 1 c.mm.}$$

Where c = total number of cells counted in the entire ruled surface of 16 sq. mm.

Into a Zeiss mixing pipette for white cells a staining solution consisting of 0.10 gm. methyl violet, 50.0 c.c.aq. dist. and 2.0 c.c. glacial acetic acid is drawn up to mark "1," after which the spinal fluid is added to the mark "11" and the pipette well shaken for about 5 minutes. The first drop is rejected, after which a drop is put into the counting chamber. A counting is made of all squares with a lens of medium magnification.

The total number of cells found, divided by 3, gives the cell number per c.mm. A cell count of 10 and upwards per c.mm. is pathologic, between 5 and 10 doubtful.

An increase of cells (pleocytosis) deals principally with *lymphocytes*. Leucocytosis is met with in tuberculous, purulent, and epidemic forms of meningitis and in brain abscess.

3. *The Albumin Determination* includes "globulins" and "total albumin amount."

The examination is made ad modum *Nonne-Bisgaard* with the *Bisgaard armamentarium* (see Fig. 9). The principle involved in the method is partly that of *Heller's* urinary test; namely, to produce a ring-like coagulation of the borderline between the spinal fluid and the reagent, and partly by a *systematic dilution* of the spinal fluid, so as to find the greatest degree of dilution which barely gives a reaction. *The figure representing this titration index is an indicator of the degree of reaction.*

(a) *The Globulin Reaction (Nonne's "Phase I")*.—By means of pipette *b* (divided into $\frac{1}{50}$ c.c.), the spinal fluid is aspirated to mark 0.33 and transferred into a clear test tube. With another pipette salt solution (0.9 per cent) is aspirated to the line indicating 0.67 and added to the spinal fluid in the test tube. The mixture

is then well shaken. By means of pipette *c* an *equal part* (i.e., 1 c.c.) of a *saturated neutral solution of ammonium sulphate* is added by allowing it to trickle down the side to the *bottom* of the test tube. This mixture is

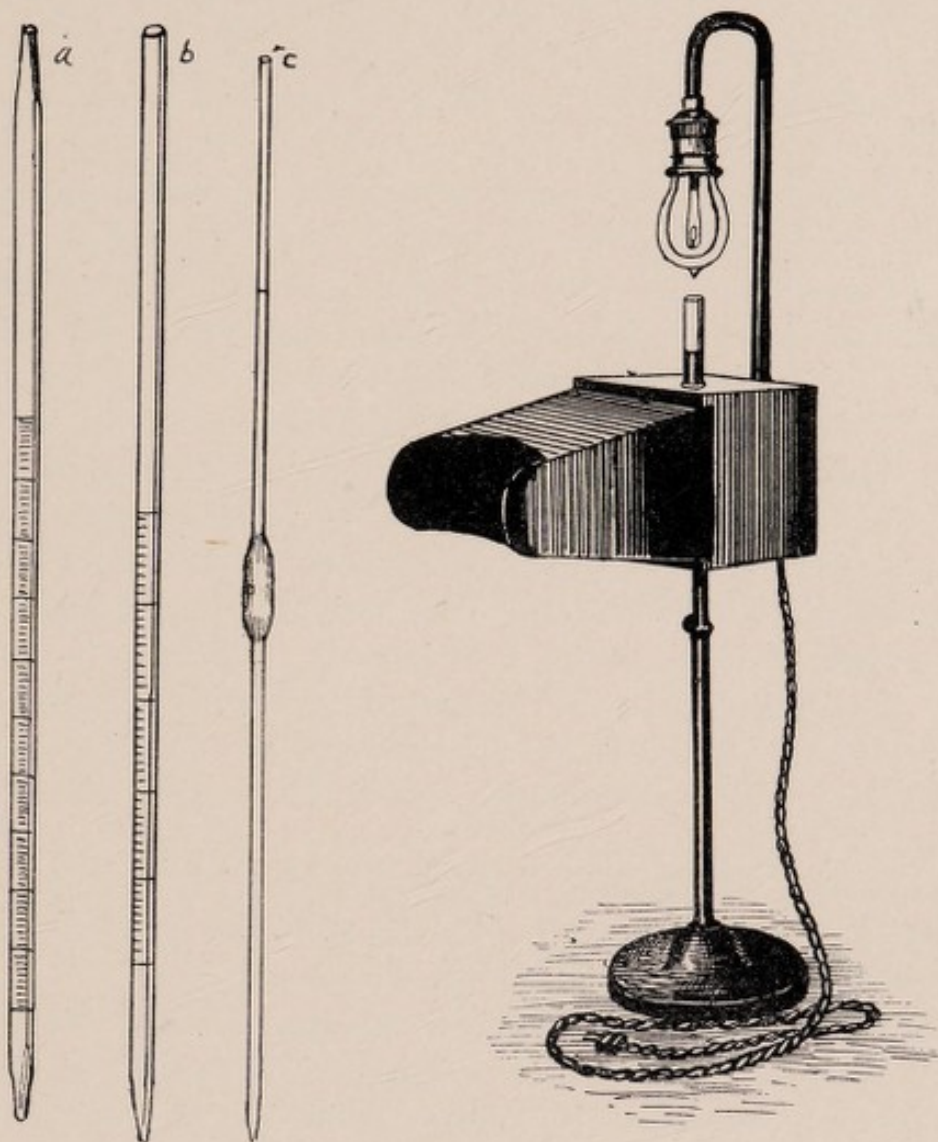


Fig. 9.

then put into a dark box or camera-like box closed at one end (Fig. 9), and left *untouched for 3 minutes*, after which the possible ring coagulation is observed through

the central opening of the protruding part of the box which allows the exclusion of light. A positive reaction is expressed by the figure "3" which is the index of the titration and is to be followed by tests for *higher degrees* of dilution (0.2 c.c. spinal fluid, 0.8 c.c. NaCl solution = titration "5," etc.).

Reaction obtained at a dilution index of above 2 is pathologic.

(b) *The Total Amount of Albumin.*—A stock solution is made by taking 0.2 c.c. spinal fluid into one of the graduated pipettes and mixing this with 1.0 c.c. chloride of sodium solution (10 times diluted). Of this 0.5 c.c. is to be taken and mixed with 0.5 c.c. NaCl solution. By means of pipette *c* 1 c.c. *nitric acid* is to be allowed to trickle into the bottom of the test tube which is then placed in the camera box to remain for 3 *minutes* after which the ring is read off as before stated. If the reaction is positive, the total "albumin amount" is expressed by the figure "20" (i. e., the dilution index). One should then as before test with higher degrees of dilution (0.33 c.c. of the stock solution to 0.67 c.c. of the NaCl solution, a positive reaction being indicated by "30," etc.).

Reaction obtained at a dilution over 20 is pathologic.

4. *The Bacteriologic Examination.* In forms of meningitis the spinal fluid may contain: *Diplococcus lanceolatus*, *Diplococcus intracellularis meningitidis* (*Weichselbaum*), tubercle bacilli, staphylococci, etc., these may sometimes be found by smears stained with methylene blue according to *Gram*, or *Ziehl-Neelsen*, etc., but frequently only through cultures.

In cases of tumor formation about the meninges (carcinoma, sarcoma) tumor cells specific to the particular form may be found. In recent cerebral hemorrhage

sometimes numerous erythrocytes are present. Spinal fluid of a *yellow color* (xanthochromia) may be observed, for instance following brain trauma, old hemorrhage and tumor about the brain and spinal cord.

Pleocytosis is found: (a) in syphilitic cerebral disease (see below); (b) in cases of purulent, epidemic and tuberculous meningitis, (c) in certain other diseases, such as herpes zoster and septic, general infections, etc.

An increase of albumin is found in the same conditions as just stated under *a* and *b*, and it is inconstantly observed in genuine (?) epilepsy, disseminated sclerosis (sometimes combined with pleocytosis), in tumors of the brain and spinal cord, in compression myelitis unaccompanied by pleocytosis and in cerebral arteriosclerosis, etc.

The spinal fluid is *normal in contents* in alcoholic neuropsychic disease, manio-depressive psychosis, dementia precox, Huntington chorea, paralysis agitans, bulbar paralysis, syringomyelia, spinal muscular atrophy, diffuse myelitis, and usually in acute poliomyelitis.

The Pleocytosis, the Increase in Albumin, the Wassermann Reaction (W. R.) of the Spinal Fluid Combined with W. R. of the Blood Serum ("the Four Reactions") Are of Decided Diagnostic Importance in the Consideration of Luetic Neuropsychic Disease.

1. *In General Paresis* we find as a rule all four reactions positive; W. R. in the spinal fluid being found positive with small quantities of liquor (0.2). There is a marked globulin reaction as compared with the nitric acid reaction and a medium or pronounced pleocytosis.

2. *In Tabes* we usually find pleocytosis; most often "Phase 1;" the nitric acid index slightly increased or normal, W. R. of the liquor, but frequently not obtained until larger quantities of the fluid are used (0.6 to 1 c.c.), W. R. of the blood in about 70 per cent of the cases.

3. *In Luetic Meningoencephalitis* (and isolated gummata): frequently marked pleocytosis, greatly increased nitric acid index, relatively low globulin index, positive W. R. in the fluid but sometimes not until larger quantities are used, most frequently a positive W. R. of the blood.

4. *In Luetic Endarteritis*: pleocytosis, increase of albumin as under 3, the W. R. of the fluid is *often negative* even with undiluted fluid, the W. R. of the blood is as a rule positive.

In cases of endarteritis of the small vessels of the cortex all reactions of the spinal fluid may be negative.

10. Serologic Examination

At the present time it is principally the Wassermann test which is of importance to the psychiatric-neurologic diagnostique but its importance is often very great. *The indications* for the performance of the test are as a whole those mentioned in connection with the lumbar puncture. A positive reaction of the blood serum can of course *but* show that the patient has contracted syphilis, *not eo ipso*, that the nervous or mental disease from which he is suffering is luetic, just as a negative result of the test does not exclude a possible syphilitic psychosis or nervous disease. In such case the lumbar puncture must be resorted to, to fill out the gap.

With regard to the technic of the Wassermann test we refer to the textbooks on serology.

The dialysis method of Abderhalden for the demonstration of "decomposition products" in the blood has not as yet given diagnostically useful results as pertaining to mental diseases. The same may be said of the pharmacologic functional tests.

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