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THE MIND TWIST AND BRAIN SPOT HYPOTHESES IN PSYCHOPATHOLOGY AND NEUROPATHOLOGY

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When my friend and colleague, Professor Angier, desired that I should write a review of my general attitude toward the problems of psychopathology in their relation to the more general problems of cerebral function and of consciousness, I was minded to refuse. The point was that I considered that my ideas were little more than a mass of unproved hypotheses. However, I had just been meditating on the results of the first ten years of the Bullard Professorship of Neuropathology in the Harvard Medical School, and found myself able to draw up without great difficulty a sketch of my various unproved hypotheses, some of which I here present.

Perhaps I should preface this account of a point of view by some remarks which I hope will not be over personal. Psychologists, and especially psychiatrists, while dealing with personality day by day, are too often loath to display their own on paper. When at the Triennial Medical Congress at Washington, in 1910, I was moved in discussion to denominate two great groups of friendly opponents in the field of psychiatric theory respectively as the "mind twist men" and the "brain spot men," I was reproached by some of my best friends with making light of a grave matter. The phrases are, to be sure, of little moment; but I consider that the distinction between those who uphold the hypothesis of psychic factors as opposed to those supporting the hypothesis of encephalic factors must be drawn if we are to make any sort of progress in genuine psychopathology. The ardent parallelists (among whom, I must confess, I should not like to be numbered) would, I suppose,

say that mind twists and brain spots are all one, since everything depends upon the aspect from which one works. The methodical purist might indeed assert that he who dealt in mind twists should not commingle therewith any data concerning brain spots; and the anatomist would be sure to resent a commingling of the psychic with his own localizations. For my part, without any stringent proof, I feel that somehow the hypotheses which for better or worse I was fain to describe as the mind twist and brain spot hypotheses are in some sense and in the long run identical hypotheses. I have indeed endeavored to give expression to the concept of their essential identity in a paper entitled, "Psychopathology and Neuropathology: the Problems of Teaching and Research Contrasted,"[1] and I pointed out how pernicious in research may be the dogmatic insistence on the doctrine of psychophysical parallelism in medical and pre-medical courses in psychology, pernicious because it inhibits the free interchange of structural and functional concepts and the passage to and fro of workers in the several sciences. I went on to show that psychology and physiology have more in common than either has with such structural sciences as anatomy and histology, and that the main common element of both mental and cerebral processes is the time element as against the space element of the structural sciences. On this ground I further conceived that the mind twist and brain spot hypotheses for the explanation of certain forms of mental disease are entirely consistent with each other, since from a different angle each is dealing with the same facts.

My point of view here is not quite naïve or quite so innocent of metaphysical speculation as the anatomist often pretends to be. The attitude in question is one strongly influenced by my work for some years past in Professor Josiah Royce's Logical Seminary, in which the fundamental concepts of science have been taken up. I should not wish, however, to convey the impression that Professor Royce is in entire agreement with my point of view.

Any logician must, however, be readily convinced that the current classroom distinctions between organic and functional disease, especially between organic and functional nervous diseases, are flimsy distinctions. They often amount to saying that a disease shall remain functional only so long as the microscope or other technical tool shall fail to prove their organic nature. Such distinctions may be practical; I have even heard them termed prag-

[1] *American Journal of Psychology*, 1912, 23.

matic, although I doubt whether the true pragmatist could see much use in the distinctions as drawn. The concepts of structure and function have also been considered among Professor Royce's varied seminary topics, and several definitions have been proposed. The most interesting of these runs to the effect that the functional among diseases is the disease which is reversible, either practically or theoretically, in such wise that the original condition can be approximately regained. It is obvious that this definition, if sound, will not jibe altogether with the one above mentioned, namely: the vague concept of the functional as that which has not yet been proved to be structural.

In securing a working definition of the functional in disease, it will always prove necessary to adopt some definition of disease itself. Two obvious lines of distinction have occurred to Royce's seminarians, which may be briefly characterized as leading to the concept of the abnormal and the concept of the morbid. The abnormal is very possibly an entirely quantitative distinction, including as its leading varieties the supernormal and the subnormal. The greatly supernormal or greatly subnormal may be termed anomalous; but anomalies are not necessarily, although they prove often to be attended by, diseases. The morbid (this term is for some reason indelicate and has been somewhat illogically replaced with the term *pathological* in modern writing) may consist in, or be produced by, the abnormal; but a deeper account of the morbid is probably to consider it as a name for conditions which somehow defeat the evolutionary use or object of the cell or mechanism in question. Thus, a condition which entails the premature death of the cell or a loss of its important appendages or organoids would be a morbid condition. Thus the concept of pathology would have at its core the teleological concept of the morbid, but would have as a rule also to consider those quantitative variations from the normal which we gather under the term *abnormal.* It is a profound, but here not especially pertinent, question how far the concept of the morbid is itself also a quantitative affair. But the main point I here make concerning the concept of the morbid is that it is a biological concept and not a broadly physical one.

I should not venture here to offer these truisms if I were not convinced that the psychologist in the academic sense seems to believe, and at any rate often leads his students to believe, that psychopathology is in some sense a science of psychical anomalies; that is, of psychic processes that are figureable in curves at the

upper or lower end of the normal curves. When the academic student begins to get a grip upon the essential problems as they are presented by a patient, or by those remains of human beings which often yield the greatest returns for a given amount of investigation, he discovers that a science of supernormal and subnormal measures leaves him entirely at loose ends and does not get him a millimeter onwards with his problem. Here, it does not do to speak with authority; yet the returns to the committee of the American Psychological Association, on which I served, as those returns were prepared by Professor Franz, indicated that those concerned with the problem of interrelations betwixt psychology and medicine were split into camps along the above lines, namely: A camp of those claiming the virtues of studying the quantitatively anomalous, and a camp of those who wish to study the biologically non-adaptive or the evolutionarily unfit.

So much by way of preaching. As a practical method of getting the students, and particularly the graduate students, to appreciate the science of the psychiatrist's problem as the pathologist sees it, I have in the last few years come to express the idea in somewhat the following terms: I first beg the student to consider the nervous system as theoretically reducible to a linear system of neurones, separated in the Sherringtonian manner by synaptic planes. I then point out that, if given muscles are convulsed as in an epileptic attack, we cannot safely state that the spinomuscular neurones which supply the convulsed muscles are in any respect abnormal, except that we must admit that their existence and participation are necessary for the production of the convulsions in question. Similarly with the corticospinal neurones, whose impulses are conceived to run into the spinomuscular neurones at the appropriate synaptic planes (under various conditions of inhibition and reinforcement which may be neglected). In epilepsy, we do not often discover that the corticospinal neurones are any more visibly diseased than are the spinomuscular neurones. In point of fact, the analysis of epilepsy, as of a great variety of neuropathological conditions, involves considering one by one, at greater and greater removes from the seat of the physical phenomenon of convulsions, the successive neurones which are indispensable in the production of the symptom but are not responsible therefor. In epilepsy, in point of fact, in the majority of cases in which science has at all made out the immediate cause of the convulsions, it has been discovered that the seat of the lesion is not in the motor neurones but

on the afferent side of the apparatus. These considerations for epilepsy I brought together in a paper entitled, "On the Mechanism of Gliosis in Acquired Epilepsy."[1] I there pointed out the relation of my own "microphysical" theory, and its endeavor to describe certain pericellular conditions which might well bring epilepsy about, to the "level" theories of Hughlings Jackson, as well as the relations of my theory to the Sherringtonian concept of synapses. In that paper I gave a highly demonstrative case of epilepsy of nine weeks' duration, terminating in four days of practically continuous convulsions due to a virtually non-destructive lesion of a sensory area. There was a focal encephalitis of the right cornu ammonis which, whatever its cause, exhibited an interstitial accumulation of neuroglia cells, leaving the nerve cells virtually intact. My hypothesis was that fresh surfaces of separation had been interposed between sets of nerve elements. I supposed that these elements, having their currents in the forward direction, and being placed under fresh conditions of intimate pressure, would initiate continuous or lasting stimuli, which would set the remainder of the apparatus moving in an abnormal fashion.

The point which I wish to make for the present purpose is that in the case of epilepsy just mentioned, and in a vast majority of cases of neuropathy of every sort, we may well suppose that the neurones which lie outside the focus of disease, and the muscles, glands, or other organs which they supply, may be entirely normal and executive of their normal functions. I tried to sum this concept up in the following phrase: Neurones may be intrinsically normal whereas extrinsically abnormal; entirely normal structures may accordingly purvey and be necessary in the production of disease.

This simple concept of the intrinsically normal yet extrinsically abnormal or morbid is of great use in psychopathology. I find it dominating my own methods of thinking. When public attention was directed, in the period just preceding the Washington Congress above mentioned, to the problem of dementia præcox, it seemed to me that very probably the brains of dementia præcox patients would be found to be normal; at least it was true that some of the most eminent psychiatrists had been unable to discover, in the majority of cases of mental disease (in which field dementia præcox must largely bulk), anything abnormal, let alone morbid. Although it had become a household word that insanity was brain disease,

[1] *American Journal of Insanity*, 1908, 64.

yet there was little or no evidence or hope that the brain disease would be soon discovered. Under this assurance, I wrote a section of my paper on dementia præcox entitled, "A Study of the Dementia Præcox Group in the Light of Certain Cases showing Anomalies or Scleroses in Particular Brain Regions."[1] I there pointed out that the disease diabetes mellitus, being distinguished among other diseases by the production of great amounts of urine with an abnormal amount of sugar, might well be conceived by the tyro as a kidney disease. Yet upon investigation, it turns out that the lesions of the kidney in diabetes mellitus are negligible and inessential, and that the disease itself must be related to remote or unknown organs. This analogue points the way to a broadening of the concept of the intrinsically-normal-but-extrinsically-abnormal to include other elements than neurones, and indeed to include the chain of organs which we latterly suppose are concerned in the production of internal secretions. Just as the intrinsically normal kidney is extrinsically abnormal in diabetes mellitus in the sense that it purveys a large amount of sugar in the urine, so *might* the brain in dementia præcox be intrinsically normal yet extrinsically abnormal, in the sense of producing delusions, catatonic excitement or stupor, or other characteristic symptoms whose genuine origin might conceivably lie entirely outside the nervous system.

This was my conception of the probabilities with respect to dementia præcox when I entered upon the study of a series of brains in that disease in preparation for the Congress of 1910. It still remains my conception of conditions in the sister disease, manic-depressive insanity; but in dementia præcox I was greatly surprised to find that the vast majority of cases were distinguished as to their brains by the possession of distinct though mild lesions in the nature of anomalies, atrophies, or scleroses, which in so labile an organ as the brain must perforce have their effects upon brain functions.

This long preamble is probably justifiable in preparation for showing why a psychopathologist should find himself a localizer despite logical predilections against brain localization for psychic processes. I may briefly state the view to which my as yet unproved hypotheses seem to be leading, as follows: It seems to me that just as a tremendous leap forward was taken when the Flourens view of the interchangeability of brain parts was sup-

[1] *American Journal of Insanity*, 1910, 67.

planted by a roughly localizing view, and when the bilaterality of brain function began to be unravelled, along with the data showing the seizing of some functions by one hemisphere as against the other, so we may be now in the process of a great advance as we come to a full recognition of the value of distinguishing the parts of the cortex which lie forward of the fissure of Rolando and above the fissure of Sylvius from the parts which lie behind and below those fissures. For it seems to me that the indications are strong that the silent portions of the pre-Rolandic areas of the cortex, forming the anterior association center of Flechsig, are predominantly motor in function; whereas the correlative backward-lying association center is predominantly sensory. Just as it is convenient at some times to divide the earth into an eastern and a western hemisphere, and sometimes into a northern and a southern hemisphere, so it may be well for many purposes to distinguish the left hemisphere of the brain from the right, but for other purposes it may turn out that the pre-Rolandic and supra-Sylvian portions of both hemispheres, with their concomitant commissural fibers in the corpus callosum, should be fairly sharply distinguished from the post-Rolandic and infra-Sylvian regions of the cortex. It is true that the forward "hemisphere" is of far less bulk than the rearward and nether "hemisphere"; but this difference in size is only another illustration of the difference which holds throughout the nervous system between the afferent and efferent fiber systems which compare quantitatively always much in favor of the afferent.

It does not appear that the theoretical distinctions which are possible between these two portions of the cortex have been developed as elaborately as they should be by either the anatomists or the physiologists or the pathologists. I have personally been led to wonder whether there is any basis for considering the pre-Rolandic tissues as having anything whatever to do with consciousness, that is, with *consciousness in its cognitive sense*. This was the burden of my communication at the New Haven meeting of the American Psychological Association in the closing days of 1913.

I arrived at this idea in a concrete fashion. I found in the course of my anatomical analysis of dementia præcox brains that cases with frontal lesions were chiefly cases distinguished by the possession of delusions; that is, belonged to the so-called paranoid group of dementia præcox (to employ Kraepelin's 1899 classification). I found that the catatonic cases were correlated, not so much with pre-Rolandic atrophies, as with atrophies of the post-

central, parietal, or in some cases cerebellar, tissues. This seemed at first glance a surprising correlation, since delusions are apparently of a psychic texture, whereas the muscular spasms, inhibitions, stereotypies, and impulsivities of catatonia, suggest the efferent rather than the afferent system. Upon reflection, however, it appeared that a similar apparent difficulty lodged in the sensory basis of many epilepsies, as alluded to above. After all, it was not the content of the delusions which was so important to the patient; it was the process or formation of these delusions. It was not so much the false beliefs with which either society or the patient himself was concerned; it was rather with the maintenance of the falsely believing process, the morbid will to believe. Every one's working day is a kaleidoscope of false beliefs. But luckily they correct themselves or get supplanted in such wise that a normal attitude ensues. The psychopathology of insane delusions was consequently to be interpreted rather as a psychology of false believing, and was better conceived as a matter of behaviorism than of introspective psychology. Thus, whether my anatomical correlations were sound or not, I was able to arrive at an interesting concept of delusions as a form of conduct rather than as a form of static mental contents.

On the basis of this concept, I was led to analyze delusions of the various groups, classified (as we had by chance chosen to classify in the Danvers symptom catalogue) according to Wernicke into autopsychic, allopsychic, and somatopsychic. I quickly found that somatic delusions are far more representative of actual visceral conditions than is usually held. It is accordingly possible to conceive of many somatic delusions as virtually illusory in nature. Conclusions in this direction were published in a paper "On Somatic Sources of Somatic Delusions."[1]

This led to a study of allopsychic delusions, namely: those false beliefs dealing with the environment and especially with the social environment, which was published with A. W. Stearns in a paper entitled, "How Far is the Environment Responsible for Delusions."[2] The majority of these cases were found to be more truly instances of autopsychic or personal delusions, than environmental. This study was followed by one on the correlation between delusions and cortex lesions in the pronouncedly organic disease general paresis.[3] Incidentally, we here again found that the in-

[1] *Journal of Abnormal Psychology*, 1912–1913.

[2] *Journal of Abnormal Psychology*, 1913.

[3] With A. S. Tepper, *Journal of Abnormal Psychology*, 1913.

stances of somatic delusions sometimes complicating the picture of general paresis were usually attended by an adequate peripheral basis. Thus, a patient who described himself as blind but as having a filter over his eyes in such wise that he could see, turned out to be the victim of cell losses in the visuo-psychic type of cortex, with maximal pigmentation of the neuroglia cells. The patient should have expressed his delusion by saying that he could see but had a cortical veil preventing his perceiving properly.

More important, however, was the discovery that autopsychic delusions and that characteristic ruin of personality which we classically assign to general paresis must be correlated with frontal lobe lesions. In the non-autopsychic group, we found the lesions distributed elsewhere than in the frontal region; that is to say, we found these non-autopsychic cases failing to show the classical frontal brunt of the distinctive process. Here, then, was concrete evidence that the personality, conscious as it seems, was more closely related with the pre-Rolandic than with the post-Rolandic tissues; with the efferent mechanism more than with the afferent mechanism. It seems to me that here again we are securing evidence which supports to some extent the objectivistic or behavioristic trend in modern psychology. It seems possible that psychopathology, even in the exquisitely psychic fields of the delusions, will not gain so much by an endeavor to ferret out the innermost psychic secrets of the patient as by a careful quantitative study of his reactions in the line of conduct. If some method could be devised for obtaining the survival values of these actual processes of conduct rather than their academic quantitative values, we should be so much further on the road to a behavioristic psychopathology.

A similar line of thought follows suit in respect to catatonia. This exquisitely muscular phenomenon, like the similar phenomenon of epilepsy, turns out, as it seems to me, to be more a sensory than a motor affair. Just as delusions had less sensorial significance than they had significance on the side of action, so the catatonic and cataleptic phenomena turn out to have less significance from the side of action than from the side of the sensorial, or at all events the afferent, apparatus. Again discounting the question whether the anatomical correlations in dementia præcox upon which I founded this idea are sound or not sound, it is clear that some heuristic value must fain attach to this concept. It is, in any event, important to consider how far catalepsy is actually due to a sort of morbid kinæsthesia. Suppose a postcentral disease which should

provide to the surfaces of separation between neurones their initial stimulus, purveying as it were quasiperipheral stimuli of a given kinetic quality, then perchance the remainder of the general mechanism although quite normal (*intrinsically*) would have to react in the cataleptic way. What might seem to be a will disturbance, or a disturbance initiated in or near the precentral gyrus as a form of abnormal or morbid conduct, might perchance be executed by a thoroughly normal precentral and frontal mechanism on the basis of abnormal or morbid conditions in the post-central region. Upon this basis might be built up, in short, a kinæsthetic or quasikinæsthetic theory of catatonic and cataleptic phenomena.

An illustration from less disputed fields may serve to bring out the point. It is well known that experimental physiology has shown that there are two centers for conjugate deviation; namely, a center in the angular gyrus, which lies posterior to the Rolandic fissure, and a center in the middle frontal gyrus which lies anterior. Should we regard the results of stimulating these two areas as entirely similar? Are we to suppose that projection fibers run directly from both these cortical areas to the appropriate oculomotor neurones? Are we not rather to suppose that they stand to one another in some logical sense resembling that in which the area of Wernicke for sensory speech stands to the area of Broca for motor speech? Can we perhaps generalize that many or the majority of the complex functions for which the cerebral cortex is built are thus doubly supplied fore and aft by mechanisms which on the one hand are more closely related to conduct or behavior elaboration, and on the other hand to kinæsthetic or cognitive elaboration?

This leads me to quote with as much disapproval as I becomingly can from Wundt's expression of his anti-localizing views in the first volume on speech in his *Völkerpsychologie*. Wundt decries the conception that every cortical brain cell harbors some idea. The unregenerate physiologist, according to Wundt, holds the conscious conception that deposits of different ideas or thoughts are distributed over the cortex in districts; one for sound impressions, one for visual images, etc. These compartments of the cortex, according to Wundt, were conceived by illogical physiologists as in part occupied by ideas, and in part engaged ahead for future occupants. Destruction of a center for ideas would of course destroy ideas deposited; but fortunately these destroyed ideas could be replaced by new ones occupying cells now vacant. Such a restoration of function would not differ essentially from the process

of normal brain development so long as still disengaged cells remained available. Prior to the work of Broca, in 1861, according to Wundt, every one had thought vocal sounds to be of physical origin but words of psychic origin. A word, to be sure, required the physical aid of sound production, but nevertheless every word was really the outcome of a concept and was exactly as much a psychic affair as desire or will; but Broca showed that motor (or better, according to Wundt, ataxic) aphasia depended on lesions of a certain part of the brain (inferior frontal convolution). Wernicke followed in 1874 by showing that sensory (or better, according to Wundt, amnestic) aphasia depended upon lesions of a certain other part of the brain (superior temporal convolution). Then followed Kussmaul's scheme in 1877, which as modified by Lichtheim in 1885, seemed to show that at least thirteen different kinds of aphasia might be produced by lesions appropriately placed in different parts of the brain. Then followed the work of Meynert and the work of Munk.

I suppose there can be no objection to this schematic account of the history of the doctrine of aphasia as developed by Wundt, but I should now wonder whether it is advisable to consider that any idea, or at all events any cognitive process, can or ought to be related to such an area as that of Broca. The area of Broca, like the area near by for conjugate deviation, or the so-called graphic center or similar congeries of interrelated elements, may be supposed to be, or to take part in, a synergic mechanism for one or other purpose. The frontal part of the brain is doubtless full of these synergic mechanisms. The negation of personality entailed by frontal lobe disorder indeed indicates that the synergic mechanisms, or kinetic patterns or schemata, normally contained in the anterior association center are even capable of novelty production, of the faculty of innovation, upon which our title to supremacy as human beings depends. Accordingly, I should wonder whether the analysis of the effects wrought in such areas as that of Broca was not more a matter of behavioristic psychology than of introspective. Some might inquire whether it were not well to consider such an area as entirely physiological in its action. To this form of expression, I should have no objection if it be understood that in some way or other we must explain the correlation of personality with these forward lying cell systems. It is probable that these cell systems of the anterior association area are every whit as much entitled to psychological consideration as the cell systems of the

posterior association center. Yet the operations of the latter are very possibly on theoretical grounds far more open to introspective study than are the operations of the forward lying cell systems.

Naturally, the products of the action of the precentral gyrus or of the area of Broca do get representation on the cognitive side, that is mainly in the kinæsthetic manner and doubtless more back of the fissure of Rolando than forward thereof. On this account, introspection has been an important, or even essential, method in the analysis of behavioristic problems, since the kinæsthetic or other similar record of what is being done will often serve as the best guide for the actual course of events when the behavioristic method itself may not yet be able to cope with technical difficulties. On the other hand, after all, what the psychopathologist as well as the psychologist wants to register is the acts and deeds,—that is, the conduct,—of the individual, and on this account the operations of the anterior association center of Flechsig are of prime value.

In partial support of these ideas, I have recently studied the literature and certain casualty ward records with post-mortem studies available to me, with the object of learning how far what we term clinically consciousness and unconsciousness are consistent with extensive lesions of different parts of the cortex. Already the Crowbar Case and other similar cases stood to prove that the frontal cortex might be seriously injured without permanent impairment of consciousness in the slightest degree. An affection of character might well ensue, indeed is described as having ensued in the crowbar case; but it does not appear that consciousness, or at all events selfconsciousness, was lastingly injured in that classical case.

The considerations of this latter study led me to consider the meaning of the term consciousness. It seems to me that the term should be restricted to what it etymologically seems to signify, namely, cognition and compounds of cognition. It seems to me that the components of will and possibly those of emotion are entirely, or almost entirely, gotten into what we call consciousness by the cognitive route of kinæsthesia, and that there is great question how much elementary introspective stuff there is to the will and the emotions which cannot better be accounted for on the basis of kinæsthesia. If this account of consciousness as in a sense cognitive is a good simplification of nomenclature, I would suggest that a similar simplification in the field of the so-called unconscious is sadly needed. Some textbooks on psychology

seem to identify consciousness with mind. The unconscious is, according to these authors, surely much more than the non-conscious, and indeed has been hypostasized into a novel and mystical entity having all the old warmth and intimacy of the so-called conscious and many strange intimacies besides. If one tries various current definitions of the unconscious by replacing the term *unconscious* by such a term as *non-conscious* or *non-mental*, one discovers how much balderdash has been inflicted upon us by many exponents of mystery.

So much will suffice for a doubtless far too personal and over dogmatic account of my reaction to the present situation in psychopathology and psychology. I do not vouch for the ultimacy of any of the ideas expressed, and must place upon my friend, Professor Angier, all responsibility for the premature delivery of possibly non-viable children of fancy.

Summary.—I am sure that some of the dozen or more separate conceptions to which I have asked attention in the above review will hardly carry conviction in the present sketchy form.

1. The *mind-twist versus brain-spot hypotheses* have nowhere been discussed *in extenso* (although see articles on "The Problems of Teaching and Research Contrasted and a Study of the Dementia Præcox Group," etc., mentioned in text), and I am not sure that the distinction will strike the reader as more than a fresh sample of psychophysical parallelism. Without special title to a viewpoint, I wish however to say that personally neither parallelism nor interactionism seems to me safe ground and that some kind of identity hypothesis for all the operations concerned would be better consonant with my views. One thing will be clear from the above sketch, viz., that it may well be possible that mental operations of the introspective kind are not correlatable (in any sense) with a good part of the operations of the cerebral cortex.

2. The definition of *consciousness as equivalent to cognition and compounds of cognition* leaves the non-cognitive portions of the mind (will and emotions) only capable of introspection by the kinæsthetic and allied sensorial routes. But, whether the above definition is correct or not, it is at least clear that many authors in the past have confused the issue by identifying mind with consciousness, at a stage when neither concept was capable of exact statement.

3. The pathological evidences which have absorbed my personal attention have led me to a *reëmphasis of the Flechsig concept of*

anterior and posterior association centers, to a natural correlation of consciousness and the entire sensory portion of the mind with activities of the posterior association center, and to a similar correlation of non-conscious, *i. e.*, objectivistic or behavioristic portions of the mind (notably the voluntary faculties) with activities of the anterior association center: the *prepallium* (pre-Rolandic cortex) would thus be more closely related with behavior (kinetic and pragmatic schemata) and the *postpallium* (post-Rolandic and infra-Sylvian cortex) most closely related with consciousness.

4. But, if the *prepallium* is more an organ of behavior than the receiving *postpallial* mechanism, it is expressly to be stated that the capacity for novelty-production, or innovating power, is not to be abstracted from the prepallial neurones. Such innovating power, exquisitely mental as it seems, is not necessarily conscious in the sense of essentially cognizable. It is perhaps only the *history* of our innovations and inhibitions which we register in the postpallial mechanisms. Arguments in this direction are to be drawn from the decisive ruin of the personality which attends *prepallial* destructive processes in general paresis of the insane.

5. A sketch is offered to show that the non-conscious, *i. e.*, non-cognitive, side of delusion-formation is perhaps more important than the conscious (or contentwise) side. At least the morbid correlates of delusion-formation seem to be prepallial rather than postpallial disorder as a rule.

6. The reverse seems to hold for such apparently motor or behavior phenomena as epileptic and cataleptic phenomena: these are possibly based more often on postpallial (sensorial?, kinæsthetic?) disorder than on intrinsic disorder of behavior mechanisms.