THE ANNUAL OF PSYCHOANALYSIS

(2000). Annual of Psychoanalysis, **28:**179-200

Preliminaries for an Integration of Psychoanalysis and Neuroscience

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It is not difficult to understand why the relationship between psychoanalysis and neuroscience should interest us. Psychoanalysis is a science of the mind, and we have known since ancient times that the activities of the mind are in some intimate way connected with the tissues of the brain. This connection was established, from the start, on *clinical* grounds. Physicians through the ages recognized that diseases of the brain–unlike those of any other organ–had immediate effects on the functions of the mind. The celebrated case of Phineas Gage, which was first reported in 1848, is classically cited in this context (Harlow, 1948, Harlow 1968). A tamping rod passed through the frontal lobes of his brain, with the following results: "His physical health is good, and I am inclined to say that he has recovered … [but] his mind was radically changed, so decidedly that his friends and acquaintances said that he was 'no longer Gage'" (Harlow, 1868). Observations such as these, which demonstrate that the brain and the personality are inextricable, make it clear that the object of study in psychoanalysis is somehow intrinsically connected with the object of study of neuroscience.

Freud himself recognized this fact in his neurological writings, and he continued to acknowledge it throughout his psychological writings. Nevertheless psychoanalysis developed almost completely independently of neuroscience. We all know the reason for this: Despite the fact that Freud acknowledged that 'the mental apparatus… is also known to us in the form of an anatomical preparation,' as he put it (**Freud, 1900**, p. 536), he nevertheless always recommended that psychoanalysts should remain aloof from neuroscience. There seems at first sight to be a contradiction in this stance, but I will clarify Freud's position in a moment.

The mere fact that psychoanalysis and neuroscience developed separately for so long points to the reality that, notwithstanding the obvious connection between

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Presented at a meeting of the Contemporary Freudian Group of the British Psycho-Analytical Society on June 10, 1998.

the two fields, there is a great deal that separates them. Leaving aside the philosophical complexities of the mind/body relation for present purposes, it can safely be said that *in practice* psychoanalysis and neuroscience have separate objects of study, that they bring different methods of investigation to bear on these objects, and that the knowledge they generate is therefore of two distinct types.

This poses obvious problems for those of us who wish to forge links between these two bodies of knowledge, as can be seen from the literature that has begun to accumulate at the interface between them over the past few decades. The first investigator to explore this relationship was, of course, Freud himself. In the mid 1890s, he composed a series of drafts on the subject, one of which has survived to this day in the form of a document known to us as the "Project for a Scientific Psychology." In that work **Freud (1895)** attempted to translate what was known at the time about the deeper workings of the mind into the language of neurophysiology and anatomy. The method that **Freud (1954)** employed to achieve this translation was, as he himself acknowledged, one of *"imaginings, transpositions and guesses"* (p. 120; emphasis added). In other words, Freud relied on *speculation.* At that time (i. e., in the 1890s) the gap between the knowledge that Freud had obtained by the putative method of psychoanalytical investigation about the inner workings of the mind and the knowledge that was available to him from physiological and anatomical methods of study about the inner working of the brain was so large that he had no choice but to fall back on speculation when trying to bridge it. This gap played no small part in Freud's eventual abandonment of his "Project,"

and his description of it as “a kind of aberration” (p. 134). By 1900 he had concluded that “in the end I may have to learn to content myself with the *clinical* explanation of the neuroses” (p. 137, emphasis added; of. **Solms & Saling, 1986**). This remark about clinical explanation, as you will see shortly, is pregnant with implications for our science.

It is my contention that the cause of Freud's failure to integrate his clinical findings with the neuroscience of his day not only was the paucity of neuroscientific *knowledge* that was available to him in the 1890s, but also the absence of a suitable *method* for relating the neurological and psychological data that were available. I believe further that, despite the rapid and exponential increase in our knowledge in all branches of neuroscience, every subsequent attempt to correlate psychoanalytic and neuroscientific knowledge has stumbled on the same basic problem that Freud did 100 years ago, namely, the failure to develop a valid method for *relating* the clinical findings of psychoanalysis with the type of knowledge that is generated by the various neurological sciences. Every investigator who has written on this subject since Freud[1]—dispite the

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1 See www.neuro-psa.com for a complete bibliography. Some examples are **Epstein (1987**, **1989**, **Epstein 1995**), **Erdelyi (1985)**, **Frick (1982)**, **Galin (1974)**, **Hadley (1983**, **1992**), **Harris (1986)**, **Hartmann (1982)**, **Heibrunn (1979)**, **Hoskins (1936)**, **Joseph (1982**, **1992**), **Kokkou & Leuzinger-Bohleber (1992)**, **Levin (1991)**, **MacLean (1962)**, **McLaughin (1978)**, **Meyersburg & Post (1979)**, **Miller (1991)**, **Negri (1994)**, **Olds (1992)**, **Ostow (1954**, **1955a**, **b**, **c**, **1956**, **1959**), **Palombo (1992)**, **Peterfreund (1971**, **1975**), **Reiser (1984**, **Reiser 1990)**, **Schilder (1935)**, **Schore (1994)**, **Schwartz (1987**, **Schwartz 1988)**, **Stone (1977)**, **Winson (1985)**, **Zueler & Mass (1994)**.

brilliance of some of their intuitions–has relied on the same fundamental methodology that Freud did regarding the actual manner is which they correlated the two fields, namely, speculation.

These are some of the reasons why we are now in a situation, with the sudden increase of publications in his area over the past few years, where we have a number of competing and in many respects contradictory models of the neurological organization of the deep mental functions we study in psychoanalysis, without us having any rational basis for deciding between them. How are we to decide between the riveal points of view? I am sure you will agree that we *ouught* to be able to decide such questions for, if the mind and the brain both function in regular and lawful ways, and if those functions and regularities are related to each other in similaly lawful ways–as we have every reason to believe that they are–then it *should* be possible for us to decide such question in ordinary scientific ways.

My primary aim in this essay is to introduce you to a method by which we can achieve this ordinary scientific task. I intend to do so, first, by telling you somethings about where this method came from; second, how it works; and third, by reporting very briefly some of the findings that this method is beginning to produce about how the deeper strata of the mind are organized neurologically.

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To do that, I want first of all to take you backward into history, to trace the origins of psychoanalysis to a particular branch of neuroscience, and to show you how the psychoanalytic method grew out to that branch; then I want to trace subsequent developments in that field to show you that it still remains the natural point of contact between our two disciplines. In the prorcess I hope to be able to demonstrate that–just as we find in our clinical work–a problem, which seems insolubly complex in its present, mature form, frequently turns out to have a relatively simple structure when one traces it back to its origins.

To start at the beginning, Freud began his scientific career as a neuroanatomist, before, following a brief flirtation with psychopharmacology, he turned his attention to the problems of *clinical* neurology. By the time that Freud came to clinical neurology, it was still a very young discipline, which rested almost entirely on a single method. that method was known as *clinico–anatomical correlation*, which was carried over to the new speciality of neurology by some of the ablest practitioners of the art of internal medicine. As its name suggests, internal medicine concerned itself with the diagnosis and treatment of diseases

occurring in the *interior* of the body, which could for that reason not be apprehended directly in the living clincal case, but rather had to be inferred from their indirect manifestations in the form of external symptoms and signs. One had to wait for the death of the patient, and the pathologist's report, before one could determine conclusively whether one's diagnosis was correct. But with the accumulation of experience over generations, regarding what sort of clinical presentation during life tended to correlate with what sort of pathological-anatomical findings at autopsy, it gradually became possible for internal physicians to recognize pathognomonic constellations of symptoms and signs, and thereby to predict with reasonable accuracy what the underlying disease process was, and to conduct the treatment accordingly. This was the origin of the concept of clinical *syndromes*, a concept with which I presume many of you are familiar.

Neurology became a separate speciality of internal medicine as it became increasingly evident, not only that the brain–like any other organ–was subject to its own special pathologies peculiar to its tissues, but also that damage to different *parts* of the brain produced a wide variety of *different* clinical manifestations. When Freud trained in clinical neurology in the early 1880s, this was the art that he learned: rational diagnosis and treatment of neurological deseases by the syndrome method, based on knowledge obtained by the method of clinicoanatomical correlation. In fact, we are told that Freud was a particularly gifted practitioner of this art (**Jones, 1953**). He published a series of articles at the time attesting to his skill.

Now because, as I said at the outset, brain lesions cause *mental* changes, the clinico-anatomical method could be, and was, also put to another use, namely, the *localization of mental functions*. In the early 1860s, Pierre Paul Broca demonstrated conclusively that disease in a particular part of the brain produced a highly characteristic mental symptom, namely, loss of speech. On the basis of the clinico-anatomical correlation, Broca *localized* the faculty of speech to that small part of the brain. Ten years later, Carl Wernicke demonstrated that damage to a different part of the brain produced a different mental symptom, namely, loss of the capacity to *understand* spoken language–and he too localized that function accordingly. These two seminal discoveries were followed by a rapid series of clinico-anatomical correlations in regard to a variety of other mental functions, such as skilled movement, object recognition, and even “intelligence.” On this basis a wide range of psychological faculties were localized within a mosaic of so-called “centers” on the surface of the hemispherres of the brain. This was the origin of a subspeciality within the neurological sciences known as *behavioral neurology*.

We know from Freud's writings of that time that he was thoroughly versed in the methods and discoveries of this exciting new branch of science. In fact there is abundant evidence to suggest that the clinico-anatomical localization of mental functions was a subject of special interst to him (**Freud, 1888**), **1891**, **1893-94)**. Clearly then, Freud was aware before he conceived of psychoanalysis, that

there was a well-established method by which it was possible to correlate mental functions on a clinical basis with the functions of particular parts of the brain. But if that was so, it raises the question, *Why did Freud not use this method to identify the neurological correlates of the psychological processes that he later discovered?* And why don't *we* use it to do so today?

As I have said, Freud was an unusually gifted physician, and it didn't take him long to master the syndrome method in his diagnostic work, and the clinicoanatomical method in his ongoing research. It also did not take Freud long to discover the limits of this method. He soon came to the conclusion that it was, as he put it, just “a silly game of permutations” (**Bernfeld, 1951**, p. 214). This is how it happened. Although it is true that the clinico-anatomical method was the only viable research technique available to the nineteenth-century neurologist interested in mental functions, it was in fact used in subtly different ways within two rival schools of neurology. In the Austro-German school, within which Freud was initially trained, the emphasis fell squarely on the *anatomical* side of the clinico-anatomical equation. According to this school, the primary aim of neurological science was not simply to recognize which syndromes correlated with which lesions, but rather to *explain* the mehanism of the clinical phenomena–and thereby the corresponding normal mental functions-in anatomical and physiological terms. This approach reflected the broader ideals of the Helmholtz school of medicine.

In the rival, the French school of neurology, on the other hand, the emphasis fell very much on the

clinical side of the clinico-anatomical equation. According to this school, which collected around the personality of Charcot and the famous wards of the Salpetriere Hospital, the primary task of neurological science was not so much to *explain* the various clinical pictures, but rather to identify, classify, and *describe* them. The following quotation grphically illustrates the difference between these two ways of applying the clinico-anatomical method:

> *Charcot… neve tired of defending the rights of purely clinical work, which consists in seeing and ordering things, against the encroachments of theoretical medicine. On one occasion there was a small group of us, all students from abroad, who, brought up on German academic physiology, were trying his patience with our doubts about his clinical innovations. "But that can't be true," one of us objected, "it contradicts the Young-Helmholtz theory [of vision]." He did not reply "So much the worse for the theory, clinical facts come first" or words to that effect; but he did say something which made a great impression on us: ["Theory is good; but it doesn't prevent things from existing"] [**Freud, 1893a**, p. 13].*

This was one of Freud's favorite anecdotes.

As is well known, during his period of study at the Salpetriere in the mid 1880s, Freud moved from being under the direct, personal influence of some of the leading figures of the Austro-German school of neurology to being under the

direct personal influence of Charcot. This shift had a decisive influence on his thinking, and, in particular, on his attitude to clinico-anatomical localization.

The reason for this shift was simple. Although the differences between the German and French schools of neurology complemented each other well in regard to most physical neurological disorders, with the one school emphasizing the anatomical and the other the clinical side of the equation, there was one group of diseases–considered at the time to fall under the domain of neurology–that threw the differences between the two approaches into sharp relief. I am referring to the *neuroses*, and to hysteria and neurasthenia in particular, where no demonstrable lesion of the nervous system could be found at autopsy to account for the clinical symptomatology observed during the life of the patient. The posed no serious problems for the French school: Charcot simply proceeded to describe the pathognomonic clinical syndromes of hysteria and neurasthenia as he had done with countless other "nervous" diseases. The neuroses were for Charcot, as Freud wrote at the tiem, "just another topic in neuropathology" (**1893a**, p. 20). However, for the German school of neurologists, the problem was well nigh insoluble. How was one to explain in anatomical and physiological terms the mechanism of a clinical syndrome which had no pathological-anatomical basis? As a result, some German neurologists, Freud's teachers among them, developed elaborate *speculative* theories, whereas others simply declared that the neuroses were not fit subjects for serious scientific attention. If there was no anatomical lesion, there was no clinical syndrome.

During the crucial period that Freud studied under Charcot, this was the subject that most preoccupied him. Initially Freud became a devoted pupil of Charcot, and upon his return to Vienna, he expounded his views whenever and wherever he could–much to the irritation of his old teachers. However, with increasing clinical experience, and under the influence of the English neurologist John Hughlings Jackson, Freud gradually began to edge away from Charcot and to develop a viewpoint which was rather unique at the time. Charcot was content merely to *describe* the clinical syndromes of hysteria and neurasthenia–on the assumption that their pathological-anatomical correlates (which he believed had an hereditary etiology) would *eventually* yield to advances in microanatomical and other laboratory techniques. Freud, on the contrary, came to the view–some time between 1887 and 1893–that an understanding of *these* clinical syndromes would *never* be found in pathological anatomy, or at least *not by the method of clinico-anatomical correlation*. He based this conclusion on two major observations, which he had first made in regard to another subject in neurology, a subject that had revealed to him the *limits* of the clinico-anatomical method. This subject was the problem of aphasia–that is, precisely the subject to which the clinico-anatomical method had first been applied for the localization of mental functions–by Broca and Wernicke–20 or so years before.

First, **Freud (1891)** observed that psychological faculties are complicated things, which have their own compound internal organization, and that these

faculties break down according to the functional logic of their own internal construction, not according to the structural laws of cerebral anatomy. The laws of psychological functional systems therefore need not have any direct relationship to the structrual layout of the nervous system. For this reason, Freud concluded, psychological syndromes need both to be described *and* to be explained in their own psychological terms. Freud's second observation, closely related to the first, was the following: psychological functions are, in their essence, *dynamic* processes; they arise out of a complex interplay of forces between more elementary component functions, and they are constantly restructuring and readapting themselves to changing circumstances. Their physiological correlates can therefore never be loclized *within* discrete anatomical centers; they must be thought of as *processes*–the dynamic resultants of interactions *between* the static elements of the nervous system.

It is of crucial importance for us to note that Freud first made these observations, not with reference to hysteria or any other neurosis, but rather in a study of aphasia–that is, a syndrome that can only ever occur in the context of a definite brain lesion. In other words, these were conclusions that Freud arrived at while he was still a fully fledged neurologist. This is underlined by the fact that he quickly went on to make similar observations in regard to nonpsychological, but equally complex, functions of the brain. In his writings on the disorders of movement that occur in cerebral palsy, for example, Freud went out of his way to demonstrate that they could not be localized. In a series of monographs on the subject (**Freud and Rie, 1891**; **Freud, 1893b**, **1897**), like in his book on aphasia (1891), Freud appealed to dynamic developmental factors, rather than static anatomical ones, to explain the various movement disorders in terms of specific breakdowns of the complex functional system that supports them.

It was only later that Freud applied these principles to *psycho*pathology–between 1893 and 1900–which is when psychoanalysis was born. This is a fact of critical importance, because in the next section of this essay I want to demonstrate how these principles were subsequently developed and expanded *within* the field of neurology, and how *a neuroscientific method for studying the cerebral organization of mental functions was eventually established on precisely these principles*. That is obviously of central importance to us, in our quest for a method by which we may rejoin psychoanalysis with neuroscience.

But before I move on, let me recap and summarize Freud's standpoint. He trained in the clinico-anatomical method of localizing mental functions, within the Austro-German school of neurology, which emphasized the goal of physiological and anatomical explanation. Then he shifted allegiances to the French school, which emphasized the clinical side of the equation: the elucidation of pathognomonic clinical syndromes. He used this clinical-descriptive approach to make a number of highly valued contributions to neurology, first in studies of aphasia, then of cerebral palsy, and lastly of neuroses. In the process of doing

this work, Freud rejected the clinico-anatomical method of localizing mental functions–indeed of localizing any complex functions–withing circumscribed anatomical centers. Freud was forced to conclude that the clinico-anatomical method could only be used to localize the most elementary functions, corresponding in the mental sphere to our primary sensory modalities (of vision, hearing, taste, etc.), but that it was quite impossible to localize the neurological organization of whole mental faculties, which have supraordinate principles of organization based on their own internal construction, which constantly changes in the process of development, and in their adaptation to unfolding circumstances. For Freud, such complex functions arise out of the dynamic interplay of a variety of more elementary functions. He concluded that we should conceive of this interplay as occurring *between* the elementary structures of the brain, and therefore forego the temptation to localize them *within* those elements themselves. It was obvious to Freud, schooled as he was in the art of meticulous clinical observation, that the essential factors in the etiology and mechanism of the neuroses arose out of complex dynamics of this kind, and that they could therefore never be localized. This led Freud *the neurologist* to generalize the conclusions that he had reached in regard to speech and language and voluntary movement to the whole mental field, and to write the following fateful words in *The Interpretation of Dreams*, which marked the final split between psychoanalysis and the clinico-anatomical method:

I shall entirely disregard the fact that the mental apparatus with which we are here concerned is

also known to us in the form of an anatomical preparation, and I shall carefully avoid the temptation to determine psychicial locality in any anatomical fashion. I shall remain upon purely psychological ground, and I propose simply to follow the suggestion that we picture the instrument which carries out our mental functions as resembling a compound microscope or a photographic apparatus, or something of the kind. On that basis, psychical locality will correspond to a point inside the apparatus at which one of the preliminary stages of an image comes into being. In the microscope or telescope, as we know, these occur at ideal points, regions in which no tangible component of the apparatus is situated [1900, p. 536, emphasis added].

What Freud retained, however, and carried over into the new field of psychoanalysis, was almost everything else he had learned as a neurologist. That is, he continued to rely on the clinical-descriptive methods of the French school of neuropathology, with its special emphasis on the careful study of the individual clinical case, and the identification of regular patterns of symptoms and signs with particular pathological significance, and he continued to explain the clinical phenomena in terms of underlying natural forces and energies, as he had been taught to do by his original masters in the Helmholtz school of medicine. He also continued to believe that these forces and energies were ultimately *somehow*

describable in physical and chemical terms. All that he abandoned was the notion that psychological processes, which have complex and dynamic functional organizations, can be *localized* in discrete anatomical areas. Henceforth, rather than attempt to explain a clinical syndrome by correlating it with hypothetical damage to one or another anatomical region, as even Charcot did, Freud investigated the internal *psychological* structure of the syndrome and explained it by reference to a complex functional system, which he assumed to be dynamically represented *between* the elements of the brain.

That is why Freud continued to acknowledge throughout his scientific life that the model of the mental apparatus that he devised to account for his clinical observations was a *provisional* construct, a system of functional relations which must be represented somehow in the tissues of the brain, and that is why he continued to insist that we in psychoanalysis should "not mistake the scaffolding for the building," and so on. I am sure you are all familiar with Freud's many comments to the effect that psychoanalysis will someday be rejoined with neuroscience. I want only to remind you that he always insisted that *this would not be possible until neuroscience developed a method that was capable of accommodating the complex, distributed, and dynamic nature of the human mental process*. I will read just one quotation of this sort, written in 1939, the year of Freud's death:

The psychical topography that I have developed… has nothing to do with the anatomy of the brain, and actually only touches it at one point. [Freud is referring to the primary sensory modalities of the perceptual system.] What is unsatisfactory in this picture–and I am aware of it as clearly as anyone–is due to our complete ignorance of the dynamic nature of the mental process [p. 97].

Until that was understood, Freud insisted, psychoanalysis should continue to investigate and understand the functional organization of the mental apparatus in its own terms, using a purely clinical method, disregarding its anatomical representation.

This places psychoanalysis in a very particular relationship to the neurological sciences. It places its fundamental assumptions and basic method *within* a well-established tradition in behavioral neurology, a tradition that has always been closely associated with the clinical-descriptive emphasis first promulgated by Charcot, and that, following Hughling Jackson, has always rejected the notion that complex mental faculties can be concretely localized in the brain. I am referring to the *dynamic school of neurology*, which has been associated through the years with such outstanding physicians and theoreticians as Constantin von Monakow, Pierre Marie, Henry Head, Kurt Goldstein, Aleksandr Romanovich Luria, and, most recently, Jason Brown.

The influence of this branch of neuroscience has waxed and waned over the decades. Currently it is increasing enormously, as functional imagery and

computer simulation studies have revealed the fundamentally *nonlocalizable and dynamic* “parallel distributed processing” that underlies all mental functioning, and indeed all complex functions of the brain. The *clinical* emphasis of this branch of neuroscience, on the other hand, is on the wane; with the enormous strides that have been made in the use of technological auxiliary aids in medicine, the art of clinical judgement is no longer so highly valued, and the human factor in medicine is being lost. Ironically, one could say that psychoanalysis stands together with this branch of neurology as one of the last outposts of the great clinical traditions of internal medicine.

The important point for our purposes (looking forward rather than backward) is that Freud carried over from neurology into psychoanalysis a basic method–namely, the clinical-descriptive method, or the method of *syndrome analysis* as it later came to be known–and a basic conceptualization of brain-behavior relationships-namely, the antilocalizationist or *dynamic* conceptualization, which gives pride of place to psychological methods of analyzing mental syndromes, regardless of whether those symptoms have an organic basis. This method, and these basic principles, have determined the object of study of psychoanalysis, the way we go about studying it, and, most important of all, the sort of knowledge that psychoanalysis generates.

Now if we wish to integrate knowledge of this sort with knowledge about the brain, then our natural point of contact is with that branch of neuroscience which shares our basic assumptions, and out of which psychoanalysis grew, that is, the dynamic school of behavioral neurology-or neuropsychology as it later came to be known. If we try to relate our clinically generated psychoanalytical knowledge with knowledge about the brain generated by fundamentally incompatible methods, or by methods that Freud explicitly rejected, then we not only are confronted by the insoluble problem of having to rely on speculation (as I said before), but we also have to recognize that we may be doing violence to the basic premises upon which our discipline was built. I am sure you will agree–and this was always Freud's *most* fundamental viewpoint on the matter–that there is little point in rejoining psychoanalysis with neuroscience, if it means that we have to abandon all that psychoanalysis stands for in the process.

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What I would like to do now is describe to you one of the major developments that has occurred since Freud's death in the branch of neuroscience out of which psychoanalysis arose, because I believe that *this development provides us with a method by which we can rejoin psychoanalysis with neuroscience in a way that is compatible with Freud's basic assumptions.*

During the early 1920s, a young Russian psychologist wrote to Freud to apply for formal recognition of a new psychoanalytical society he had formed, in the Eastern city of Kazan. This man was Aleksandr Romanovich Luria. Freud

granted the recognition, and a brief correspondence ensued. A few years later Luria moved to Moscow, and joined the Russian Psychoanalytical Society. Over a period of about ten years, Luria conducted a wide range of psychoanalytic research; published a huge number of articles, monographs, and brief reports; and conducted clinical work in a local psychiatric hospital, including (it is rumored) the analysis of Dostoevsky's granddaughter. Luria was drawn to psychoanalysis, he wrote, because it was the only branch of psychology that was both solidly rooted in natural science and studied the living experience of real human beings.

However, the tide of political opinion soon turned against psychoanalysis in the Soviet Union, and by the early 1930s, fearing for his academic future, if not his life, Luria resigned from the Russian Psychoanalytical Society, abruptly ceased all psychoanalytic activities, and delivered a penitent speech in which he admitted to his ideological mistakes, namely, according to the party line of that time, that psychoanalysis “biologized” human behavior and ignored its social origins. This was a surprisingly naive remark coming from somebody with so complex an understanding of Freud's teaching, but that was not the point. Interestingly there is evidence to demonstrate that Luria never gave up his private interest in psychoanalysis, whatever his public pronouncements. Consider for example a letter he wrote to Oliver Sacks in the mid 1970s, in which Luria described the verbal tics of a patient with Gilles de la Tourette's syndrome as an introjection into the superego of the father's punitive vioce (personal communication from

Oliver Sacks to the author, March 17, 1987).

It is also very striking, in view of the charge that psychoanalysis biologized human behavior, to observe what Luria did next (after resigning from the Psychoanalytical Society). He went to medical school, specialized in neurology, and then immediately set about studying the *mental* symptoms of his neurological patients. And his first piece of research in this field–his doctoral thesis in fact–was on the exact same subject that was preoccupying Freud when he left that field some 40 years before, that is, he studied the subject of *aphasia*.

When Luria finally published the results of his efforts, in 1947, in a monograph in which he scrupulously avoided the name of Freud, he proposed a theory of the cerebral representation of language which was remarkably similar to the one that Freud had proposed in 1891 (**Luria, 1970**). I am skipping over the details here, but would like nevertheless to reproduce one brief quotation. Consider the striking similarity between Freud's suggestion that we view the mind as a complex optical instrument in which psychical locality corresponds to an ideal point in which no tangible component of the apparatus is situated, and the following statement by Luria: "[A]ll attempts to postulate that…ideas could be found in single units of the brain were as unrealistic as trying to find an image inside a mirror or behind it" (1987, p. 489). However, Luria went a step further than Freud; and this represented the essential advance that he contributed to behavioral neurology, or *neuropsychology*, as he preferred to call it.

Luria described his approach as a "neurodynamic" one. He used the following analogy to illustrate the principle:

> *Most investigators who have examined the problem of cortical localization have understood the term function to mean the "function of a particular tissue."…It is perfectly natural to consider that the secretion of bile is a function of the liver and the secretion of insulin is a function of the pancreas. It is equally logical to regard the perception of light as a function of the photosensitive elements of the retina and the highly specialized neurons of the visual cortex connected with them. [You will recall, this was the type of function that Freud believed could be localized.] However, this definition does not meet every use of the term function. When we speak of the "function of respiration," this clearly cannot be understood as the function of a particular tissue. The ultimate object of respiration is to supply oxygen to the alveoli of the lungs to diffuse it through the walls of the alveoli into the blood. The whole process is carried out, not as a simple function of a particular tissue, but rather as a complete functional system, embodying many components belonging to different levels of the secretory, motor, and nervous apparatus. Such a "functional system"…differs not only in the complexity of its structure but also in the mobility of its component parts[1973, p. 27].*

The same could be said of, for example, the function of *digestion*. Luria went on to argue that mental functions, too, can only be localized in this distributed, dynamic sense. In order to identify the different component parts which together make up the complex functional systems of the human mental apparatus, Luria devised a *new* method of clinico-anatomical correlation, known as "dynamic localization." The method works like this: If one wishes to identify the neurological organization of a complex psychological function, one's first task is to identify all the different ways in which that function breaks down with focal neurological disease in different parts of the brain. Luria described this first step as "qualification of the symptoms." One starts with each of the different ways in which the function under study breaks down, and then carefully explores the *psychological structure* of each of these symptoms, identifying precisely in what way the functional system has collapsed in each case. This is done by using psychological methods of analysis in individual clinical cases.

The second step in Luria's method is called "syndrome analysis." That is, one examines what *other* functions are disturbed, apart from the primary function under scrutiny, in each case. Again one relies exclusively on psychological methods of investigation, and one seeks to clarify the internal structure of these other, interconnected symptoms, in order to learn what they have in common with the function that is the primary focus of attention. In this way one identifies a single, *underlying* factor which can account for the full range of surface clinical manifestations.

Once you have identified the common underlying factor producing a range of psychological symptoms, you not only will have learned something about the

deep psychological structure of the syndrome in question, you also will have identified the component function that is contributed by the part of the brain that is damaged in that syndrome. In other words, you will have identified the *elementary psychological function* of one particular part of the brain. This is a major advance.

Once one has studied, by this method, the full series of different ways in which a complex psychological faculty breaks down with damage to *each part* of the brain, then one will have discovered its *distributed* neurological organization, by identifying which parts of the brain contribute, and in what way they contribute, to the complex functional system subserving that faculty as a whole. One will *not* have localized that faculty in any one part of the brain, but one will have identified the various component elements *between* which, by dynamic functional interaction, that psychological faculty is represented.

To my mind this method of Luria's marks a major step forward, because it enables us to identify the neurological organization of *any* mental function, no matter how complex, without contradicting the fundamental assumptions upon which our own discipline was built. By this method, complex psychological functions are still understood in their own, psychological terms; their dynamic nature is respected theoretically and accommodated methodologically; they are not *reduced* to anatomy and physiology, although their neurological distribution is laid bare; and something new is learned about their internal functional organization. By this method a viable *bridge* is established between the concepts of psychology and those of anatomy and physiology and all the other branches of neurological science.

I hope I have not made the neuropsychological method of syndrome analysis sound too complicated, because it really is very simple. I truly believe that this method represents the breakthrough that Freud was waiting for. That is to say, I believe that it enables us to chart the neurological organization of everything that we in psychoanalysis know about the structures and functions of the mind.

3

What I would like to do now is give you an *example* of how this method–which I am claiming is the natural point of contact between psychoanalysis and neuroscience–works in practice. I have chosen for my example a piece of research that I recently completed (**Solms, 1997**) into the neurological organization of a mental function that is of special interest to psychoanalysis. I am referring to the function of *dreaming*.

Using Luria's method to study the dreams of 361 patients with neurological lessions, my research revealed that dreaming is disturbed by damage to six different parts of the brain.

Let me first of all describe the primary effects on dreaming caused by damage to each of these parts of the brain. If the brain is damaged in the white matter

of the mediobasal frontal region, or in the inferior parietal region of either hemisphere, *the conscious experience of dreaming stops completely*. This clinical fact tells us that the basic functions contributed by these three parts of the brain are fundamental to the whole process of dreaming, for when any one of them is damaged, the manifest dream is obliterated entirely. Why that should be the case is revealed in an analysis of the psychological syndrome within which the loss of dreaming is embedded. I return to that point in a moment.

First let me describe the other ways in which dreaming is disrupted by neurological disease. If the brain is damaged in the ventral occipito-temporal region, then the conscious experience of dreaming persists, but *the patient's dreams are devoid of any visual imagery*. Strange as it may seem, patients with damage to this part of the brain have completely nonvisual dreams. (Cases have also been described in which only selected aspects of visual imagery are disturbed–such as, for example, color imagery.)

If, on the other hand, damage is situated in the proximity of the temporallimbic region, and if the lesion is accompanied by a discharging focus (that is, by seizure activity), then the patient experiences *recurring*,

stereotyped nightmares. These nightmares stop if the seizure disorder is brought under control.

Finally, if the damage is situated in the frontal-limbic region (including the anterior cingulate gyrus and basal forebrain nuclei), the patients experience a *massive increase in the frequency of dreaming;* they sometimes experience *continual dreaming;* and they have *great difficulty in distinguishing between dreams and real experiences*.

So these symptoms qualify the different ways in which dreaming can be disrupted by damage to the human brain. Now as I have said, in order to discover what the cause of the breakdown of dreaming is in each of these six instances, it is necessary to study the constellation of other psychological symptoms which accompany the changes in dreaming, following damage to each of these areas. This enables the investigator to isolate the elementary underlying factor, which is common to all of these symptoms, and which is therefore contributed by the part of the brain in question to the overall process of dreaming.

So what are the six elementary factors that are contributed by each of these parts of the brain? Unfortunately, due to limitations of space I cannot describe the full richness of the psychological syndromes from which we have inferred these underlying factors. As a result I am going to have to oversimplify things somewhat. For expository purposes I can only say that an analysis of the psychological syndromes associated with lesions to the six areas of the brain concerned reveals the following basic factors: The mediobasal frontal white matter contributes a general *motivational* factor to mental functioning. The left inferior parietal lobule contributes a factor of *quasi-spatial synthesis*, which is fundamental for *symbolic* mental operations. The right inferior parietal lobule contributes a factor of *concrete spatial representation*. The ventral occipito-temporal

region contributes a factor of *revisualization*, which is essential for *visual mental imagery*. The temporal-limbic region contributes an *emotional arousal* factor, and the frontal-limbic region contributes a factor of *selectivity*, or of *selective activation and inhibition*, which is essential for processes such as attention, reality testing, and judgement.

These six factors together make up the functional system of dreaming, or to put it differently, the process of dreaming arises out of a dynamic interaction between these six factors, which are contributed by six parts of the brain. An analysis of the special structural and functional properties of these six different brain regions, and of the dynamic relations between them, provides a basic scientific understanding of the anatomy and physiology of dreaming.

Finally, to arrive at a truly comprehensive account of the neurological organization of dreaming, it is also necessary to study the component functions of those regions of the brain which do *not* appear to be involved in the process of dreaming. This simultaneously reveals which elementary functions of the human mental apparatus are *not* involved in the psychological construction of dreams. For the purposes of this essay, I will discuss just two of these regions I believe are of particular interest.

The first of these is a core brainstem region, the mesopontine tegmentum. Although damage to this part of the brain severely disrupts the process of *REM sleep*, the *conscious experience* of dreaming persists in these patients. This suggests an unexpected dissociation between the physiological process of REM sleep and the conscious experience of dreams (Solms, **1995**, **2000**). This dissociation is confirmed by the fact that lesions in the forebrain regions discussed earlier, which lead to a complete cessation of the conscious experience of dreaming, have *no effect* on the physiological phenomena of REM sleep.

The other region of interest that I want to draw attention to is the dorsolateral frontal convexity. This part of the brain is enormously important for the *executive* control of waking mental life and of voluntary *motor* activity. However, damage to this region has no effect at all on the conscious experience of dreams. This suggests, not surprisingly, that secondary process thinking and volitional motor activity have little to do with the process of dreaming.

Now if we take a step back and look at all of these factors together, we can arrive at a model of how the dynamic process of dreaming as a whole is organized in the tissues of the brain. On the basis of my research, I have proposed the following model, every detail of which is accessible to empirical verification, by a variety of neuroscientific methods.

It appears that dreaming is stimulated by an *arousal* process. The most common arousal process that

stimulates a dream is the state of neurophysiological activation which regularly occurs every 90 minutes during sleep–namely, the state of REM, which is activated by deep brainstem structures. However, this is only one of the many arousal phenomena that may trigger the process of

dreaming—*and it is by no means the essential one*–for dreaming occurs quite normally without it. Another arousal process that can stimulate a dream is a focal discharge in the temporal-limbic region. However, this is a pathological arousal process, which cannot be bound by the dream process, and which therefore results in anxiety-dreams, or nightmares.

The next important component of the dream process is contributed by a fiber pathway in the mediobasal frontal region. This region of the brain *motivates* appetitive interest in the world (appetitive interest is the term that modern neurobiologists use for what we would call "libidinal interest"). This region channels endogenous arousal processes in the direction of volitional motor activity. An arousing stimulus only triggers the dream work proper if it engages this quasilibidinal brain mechanism. The mediobasal frontal cortex to which this pathway projects also *inhibits* appetitive drives, and, therefore, together with the *selective* structures of the frontal-limbic region, they deflect the arousal process away from the (dorsolateral frontal) executive and motor systems of the brain. These latter systems are inhibited during sleep. But if the brain is damaged in mediobasal frontal cortex and the frontal-limbic region, it seems that the inhibition of these motor systems breaks down, with the result that goal-directed motor activity is instituted, and normal dreaming becomes impossible. This conceptualization of the process is supported by the fact that patients with damage to this region of the brain have *severely disturbed sleep*. If the brain is damaged in the basal forebrain and/or anterior cingulate gyrus, however, the disturbance of inhibition is only partial, with the paradoxical result that there is an *increase* in dreaming and in dreamlike thinking.

Next, assuming that there is a sufficient degree of frontal inhibition, the focus of the nocturnal arousal process shifts to the *posterior* systems of the brain, which regulate *perceptual* functions, and the higher *spatial and symbolic* operations, which are based on perception (inferior parietal and ventral occipitotemporal regions). This, then, becomes the primary "scene of action" of the manifest dream. Here the three other factors that I mentioned come into play: *symbolization*, *spatial thinking*, and *visual mental imagery*. Among these three factors, it appears that *symbolization* and *spatial thinking* are the most important ones, for in their absence, dreaming again becomes impossible, and the whole process breaks down. Visual mental imagery is a less important factor, because the entire mental process of dreaming runs its course without it, the only difference being that the final conscious product is devoid of *visual imagery*. I am therefore inclined to place this factor of visual representation at the *terminal end* of the process of dream generation that I have described.

This overall picture suggests that dreaming is a *regressive* mental process, both triggered by and dependent on nocturnal states of arousal. These arousal states are channeled and inhibited by the systems controlling goal-directed behavior. They are deflected away from the motor systems, toward the perceptual

systems. The higher perceptual systems represent the arousal process in the form of symbolic and spatial syntheses, which are projected regressively onto the lower visual zones. In this way the state of sleep is preserved. If, however, the nocturnal arousal process is excessive, such as occurs with seizures or defective frontal inhibition, then this sleep-protection mechanism fails, and the dreamer is disturbed, either by anxiety or by the innervation of volitional motor activity.

What Luria's method reveals about the neurological organization of dreaming, therefore, is strikingly compatible with Freud's classical theory. Also, because of the centrality of dreaming in Freud's models of the mind, it provides us with a first foothold on the anatomical and physiological representation of some crucial psychoanalytic concepts, including aspects of the libido, censorship, symbolization, topographic regression, and so on. Moreover, although I don't have time to go into all these details here, by identifying the specific tissues of the brain that are involved in the different psychological components of dreaming, it becomes possible to study the finer anatomical, physiological, and chemical correlates of that theory[2]. That

is why I am insisting that the method of dynamic localization provides psychoanalysis with a *conceptual gateway* to the basic neurosciences, and thereby to the enormous advances in knowledge which technological innovations in those fields have yielded in recent years. The potential benefits to psychoanalysis are so obvious that I need hardly enumerate them.

I hope that this brief and oversimplified example makes it clear enough how human mental functions are represented in the tissues of the brain, in the form of complex functional systems that arise out of dynamic interactions between a number of elementary component parts—just like an image arises out of the

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2 For example, closer analysis of the anatomical data reveals that the structures in mediobasal frontal white matter that are crucial for the generation of dreams are the basal forebrain fiber pathways that connect midbrain dopaminergic nuclei with mediobasal frontal cortex (the mesocortical-mesolimbic dopaminergic pathway). This suggests that whatever this fiber pathway does is critical for the function of dreaming (**Solms, 2000**). These are precisely the fibers that were targeted by the modified prefrontal leukotomy procedure which was so popular in the 1950s. There is evidence to suggest that modern antipsychotic medications act on this same pathway (**Breggin, 1980**). A review of the older psychosurgical literature reveals that cessation of dreaming was a common consequence of prefrontal leukotomy (**Solms, 1997**). Evidently whatever it was that prevented leukotomized patients from sustaining their psychotic symptoms also prevented them from generating dreams. I am unaware of any research into the effects of modern antipsychotic medications on dreaming. However, there is considerable evidence that dopamine agonists in general (e.g., L-dopa) stimulate excessive dreaming and that dopamine antagonists (e.g., haloperidol) suppress it (**Sacks, 1985**, **1990**, **1991**). If we review classical dream theory in the light of these findings, we have an empirical basis for linking the libidinal drive (or important manifestations thereof) with mesocortical-mesolimbic dopaminergic pathways. It is therefore not without interest that contemporary neuroscientists include these pathways in the "curiosity-interest-expectancy command systems of the brain … which instigate goal-seeking behaviors and an organism's appetitive interactions with the world" (**Panksepp, 1985**, p. 273).

compound optical instrument, as **Freud (1900)** suggested in *The Interpretation of Dreams*.

I hope also that this example shows how the method of syndrome analysis makes it possible to identify the component parts of the brain between which a complex mental function is distributed, and what the elementary contribution is that each of those parts contributes to the functional system as a whole. This is the scientific yield of the method that we have developed in neuropsychology over the past 60 years, since the death of Freud.

4

Before I can end this essay, and rest my argument for an integration of psychoanalysis and neuroscience on the basis of this method, we must take account of the fact that the research I have just described studied only the *manifest* dream process. In other words, it only studied directly the effects that damage to different parts of the brain have on the conscious experience of dreaming, and it had to infer the underlying unconscious mechanisms from the manifest symptoms. This is because we cannot lay bare the full unconscious structure of a psychological syndrome by examining a neurological patient at the bedside, and still less by assessing him or her in a neuropsychological laboratory. To gain more direct access to these deeper mental strata of a patient, regardless of whether the patient has a brain lesion, we need to get to know the patient as a *person*, within an analytic *relationship*, in a reliable professional setting, within which we can with their confidence through tact and understanding, and by analyzing their resistances, and then unhurriedly observing the way in which the internal determinants of the symptoms gradually unfold in the transference, and by testing the hypotheses that occur to us in this regard in the form of appropriate interpretations, and observing the effects that these have on the subsequent analytic material, and so on. In other words, we can only properly elucidate the *dynamically unconscious* structure of a mental symptom by means of the *psychonalytic* method.

We all know that this is not the easiest way to study a psychological syndrome, but we also know that *it is the only true and reliable method when it comes to those deeper aspects of mental life that neuropsychology has left unstudied* but that have always been of central concern to *us* in psychoanalysis,

namely, the dynamically unconscious structure of the human personality. In fact, the emotional resistances which conceal the internal structure of the personality probably explain why the neurological organization of this, the most important aspects by far of human mental life, have still not been systematically explored by the method of syndrome analysis. This is the scientific contribution that I believe psychoanalysis can make to neuroscience, and this is the next step that I believe we must now take.

Ironically we owe the development of a clinical procedure for analyzing these deeper mental strata to the fact that Freud *abandoned* neuroscientific methods of investigation when he realized that they were (at that time) unable to accommodate the dynamic nature of human mental processes. Now it seems the time has come for us to reintroduce the fruits of his labors to the neuroscientific field out of which they originally grew. In doing so–although I do not wish to underestimate the enormity of the task before us–I believe that we will be able to gradually integrate psychoanalysis and neuroscience, on a solid *clinical* basis, in a way that is beneficial to both fields, without ignoring any of the valuable lessons that the pioneers of psychoanalysis fought so long and hard to learn.

What I am recommending, therefore, and what I believe will provide the essential cornerstone for a lasting integration of psychoanalysis and neuroscience, is a fully *psychoanalytic* investigation of patients with focal neurological lesions. In other words I am recommending that we chart the neurological organization of the deepest strata of the mind, using a psychoanalytic version of syndrome analysis, by studying the deep structure of the mental changes that can be discerned in neurological patients within a psychoanalytic relationship. If I had more space, I would have liked to describe the preliminary results of a study that my wife and colleague, Karen Kaplan-Solms and I began fourteen years ago, using precisely these methods (**Kaplan-Solms & Solms, 2000**). We have so far studied the subjective life of 35 patients with focal brain lesions by taking them into psychoanalysis or psychoanalytic therapy. Colleagues in America, Austria, and Germany are begining to undertake similar studies. This research is starting to reveal the neurological organization of those deeper functional systems which only the psychoanalytic method of investigation *can* reveal. We report on this research in detail in our forthcoming monograph (**Kaplan-Solms & Solms, 2000**). Unfortunately I only have space here to make the point that *it is now possible*, using the methods that I have described, to elucidate the neurological organization of the deepest mental functions that we have traditionally studied in psychoanalysis using purely psychopathological material.

I hope that I have conveyed this point convincingly, despite the fact that I have only been able to *hint* at how my way of approaching the problem actually works in practice, and have only been able to describe a *fragment* of the sort of data that it generates. Nevertheless I hope that I have at least been able to convince you of *the principle* that this is a worthwhile way to proceed. I will know that I really have succeeded in doing so if some readers put into practice the method I am recommending, and judge for themselves if it is capable of achieving what I am claiming for it. An enormous scientific effort lies before us, so I need hardly say that the more of us who involve ourselves in it, the better.

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