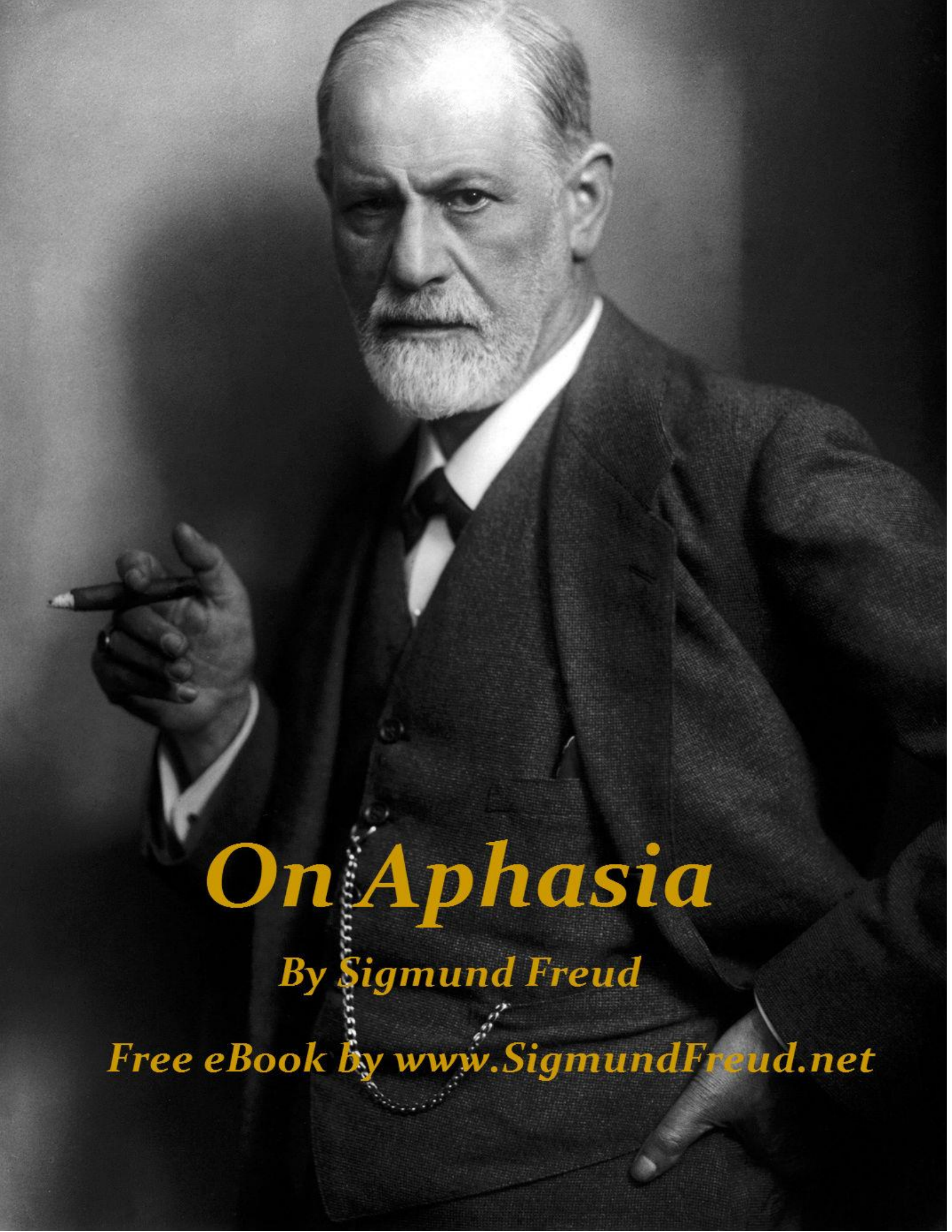

On Aphasia

By Sigmund Freud

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Introduction

After a firm basis for the understanding of cerebral disturbances of speech had been established by the discovery and definite localization of a motor and a sensory aphasia (Broca and Wernicke), the authorities set about tracing the more subtle symptoms of aphasia as well to factors of localization. In this way they arrived at the hypothesis of a conduction aphasia, with subcortical and transcortical, and motor and sensory forms. This critical study is directed against this view of speech disorders and it seeks to introduce for their explanation functional factors in place of the topographical ones. The forms described as subcortical and transcortical are not to be explained by a particular localization of the lesion but by conditions of reduced capacity for conduction in the apparatus of speech. In fact there are no aphasias caused by subcortical lesion. The justification for distinguishing a central aphasia from a conduction aphasia is also disputed. The speech area of the cortex is seen rather as a continuous region of the cortex inserted between the motor fields of the cortex and those of the optic and auditory nerves--a region within which all communication and association subserving speech function takes place. The so-called speech-centres revealed by the pathology of the brain correspond merely to the corners of this field of speech; they are not distinguished functionally from the interior regions; it is only on account of their position in relation to the contiguous cortical centers that they produce more obvious signs when they become disordered.

The nature of the subject treated here called at many points for a closer investigation of the delimitation between the physiological and the psychological approach. Meynert‘s and Wernicke‘s views on the localization of ideas in nervous elements have had to be rejected and Meynert‘s account of a representation of the body in the cerebral cortex has required revision. Two facts of cerebral anatomy, namely (1) that the masses of fibres entering the spinal cord are constantly diminished on passing upwards, owing to the interposition of grey matter, and (2) that there are no direct paths from the periphery of the body to the cortex - these two facts lead to the conclusion that a really complete representation of the body is present only in the grey matter of the cord (as aprojection‘), whereas in the cortex the periphery of the body is only represented in less detail through selected fibres arranged according to function.

Clinical Studies of the Unilateral Cerebral Palsies of Children.

(In collaboration with Dr. O. Rie.)
(No. III of Beiträge zur Kinderheilkunde edited by Dr. M. Kassowitz.)

A monograph describing this affection, based on studies of material in the First Public Institute for Children‘s Diseases in Vienna, directed by Kassowitz. In ten sections it deals with (1) the history and literature of the cerebral palsies of children;(2) 35 observations of the authors‘ own, which are then summarized in tabular form and described individually; (3) the analysis of the individual symptoms of the clinical picture; (4) the pathological anatomy; (5) the relations of cerebral palsy to epilepsy and, (6) to infantile poliomyelitis; (7) differential diagnosis and (8) therapy. Achoreatic paresis‘ is described by the authors for the first time; it is distinguished by peculiar characteristics in its onset and course, and in it the unilateral paresis is from the beginning represented by hemichorea. There is further an account of the findings of an autopsy (lobar sclerosis as a result of an embolism of the middle cerebral artery) on a woman patient described in the Iconographie de la Saltpêtrière. Emphasis is laid on the close relations between epilepsy and the cerebral palsies of children, in consequence of which some cases of apparent epilepsy might deserve to be described ascerebral palsy without palsy‘. In connection with the much discussed question as to the existence of a polioencephalitis acuta, which is supposed to constitute the anatomical basis of unilateral cerebral palsy and to offer a complete analogy with poliomyelitis

infantilis, the authors argue against this hypothesis of Strümpell's; but they hold firmly to the expectation that a modified view of poliomyelitis acuta infantilis will allow of its being equated with cerebral palsy on another basis. In the therapeutic section are collected the hitherto published reports on the intervention by brain surgeons directed to the cure of genuine or traumatic epilepsy.

A case of successful treatment by hypnotism with some remarks on the origin of hysterical symptoms through "counter will".

A young woman after the birth of her first child was compelled to give up breast-feeding it owing to a complex of hysterical symptoms (loss of appetite, sleeplessness, pains in her breasts, failure of milk-secretion, agitation). When, after the birth of a second child, these obstacles recurred, deep hypnosis on two occasions, accompanied by countersuggestions, succeeded in removing the obstacles, so that the patient became an excellent nursing mother. The same result was brought about a year later in similar circumstances after two more hypnoses. Some remarks are appended on the fact that it is possible in hysterical patients for distressing antithetic or anxious ideas to be realized which normal people are able to inhibit; several observations of tic are traced back to this mechanism of counter-will.

An obituary of the master of neuropathology who died in 1893 and among whose pupils the present writer numbers himself.

On a symptom which often accompanies enuresis nocturna in children.

In perhaps half the cases of children suffering from enuresis we find a hypertonia of the lower extremities the significance and implications of which are unexplained.

On the psychical mechanism of hysterical phenomena.

(Preliminary communication in collaboration with Dr. J. Breuer.)

The mechanism to which Charcot traced back hystero-traumatic paralyses, and the assumption of which enabled him to provoke them deliberately in hypnotized

hysterical patients, can also be made responsible for numerous symptoms of what is described as non-traumatic hysteria. If we put the hysteric under hypnosis and lead his thoughts back to the time at which the symptom in question first appeared, a memory of a psychical trauma (or series of traumas) belonging to that time awakens in him with hallucinatory vividness, the symptom having persisted as a mnemic symbol of the trauma. Thus hysterics suffer mainly from reminiscences. If the traumatic scene which has been arrived at in this way is reproduced vividly, accompanied by a generation of affect, the symptom which has hitherto been obstinately maintained disappears. We must therefore suppose that the forgotten memory has been acting like a foreign body in the mind, with the removal of which the irritating phenomena cease. This discovery, first made by Breuer in 1881, can be made the basis of a therapy of hysterical phenomena which deserves to be described ascathartic.

The memories which are revealed aspathogenic, as the roots of hysterical symptoms, are regularlyunconscious to the patient. It seems that by thus remaining unconscious they escape the wearing-away process to which psychical material is normally subject. A wearing-away of this sort is brought about by the method ofabreaction. Pathogenic memories avoid being dealt with by abreaction either because the experiences concerned have occurred in special psychical states to which hysterical persons are inherently inclined, or because those experiences have been accompanied by an affect which brings about a special psychical state in hysterical persons. A tendency to asplitting of consciousness‘ is accordingly the basic psychical phenomenon in cases of hysteria.

An Account of the Cerebral Diplegias of Childhood (in Connection with Little‘s Disease.) (No. III, New Series, of Beiträge zur Kinderheilkunde edited by Dr. M. Kassowitz.)

A supplement to the Clinical Study of the Unilateral Cerebral Palsies of Children summarized under XX above.The history, pathological anatomy and physiology of the affection are treated here in the same order as in the earlier monograph, and the relevant clinical pictures are illustrated by 53 observations made by the author himself. It was, however, necessary in addition to take into account the range of forms that must be described ascerebral diplegias and to point out their clinical similarity. In face of the differences of opinion that prevail in the literature of these

disorders, the author has adopted the standpoint of an earlier authority, Little, and has thus arrived at the erection of four principal types, which are described as general spasticity, paraplegic spasticity, general chorea and bilateral athetosis, and bilateral spastic hemiplegia (spastic diplegia).

General spasticity includes the forms which are usually referred to asLittle‘s disease‘. Paraplegic spasticity is the name given to what was earlier regarded as a spinal affection, tabes spastica infantilis. The spastic diplegias correspond most easily to a doubling of unilateral cerebral palsies, but are characterized by a superfluity of symptoms which finds its explanation in the bilateral nature of the cerebral affection. The justification for including general chorea and bilateral athetosis among these types is provided by numerous characteristics of the clinical picture and by the existence of many mixed and transitional forms which link all these types together.

A discussion follows of the relations of these clinical types to the aetiological factors which are here assumed to be operative and to the insufficient number of post-mortem findings that have been reported. The following conclusions are reached:

Cerebral diplegias can be divided according to their origin into (a) those congenitally determined, (b) those arising at the time of birth and (c) those acquired after birth. But it is extremely rarely that this distinction can be drawn from the clinical peculiarities of the case, and not always possible from the anamnesis. All the aetiological factors of the diplegias are enumerated: prenatal (trauma, illness, or shock affecting the mother, place of the child in the family); operative at the time of birth (the factors stressed by Little, namely premature birth, difficult labour, asphyxia); and after birth (infectious diseases, trauma or shock affecting the child). Convulsions cannot be regarded as causes but only as symptoms of the affection. The aetiological part played by inherited syphilis is recognized as important. There is no exclusive relation between any one of these aetiologies and any one type of cerebral diplegia, but preferential relations are often apparent. The view that cerebral diplegias are affections with a single aetiology is untenable.

The pathological findings in the diplegias are of many kinds, and in general the same as in the hemiplegias; for the most part they are in the nature of end stages,

from which it is not invariably possible to infer back to the initial lesions. They do not as a rule allow of a decision as to the aetiological category to which a case is to be referred. Nor is it usually possible to deduce the clinical picture from the post-mortem findings; so that the assumption that there are intimate and exclusive relations between clinical types and anatomical changes must also be rejected.

The pathological physiology of cerebral diplegias has an essential connection with the two characteristics by which both general and paraplegic spasticity are distinguished from other manifestations of organic disease of the cerebrum. For in both these clinical forms contracture predominates over paralysis and the lower extremities are affected more severely than the upper ones. The discussion in this paper reaches the conclusion that the more intense affection of the lower extremities in general and paraplegic spasticity must be connected with the localization of the lesion (meningeal haemorrhage along the median fissure) and the preponderance of contracture with the superficiality of the lesion. The strabismus of diplegic children, which is particularly common in paraplegic spasticity and where premature birth is the aetiology, is traceable to the retinal haemorrhages in new-born children described by Königstein.

A special section directs attention to the numerous instances of the familial and hereditary occurrence of children‘s diseases which show a clinical affinity with cerebral diplegias.

On familial forms of cerebral diplegias.

An observation of two brothers, one six and a half and the other five years old, whose parents were blood relations, and who present a complicated clinical picture which has gradually developed, in the one case since birth and in the other since the second year. The symptoms of this familial disorder (lateral nystagmus, atrophy of the optic nerve, alternating convergent strabismus, monotonous and, as it were, scanning speech, intention tremor of the arms, spastic weakness of the legs, accompanied by high intelligence) give grounds for constructing a new affection which is to be regarded as a spastic counterpart to Friedreich‘s disease. Emphasis is laid on the far-reaching similarity of these cases to those described as multiple sclerosis by Pelizaeus in 1885.

The cerebral diplegias of children.

A summary of the findings in the monograph abstracted above.

Some points for a comparative study of organic and hysterical motor paralyses.‘

A comparison between organic and hysterical paralyses made under the influence of Charcot in order to arrive at a line of approach to the nature of hysteria. Organic paralysis is either periphero-spinal or cerebral. On the basis of discussions in my critical study on the aphasias, the former is described as projection paralysis and is paralysis en détail, and the latter is described as representation paralysis and is paralysis en masse. Hysteria imitates only the latter category of paralyses but has freedom to specialize which makes it resemble projection paralysis; it can dissociate the areas of paralysis which regularly occur in cerebral affections. Hysterical paralysis has a tendency to excessive development; it can be extremely in tense and yet strictly confined to a small area, while cortical paralysis regularly increases its extent with an increase in its intensity. Sensibility behaves in a directly contrary manner in the two kinds of paralysis.

The special characteristics of cortical paralysis are determined by the peculiarities of cerebral structure, and allow us to infer back to the anatomy of the brain. Hysterical paralysis on the contrary behaves as though there were no such thing as cerebral anatomy. Hysteria knows nothing of the anatomy of the brain. The alteration which underlies hysterical paralysis can have no resemblance to organic lesions but must be looked for in the conditions governing the accessibility of some particular circle of ideas.

The neuro-psychoses of defense: an attempt at a psychological theory of acquired hysteria, of many phobias and obsessions and of certain hallucinatory psychoses.

The first of a series of short papers which now follow and which are directed to the task of preparing a general exposition of the neuroses on a new basis which is now in hand.

The splitting of consciousness in hysteria is not a primary characteristic of this neurosis, based on degenerative weakness, as Janet insists. It is the consequence of a peculiar psychical process known as defense which is shown by some short reports of analyses to be present not only in hysteria but in numerous other neuroses and psychoses. Defense comes into operation when an instance of incompatibility arises in ideational life between a particular idea and theego‘. The process of may be figuratively represented as though the quota of excitation were torn away from the idea that is to be repressed and put to some other use.. This can occur in a variety of ways: in hysteria the liberated sum of excitation is transformed into somatic innervation (conversion hysteria); in obsessional neurosis it remains in the psychical field and attaches itself to other ideas which are not incompatible in themselves and which are thus substitutes for the repressed idea. The source of the incompatible ideas which are subjected to defense is solely and exclusively sexual life. An analysis of a case of hallucinatory psychosis shows that this psychosis too represents a method of achieving defense.

Obsessions and phobias: their psychical mechanism and aetiology.

Obsessions and phobias are to be distinguished from neurasthenia as independent neurotic affections. In both it is a question of the linkage between an idea and an affective state. In phobias the latter is always the same, namely anxiety; in true obsessions it can be of various kinds (self-reproach, sense of guilt, doubt, etc.). The affective state emerges as the essential element of the obsession, since it remains unaltered in the individual case, whereas the idea attached to it is changed. Psychical analysis shows that the affect of the obsession is justified in every instance, but that the idea attached to it represents a substitute for an idea derived from sexual life which is more appropriate to the affect and which has succumbed to repression. This state of affairs is illustrated by numerous short analyses of cases of folie du doute, washing mania, arithmomania, etc., in which the reinstatement of the repressed idea was successful and accompanied by useful therapeutic effects. The phobias in the strict sense are reserved for the paper on anxiety neurosis